Central Resetting of Neuromuscular Steady States May Underlie Rhythmical Arm Movements

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1School of Rehabilitation and 2Department of Physiology, University of Montreal, Montreal, Quebec, Canada; 3School of Physical and Occupational Therapy, McGill University, Montreal, Quebec, Canada; 4Jewish Rehabilitation Hospital Research Site of the Centre for Interdisciplinary Research in Rehabilitation (CRIR), Montreal, Quebec, Canada; and 5Institute of Neurology, Russian Academy of Medical Sciences, Moscow, Russia

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Ustinova, Ksenia I., Anatol G. Feldman, and Mindy F. Levin. Central resetting of neuromuscular steady states may underlie rhythmical arm movements. J Neurophysiol 96: 1124–1134, 2006. First published May 17, 2006; doi:10.1152/jn.01152.2005. Changing the steady-state configuration of the body or its segments may be an important function of central pattern generators for locomotion and other rhythmical movements. Thereby, muscle activation, forces, and movement may emerge following a natural tendency of the neuromuscular system to achieve the current steady-state configuration. To verify that transitions between different steady states occur during rhythmical movements, we asked standing subjects to swing one or both arms synchronously or reciprocally at ~0.8 Hz from the shoulder joints. In randomly selected cycles, one arm was transiently arrested by an electromagnetic device. Swinging resumed after some delay and phase resetting. During bilateral swinging, the nonperturbed arm often stopped before resuming swinging at a position that was close to either the extreme forward or the extreme backward arm position observed before the perturbation. Oscillations usually resumed when both arms arrived at similar extreme positions when a synchronous bilateral pattern was initially produced or at the opposite positions if the initial pattern was reciprocal. Results suggest that a central generator controls both arms as a coherent unit by producing transitions between its steady state (equilibrium) positions. By controlling these positions, the system may define the spatial boundaries of movement. At these positions, the system may halt the oscillations, resume them at a new phase (as observed in the present study), or initiate a new motor action. Our findings are relevant to locomotion and suggest that walking may also be generated by transitions between several equilibrium configurations of the body, possibly accomplished by modulation and gating of proprioceptive reflexes.

INTRODUCTION

In animals and humans, rhythmical movements (e.g., locomotion) are thought to be generated by central pattern generators (CPGs) that may influence the frequency and amplitude of oscillations, the sequence of muscle recruitment, and adaptation to changes in external conditions or task demands (Dietz 2002; Grillner and Wallen 1985; Shik and Orlovsky 1976; Stein 2005; Stein et al. 1995; Zehr and Haridas 2003). Adaptability of the CPG is based not only on proprioceptive and cutaneous feedback but also on the ability of spinal and supra-spinal systems to modulate or “gate” proprioceptive inputs to muscles, shift the phase of rhythmic generation (“phase resetting”), modify the phase relationships between different extremities, e.g., from in-phase to anti-phase (“phase transition”) (Kelso 1984), switch to other rhythmical or non-rhythmical actions, interrupt or cease the movement. These control abilities are helpful in many situations, for example, in changing the movement direction, avoiding obstacles, and reacting to sudden perturbations such as stumbling.

It has recently been proposed that the neuronal control of rhythmical arm movements in humans is regulated by proprioceptively modulated CPG activity similar to that of the legs during locomotion (Dietz et al. 1994; Zehr and Duysens 2004; Zehr and Haridas 2003) and may share some of the characteristics of interlimb coordination described for locomotion in cats (for review, see Dietz 2002). In humans, the coupling strength between the arms appears to be weaker than between the legs. The stronger inter-leg coupling is likely related to the need to compensate the body weight and to rhythmically transfer the weight from side to side during locomotion. The CPG circuits may be reconfigured to produce different types of rhythmical activities in humans and animals (Berkinblit et al. 1978; Fukson et al. 1980; Schaal et al. 2004; Smits-Engelsman et al. 2002; for reviews see Marder 2000; Dietz 2002; Stein 2005). The relative duration of movement phases depends on the motor task and, due to afferent feedback, on the position of the moving limbs as has been shown for scratching and fictive locomotion in spinal or decerebrated cats (Berkinblit et al. 1978; Pearson and Rossignol 1991; Saltiel and Rossignol 2004) as well as for the wiping reflex in spinal frogs (Fukson et al. 1980).

A common feature of all types of rhythmical activities is the alternating, with some overlapping, activation of opposing muscle groups. Such alternating activation is usually produced by two mutually inhibiting groups of interneurons (half-centers) that are facilitated by different descending systems and which in turn facilitate, for example, flexor and extensor motoneurons (Brown 1911; Grillner 1981; Jankowska et al. 1967). The switching from the activity in one half-center to the other is defined by the state of reflex loops and by intrinsic limitations in the duration of the inhibition by the active half-center (Grillner 1972; Pearson 1975; Shik and Orlovsky 1976). CPG networks have been shown to be flexible in producing and blending different motor behaviors (Burke et al. 2001; Dietz 2002; Jankowska et al. 2003; Stein 2005).

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Although some rhythmical activities may be similar in timing, they may differ spatially (e.g., walking on an even surface compared with stair climbing; scratching different parts of the body; hammering toward a vertical compared with a horizontal surface) necessitating another element of control related to the spatial requirements of the task. The same timing of electromyographic (EMG) activity, such as the relative duration of flexor and extensor phases can be produced at different limb positions, implying that EMG timing per se does not predetermine where, in spatial coordinates, the motor action occurs.

It has been shown that cortico-spinal, vestibulospinal, reticulospinal, rubrospinal tracts, and intra-spinal systems are able to change the threshold positions at which proprioceptive feedback becomes effective in activating appropriate limb muscles (Asatryan and Feldman 1965; Feldman 1986; Feldman and Orlovsky 1972; Matthews 1959; Nichols and Steeves 1986). This implies that the spatial coordinates in which rhythmical or other motor actions occur are defined by specific neurological parameters, such as the threshold positions at which proprioceptive and cutaneous reflexes and muscles become active, rather than by EMG characteristics. By setting such thresholds, the CPG can influence the steady (equilibrium) state (Feldman 1980) and thereby elicit EMG patterns and muscle forces tending to bring the system to this state. The CPG may thus produce rhythmical transitions between several equilibrium positions, defining not only the orientation but the spatial boundaries of the rhythmical movement. Using this approach, it has been suggested that walking may result from CPG-evoked changes in the equilibrium configuration of the body leading to steps and continuous displacement of the body without loss of balance (Feldman and Levin 1995; Günther and Ruder 2003).

This major function of the CPG—the spatial organization of rhythmical movements, especially in terms of the type and number of equilibrium states traversed by the system—has not been investigated in a systematic way. To address this issue, we analyzed, by perturbation methods, a comparatively simple rhythmical movement: swinging one or both arms moving in-phase (synchronously) or anti-phase (reciprocally) about a rhythmical movement cycle when the arm was moving forward. The second arrest was made after the next three to four cycles when the arm was moving backward (Fig. 1B). In terms of phase angles, each cycle was defined as starting from 0° (the point t₀ of the maximal backward deviation of the wrist) and ending at 360° (the next similar point). The point of the maximal forward deviation of the wrist (t₁) was close to 180°. A computational algorithm was used to identify the phases of oscillation in the first cycle to automatically deliver perturbations in the later cycles.

**METHODS**

Fourteen healthy subjects (8 men, 6 women, 51.7 ± 12.9 yr old; who comprised a control group for another, clinical study) participated in the study after signing informed consent forms approved by the institutional ethics committee. All subjects were right-handed according to the Edinburgh Handedness Inventory Scale (Oldfield 1971).

**Experimental procedures**

While standing, participants swung left, right, or both arms in synchrony or reciprocally. The arms swung forward or backward in the sagittal plane(s) with the rotation centered about the shoulder joints. Subjects were asked to swing their arms as naturally as possible at a standard frequency. Before each trial, movements were standardized by synchronizing them with an auditory signal from a metronome oscillating at 0.8 Hz, close to the frequency of self-paced arm swinging (Ustinova et al. 2004). When data collection started, subjects continued to produce arm swinging at about the same frequency without external pacing.

In each trial (~12 movement cycles), one arm was unexpectedly and briefly perturbed (arrested) in two randomly selected nonsequential cycles. The perturbation was delivered through a light but rigid plastic rod (1.4 m long). One end of the rod slid on low-friction bearings inside a cylindrical electromagnet (solenoid) attached by another universal joint to a bar of adjustable height situated behind the subject (Fig. 1A). When the electromagnet was turned off, the resistance of the apparatus to arm movement was negligible. When activated by an electrical pulse, the electromagnet clamped the rod and briefly interrupted the movement of the arm at the level of the wrist, after which the arm continued swinging. The duration of the electrical pulse eliciting the perturbation in all trials was fixed at 150 ms, which resulted in a mechanical stimulus (arm arrest) of ~200 ± 40 ms. The difference was due to the magnet’s hysteresis and variations in friction. Given the cycle period (~1,400 ms), the variation in the perturbation duration was relatively low. Extreme values of duration (100 and 350 ms) occurred only occasionally (in ~3% of all perturbations/subject).

To avoid the subject anticipating the perturbation, the first perturbation in each trial was applied in the randomly chosen third or fourth movement cycle when the arm was moving forward. The second arrest was made after the next three to four cycles when the arm was moving backward. Perturbations were applied only to the moving arm. Perturbations occurred only in the first 30% of each movement cycle, with the first 2% of the cycle left unperturbed (Fig. 1C). The perturbation onset (ts) was preset for each trial, and the position at which rhythmic swinging resumed, respectively; Δt, the interval defining the phase shift (Δφ, see METHODS) elicited by perturbation.

![FIG. 1. Experimental set-up (A) and effects of temporary arrests of the arm when the arm moved forward or backward during unilateral swinging (B and C). Actual (black curves), projected (gray) and retro-trajectories (dashed) of wrist motion are shown. Projected trajectories: actual wrist trajectories extrapolated beyond the point (t₀) of the perturbation onset. Retro-trajectories: postperturbation trajectories shifted backward in time. T, preperturbation cycle duration; s₁, s₂, the extreme wrist positions during preperturbation swinging; t₀, the time when a maximal backward wrist deviation occurred; t₁, the onset and the position at which rhythmic swinging resumed, respectively; Δt, the interval defining the phase shift (Δφ, see METHODS) elicited by perturbation.](image-url)
In session 1 (9 subjects, 6 men 3 women), arrests were initiated in two angular windows (80° ± 20° or 280° ± 27°) in each trial, i.e., about half way between the maximal arm excursions when the arm moved forward and backward, respectively. During unilateral movements, perturbations were applied to the moving arm (5 trials for each arm). During bilateral movements, perturbations were applied to one arm (5 trials) and then to the other arm (5 trials) for each type of coordination (synchronized and reciprocal).

Session 2 (5 other subjects, 2 men and 3 women) was focused on the phase-dependent effects of perturbation in bilateral synchronous and reciprocal movements. The right arm was arrested in randomly selected cycles but the perturbation was applied at 10 approximately equidistant phases of oscillations (in 2 different cycles in each trial), instead of only two phases as in session 1. The number of trials was also increased from 5 to 10.

Subjects were asked to continue swinging despite the perturbation and to maintain the same amplitude of arm swinging throughout the whole trial. Subjects could see their arms but no additional feedback about the effects of perturbations was provided during any trial.

Data analysis

Three-dimensional kinematic data were collected with reflective markers placed on the dorsum of both hands proximal to the head of the third metacarpals. Marker movements were recorded with a Vicon Motion Analysis 512 system (Oxford Metrics; sampling rate: 120 Hz). The displacement of the left and right hand markers (arm displacement), the amplitude, and the cycle period (T) of swinging were computed after low-pass filtering the raw data at 6 Hz. The amplitude of arm swinging was calculated as the peak-to-peak arm displacement in the sagittal (anterior-posterior) direction and period T, as the time between two sequential maximal backward deviations in pre-perturbation cycles (Fig. 1B).

To characterize responses to the perturbation, a section of the sine-like curve representing the pre-perturbation oscillations was copied and shifted forward by several cycles thus projecting the actual curve beyond the point of perturbation. We thus obtained a projected trajectory, i.e., the trajectory that could be observed if it were not affected by the perturbation (Fig. 1B, gray curves). The time when the actual and projected trajectories began to diverge was identified as the onset (t_r) of perturbation. The respective phase of the arrest onset was measured from the point (t_r) of the preceding maximal backward deviation of the arm (Fig. 1B)

\[ \varphi_a = 360^\circ (t_r - t_0)/T \]

T is the time between the adjacent backward positions in the cycle preceding the perturbation.

The phase shift resulting from the perturbation was measured as

\[ \Delta \varphi = 360^\circ \Delta t/T \]

Here \( \Delta t \) is the time between the adjacent actual and projected backward positions in the first restored postperturbation cycle (Fig. 1B). According to this definition, a positive shift means that the oscillations after perturbation are in advance of those before the pre-perturbation oscillations and a negative shift means that the renewed oscillations are delayed with respect to the pre-perturbation oscillations.

To determine how quickly rhythmic oscillations were restored after the perturbation, we used a retro-trajectory (Fig. 1C, dashed curve) by taking a section of rhythmic oscillations established some time after the perturbation and shifting it back in time by two to three cycles. The first point at which the actual and the retro-trajectories converged identified the time, \( t_r \), when oscillations were fully re-established (reset). The difference, \( t_r - t_r \), represents the interval when the rhythmic oscillations were interrupted by the perturbation. Index \( I \) expresses the same event in terms of a fraction of the cycle it occupies

\[ I = (t_r - t_0)/T \]

To facilitate the data presentation, only projected trajectories are shown in figures. Retro-trajectories were used to identify points \( t_r \) (short vertical lines in Fig. 2) but are not shown in the figures.

We determined the arm position, \( s_t \), achieved at time \( t_r \), i.e., the position at which rhythmic oscillations were re-initiated. We also estimated how far this position deviated from the extreme forward and backward arm positions (Fig. 1C, positions \( s_a \) and \( s_b \)). We normalized this deviation (D) to the movement amplitude, \( A = s_t - s_0 \), and expressed it as percent of the amplitude

\[ D = 100(s_t - s_0)/A \]

According to this formula, \( D = 0 \% \) if \( s_t \) coincides with \( s_0 \) and \( D = 100 \% \) if \( s_t \) coincides with \( s_0 \). Intermediate values of D indicate the cases when \( s_t \) falls in between the two positions.

To characterize muscle responses to perturbations as well as the muscle activity at the positions at which oscillations were re-initiated, we recorded EMG activity of a shoulder flexor, anterior deltoid (AD) and a shoulder extensor, posterior deltoid (PD) from both arms. EMG signals were recorded with active bipolar surface electrodes, amplified, filtered (75–500 Hz; the lower frequency was chosen to exclude movement artifacts) and digitized (sampling rate: 1,080/s). To analyze a muscle response to perturbation, we defined the time between the beginning of arm perturbation and next postperturbed muscle burst. The beginning of muscle burst was defined as the time when muscle activity exceeded mean ±2 SD of its background amplitude, while the offset of the muscle burst as the time when muscle activity fell <10% of maximum EMG amplitude in the same cycle of movement. We used three pre-perturbation cycles to obtain EMG envelopes (low-pass frequency: 20 Hz) by computing the root mean square (RMS) of the EMG signal for each of four muscles in each of nine arm positions (separated by ~45°) of the movement cycle. These RMS signals were compared with those in the perturbed period for each of the four muscles. The EMG magnitudes of each muscle at the positions at which oscillations were restored were compared with those at the respective positions before the perturbation.

Two-way ANOVAs were used for within-group comparisons. The mean periods and amplitudes in the two cycles preceding the first perturbation were analyzed for each movement condition (unilateral, bilateral in-phase and bilateral anti-phase swinging; right and left arms; forward and backward directions). The between-arm difference in phase shift (\( \Delta \varphi \)) and the position at which oscillations were re-initiated (D) was determined based on the factors: arm (perturbed, nonperturbed) and direction of perturbation (forward, backward) compared separately for each side. To determine whether or not the shift in the phase depends on the phase of arrest onset (\( \varphi_a \)), we used Pearson correlation analysis. The magnitude of EMG activity was analyzed using two-way ANOVAs with factors: position (extreme pre-perturbed forward/backward, position of re-initiation of oscillations) and muscle (agonist, antagonist) separately for unilateral and bilateral synchronized and reciprocal swinging. Significance levels of \( P < 0.05 \) were used for all tests.

RESULTS

Unless stated otherwise, data from session 1 characterize major effects of perturbation (arm arrests at 2 phases, when the arm was oriented more or less vertically and moving forward or backward). Then data from session 2 (arm arrests at 10 phases in the cycle) describe in more detail how these effects depended on the phase of perturbation.
Oscillations are interrupted by perturbations but are rapidly restored

Cycle periods (mean ± SD) prior to perturbations were similar during both unilateral and bilateral swinging for both right and left arms in each subject [−1.40 ± 0.20 s; F(2,16) = 1.53, P > 0.05]. The same was true for movement amplitudes prior to perturbations [range of means 68–76 cm; F(2,16) = 2.14, P > 0.05]. In 90–95% of both uni- and bilateral movements, the perturbation interrupted the rhythmic arm swinging as can be seen from the comparison of the duration of the transitional interval (interval between the maximal backward arm deviations immediately before and after the perturbation) with the period of swinging prior to the perturbation (Figs. 1, B and C, and 2). In particular, in unilateral swinging, the transitional interval increased from 1.38 ± 0.18 to 1.62 ± 0.13 s in 70% (Fig. 2, A and C), decreased to 1.25 ± 0.06 s in 23% (B), or remained unchanged in 7% of cases (D). Perturbation also influenced the transitional interval during bilateral synchronized and reciprocal swinging (Fig. 2, E–H) but to a lesser degree than during unilateral swinging [increased to 1.56 ± 0.09 s; decreased to 1.30 ± 0.05 s; F(2,16) = 6.32, P < 0.01]. There were, however, noticeable differences in the behavior of the two arms: when one arm was arrested, the nonperturbed arm continued to move without obvious distortions until it stopped at one of the two extreme positions (Fig. 2, E–H).

Released from the arrest, the perturbed arm usually resumed motion in the previous direction (Figs. 2, A and C, and 3A, upper curve). However, in ~25% of cases, the arm moved in the opposite direction and resumed oscillations from a position close to the maximal arm deviation (Figs. 2B and 3A, lower kinematic curve). During bilateral movements, the perturbed arm, when released, began to move in the previous direction (in ~75% of cases, Fig. 2, E–G) or in opposite direction (25%, Fig. 2H) and resumed oscillations when it reached or approached one of the two extreme positions.

Group data from session 2 show that the probability of movement reversal after the arm arrest depended on the phase of perturbation (Fig. 3B). Released after the arrest occurring soon after the arm began to move forward, the arm usually (in >85%) resumed motion in the forward direction. When the perturbation approached the extreme forward position, reversals in direction became more probable. Similar changes in probability occurred when the arm was arrested during backward movement (Fig. 3B). ANOVAs revealed significant differences in the number of cases with and without movement reversals [F(4,20) = 5.97, P < 0.01] for perturbations applied during early (phases: 36 ± 8 and 80 ± 9°) versus later (phases: 126 ± 6 and 159 ± 3°) stages of forward motion. The changes in reversal/no reversal numbers did not differ for forward and backward or synchronous and reciprocal arm movements.

Soon after the end of perturbation, the oscillation period and the movement amplitude rapidly returned to their preperturbed values in all subjects and conditions. The rhythmic oscillations were restored at the positions marked by short vertical lines in Fig. 2. In all cases, the transition to rhythmic oscillations was accomplished in less than 3/4 of the preperturbed period as defined by the index (I) showing the fraction of the cycle when the movements differed from the preperturbed ones (see METHODS). This index varied from 0.56 to 0.75 of the cycle period for unilateral and from 0.53 to 0.70 for bilateral swinging. Two-way ANOVAs revealed no differences in this index for perturbed and nonperturbed arms during bilateral swinging [F(1,16) = 1.14, P > 0.05 for right side perturbation; F(1,16) = 1.97, P > 0.05 for left side perturbation] with no effect of the direction of the arrest. These findings imply that applied to one arm, the perturbation disturbed the movement of both arms (bilateral effects). Although the perturbation began to influence the nonarrested arm later than the arrested arm, rhythmic oscillations re-started simultaneously in both arms.

![Fig. 2. Typical effects of temporary arrests on the perturbed (asterisk) and nonperturbed arm during unilateral (A–D) and 2 types of bilateral swinging (E and F and G and H). Data from session 1 for subjects S1–S3. Black and gray curves: actual and projected trajectories, respectively. Dashed vertical lines indicate the onset of perturbation. Short vertical lines indicate the onset of rhythmic oscillations after perturbation.](JNeurophysiol-Vol-96-Issue-5-Fig-2.png)
Phase resetting

As a rule, the restored oscillations were shifted in time compared with the initial oscillations as is obvious from the distance ($\Delta t$) between the actual and projected trajectories (Figs. 1, B and C, and 2). In other words, in the majority of cases, perturbations resulted in phase resetting. Phase shifts for each of the two phases of perturbation in session 1 could be negative (Fig. 2B), positive (A, C, and E–H), or zero (D), which reflected the changes in the transitional interval (see preceding text). The probability (frequency) of phase shifts was characterized by a somewhat asymmetric bell-shaped distribution across the interval between $-100$ and $150^\circ$ for both unilateral and bilateral movements (Fig. 4), with the peak of the distribution located in an interval between $0$ and $50^\circ$. On average, negative shifts ($-2.9 \pm 16.5^\circ$ for unilateral and $-19.8 \pm 9.3^\circ$ for bilateral swinging) were smaller in terms of absolute values than positive shifts ($64.6 \pm 33.5$ and $47.7 \pm 22.3^\circ$, respectively). Phase shifts were similar for the two bilateral patterns of swinging (synchronous or reciprocal), regardless of the side of perturbation [$F(1,16) = 0.78$, $P > 0.05$ for the right side; $F(1,16) = 1.10$, $P > 0.05$ for the left side].

FIG. 3. Typical examples (A) and group data (B) showing that the probability for the perturbed arm to resume the previous direction or move in the opposite direction after arrest depends on the phase of perturbation. A: actual and projected hand displacements (top) and effects of the arm arrest on electromyographic (EMG) signals (session 1). Vertical lines indicate the onset of perturbation. B: number of cases (of 50 for the group) in which the previous direction was resumed (●) or reversed (□) for different phases of perturbation (session 2). Encircled on the abscissa are the positions at which the arms arrived before resuming oscillations.

FIG. 4. The probability (in % of cases) of different phase shifts elicited by perturbations during unilateral, bilateral synchronized and bilateral reciprocal swinging for arrests during forward (■) and backward (□) arm motion (session 1).
Data from session 2 show that the magnitude of phase resetting was correlated with the phase of arrest (Fig. 5, r ranged from −0.61 to −0.83, P < 0.01). Correlation was done separately for arrests during motion in the forward (from 0 to 180°) and backward (from 180 to 360°) directions. Except for the points close to the maximal forward and backward arm positions, positive shifts prevailed in response to arrests at any phase of movement. The number of positive or negative shifts was maximal when the arrests occurred near the midpoint of the forward or backward movement range (as was the case in session 1) and were minimal when the perturbation was delivered near the extreme forward (180°) or backward (360°) positions, whether the movement pattern was synchronous or reciprocal.

Thus perturbations elicited phase shifts in oscillations of both the perturbed and nonperturbed arms (bilateral effects). The phenomenon of phase resetting was observed in all subjects regardless of the type of bilateral coordination (synchronized or reciprocal) or the side and the direction of perturbation. Different subjects also showed similar distributions of positive and negative phase shifts in all experimental conditions.

Transitions between equilibrium (steady) states during rhythmic arm movements

After perturbations of unilateral swinging, rhythmical oscillations usually resumed when the arm reached a position that was close to either the maximal forward or maximal backward deviation attained during preperturbed oscillations (Fig. 2, A–C; short vertical lines; see METHODS on how these positions were identified; Fig. 6A, left).

The behavior of the nonperturbed arm during bilateral swinging was most indicative of the positions at which the system could temporarily stop before resuming oscillations. For example (Fig. 2, E and G, top), when the left arm was briefly arrested during a forward movement, the nonarrested right arm continued to move and stopped later at the point of maximal forward deviation. The nonperturbed arm could also stop, although briefly, near the maximal backward position (F and H, bottom). Note that the nonperturbed arm could stop at one or the other extreme position, which could occur both during synchronized and reciprocal patterns of swinging. Typically, oscillations resumed when, after being released, the arrested arm arrived at a similar extreme position for synchronized arm swinging (E and F, short vertical lines) or at the opposite position for reciprocal arm swinging (G and H).

The positions at which oscillations were re-initiated were similar in all subjects as illustrated by the histograms in Fig. 6A. The positions at which oscillations were re-initiated somewhat undershot the extreme positions. During bilateral synchronized swinging, oscillations of both arms were reset practically at the same position [forward or backward; F(1,16) = 0.97, P > 0.05], whereas during reciprocal swinging, one arm resumed oscillations from the forward whereas the other arm from the backward position [F(1,16) = 28.5, P < 0.001]. Regardless of the number of phases at which perturbations were produced (2 in session 1 and 10 in session 2), the positions (forward and backward) at which the oscillations resumed were the same (Fig. 6, A and B). There were no cases when the nonperturbed arm stopped at a near vertical position (which would have been the case if this position played the role of the equilibrium position relative to which oscillations occurred).

Thus oscillations were re-initiated only when the unilaterally swinging arm or both arms swinging synchronously arrived at similar positions near the maximal forward or backward arm deviations. During reciprocal swinging, the oscillations resumed when the two arms were almost maximally deviated in opposite directions.

EMG effects

Figure 7 is a representative example of EMG patterns obtained in the present study. During rhythmic oscillations before the perturbations, EMG bursts in opposing muscle pairs (AD and PD) of the same arm showed a reciprocal pattern with some degree of co-activation. EMG bursts occurred between the points of extreme deviations of the arm(s) so that the EMG activity for all recorded muscles at the extreme points was minimal (although not 0) compared with the EMG activity at other phases of the cycle (Fig. 7). Data for the group of nine subjects (Fig. 8) showed that the minimal activity was <10% of the individual maximal EMG burst amplitude for each muscle. This was the case for both uni- and bilateral movements (Figs. 7 and 8). Figure 7 also shows that after perturbations, oscillations were re-initiated when the state of minimal activity in all muscles was re-established to the preperturbed minimal level, whether the move-
ments were unilateral, bilateral synchronized, or bilateral reciprocal $[F(1,16) = 1.21, P > 0.05$, for unilateral; $F(1,32) = 2.79, P > 0.05$ for bilateral synchronized; and $F(1,32) = 3.65, P > 0.05$ for reciprocal swinging].

Mechanically, the perturbation intermittently prevented shortening of the agonist muscle so that the muscle appeared to be lengthened (stretched) with respect to the nonperturbed length. As a result, the perturbation elicited a short-latency (30 ± 4 ms), EMG burst in the agonist muscles. This EMG burst was accompanied by a short silent period (29 ± 5 ms) in the activity of the antagonist (AD) muscle as illustrated in Fig. 9 for movement in the backward direction. After this EMG burst, the agonist muscle was silent (80 ± 23 ms), probably due to postspike hyperpolarization of motoneurons after synchronous activation (e.g., Baldissera and Parmiggiani 1979; Merton 1951). The subsequent changes in the EMG activity were related to the direction of motion after the end of the arm arrest. The re-appearance of a prolonged burst of activity in the agonist muscle (PD in Fig. 7A) 130 ± 32 ms after the arrest onset indicated that the previous movement direction was resumed. This burst terminated when the respective maximal arm deviation (backward in Fig. 7A) was reached. In contrast, if the movement direction was reversed (Fig. 7, B and C), no burst in the PD muscle appeared and, instead, an EMG burst was generated in its antagonist (AD) until the opposite (forward) arm deviation was reached. The patterns of initial EMG responses to perturbations were qualitatively similar even though the phase shifts differed substantially (negative shift in B and 0 shift in C). By comparing the intervals between sequential minima of muscle activity (dashed vertical lines in B–E) before and after the perturbation, one could determine whether or not the phase was shifted both for uni- and bilateral swinging.

During bilateral swinging (Fig. 7, D and E), the patterns of EMG responses of muscles of the perturbed arm resembled those of muscles during unilateral movements. The EMG patterns of the nonperturbed arm reflected the necessity to wait for the perturbed arm to arrive at the appropriate position before resuming swinging. This was especially apparent in those cases when the waiting period was comparatively long (Fig. 7E). Then the activity of the agonist muscle (LAD in Fig. 7E) of the nonperturbed arm was also prolonged, more powerful, and accompanied by increased activity of the antagonist muscle (co-activation) until the activity of all four muscles was minimized just before reaching the position(s) at which the regular swinging resumed.

D I S C U S S I O N

Basic findings

Phase resetting regularly occurred in response to a transient arrest of one arm whether it swung alone or together with the other arm. In the latter case, the nonperturbed arm continued to move but usually stopped at a position that was close to the
extreme forward or backward position observed before the perturbation. Regardless of the phase at which the perturbation was made, oscillations usually resumed when both arms arrived at similar extreme positions if a synchronous bilateral pattern was initially produced or at opposite extreme positions if the initial pattern was reciprocal. In the case of a synchronous movement pattern, the arms usually stopped together before resuming oscillation with the probability of stopping at either extreme forward or backward position depending on the phase of perturbation. In the case of reciprocal pattern, the

FIG. 7. The influence of perturbation on arm movement and EMG activity of shoulder muscles during unilateral (left) and bilateral swinging (right). AD, PD, anterior and posterior deltoid muscles (R, right; L, left side). Long vertical lines: the time when the EMG activity of all recorded muscles was minimal; $s_f$ and $s_b$ indicate the respective wrist positions. Short vertical lines: the time when oscillations were re-initiated. Gray curves: projected wrist trajectories, as in Fig. 1B. EMG responses to perturbation (rectangular in A) are shown in more detail in Fig. 9.

EMG ACTIVITY

FIG. 8. Averaged EMG activity (RMS) of anterior deltoid (AD, □) and posterior deltoid (PD, ■) of perturbed (P) and nonperturbed (N) arms at different arm positions (session 1). [arrows], positions at which EMG was minimal compared with the activity at other points in the cycle.
choice of one of the two reciprocal positions of the arms for restarting oscillations was also probabilistic and phase-dependent.

**Phase resetting**

Because the perturbed arm reached a steady-state position after the nonperturbed arm thus delaying the re-initiation of swinging, the magnitude of the phase resetting was mainly defined by the behavior of the perturbed arm as supported by the finding that the distributions of phase shifts for uni- and bilateral swinging were similar (Fig. 4). For the same reason, the direction and position to which the perturbed arm moved after the arrest was defined by the previously reached position of the nonperturbed arm and by the required inter-limb coordination.

Our findings on phase resetting and phase-dependent responses to perturbation resemble previous findings in biological systems (Adamovich et al. 1994; Sternad et al. 2002). Phase resetting and phase-dependent responses to perturbation have also been observed during scratching in turtles (Stein and Grossman 1980). Adaptive reactions to perturbation of ongoing locomotion in decerebrated cats may be produced without any phase resetting, for example, in response to a moderate electrical stimulation of different descending systems or after a skin reflex when the dorsum of the foot touches the obstacle, producing limb flexion and walking over the obstacle (Drew and Rossignol 1984; Forssberg et al. 1975; Shik and Orlovsky 1976). In contrast, arrests of a hind limb during the flexor phase of gait in the decerebrated cat enhance the duration and amplitude of the flexor activity while perturbation applied during the extensor phase shortens the subsequent flexor phase, thus producing phase resetting (Duy sens and Pearson 1980; Lam and Pearson 2001). Phase resetting during locomotion in cats can be mediated by the motor cortex and cerebellum (Drew 1993; Russell and Zajac 1979). Arrests of the swinging leg in humans elicit bilateral rearrangements of EMG activity depending on the time when the arrested leg was released (Dietz et al. 1986). During human gait on a treadmill, when an obstacle suddenly appears in the beginning of the swing phase, the leg is flexed to elevate the foot and step over the obstacle (Schillings et al. 2000). If the obstacle appears later during the swing phase, the foot is rapidly placed on the ground to prevent stumbling and to maintain the body balance. Similar phase-dependent responses to perturbation were obtained by Forner Cordero et al. (2003), who used a rope to mechanically arrest the swinging leg at the level of the ankle. Although the maintenance of balance was not critical in the present study, both phase-dependent resetting and transitions of the limbs to appropriate steady positions had a similar functional meaning: rapidly restoring the ongoing motor process, in our case, the required pattern of single or bilateral swinging.

The dependency of responses on the time of perturbation in the cycle probably results from modulation and gating of proprioceptive and cutaneous reflexes by the CPG (Fais et al. 1999; Zehr and Kido 2001), i.e., from changes in reflex thresholds. It has been suggested that modulation of reflex thresholds may be inherent in many types of active movement, either discrete or rhythmical (Feldman and Levin 1995). In particular, the stretch reflex threshold has spatial dimension (see Introduction). By shifting reflex thresholds in spatial coordinates, the CPG may prevent posture-stabilizing mechanisms from resisting movement from a previously stabilized posture and, moreover, use these mechanisms for movement production (for details, see Feldman and Levin 1995; Ostry and Feldman 2003; Von Holst and Mittelstaedt 1973).

**Control of spatial and temporal characteristics of rhythmical movements**

The findings that unilateral perturbations produced substantial bilateral effects with phase resetting and re-initiation of swinging when both arms reached appropriate positions imply that muscles of both arms in the investigated tasks were controlled together as a coherent unit (Bernstein 1967; Schmidt et al. 1979). This occurred in spite of the fact that the control process might involve several neural structures—brain hemispheres (Donchin et al. 1998; Wiesendanger et al. 1996)—as well as spinal and brain stem circuits forming the CPG (Schaal et al. 2004; Smits-Engelsman et al. 2002; Zehr et al. 2004). Central and reflex-mediated interactions between muscles of the two limbs (e.g., Archambault et al. 2005; Nichols 1994) may be fundamental in converting two separate limbs into a functional unit so that a mechanical perturbation of one limb may nonmechanically influence the motion of the other limb as was the case in our study. By establishing mutually facilitatory or inhibitory reflex interaction between muscles of two limbs, the CPG or/and independent descending systems could elicit a desired pattern of bilateral swinging. Different discrete relations (e.g., 2:1) between the frequencies of moving limbs (Dietz et al. 1994; Serrien and Swinnen 1998; Shik and Orlovsky 1976; Stein et al. 1995; Von Holst 1973) may be produced by changing the pattern of inter-limb interaction.
The frequency and spatial boundaries of arm oscillations may be controlled by influencing the rate and range of changes in the threshold arm configuration, respectively. The temporal and spatial aspects of oscillations are likely controlled separately as indirectly follows from the observation that spatial but not temporal asymmetry in the arm movement may occur in patients with unilateral motor deficits due to stroke-related brain damage (Ustinova et al. 2006). If the vertical position was the position of equilibrium, then, arrested at this position, the arm would remain there for some time after being released. Instead, the arm sped up away from this position as soon as it was released (Fig. 2). Our data also show that there are two steady-state positions at which oscillations could terminate and resume. No substantial overshoots or terminal oscillations were observed when the arms arrived at these positions, implying that these positions were stable (see Figs. 1, B and C, and 2). Thus the data support the suggestion (Feldman 1980) that arm swinging results from rhythmic shifts in the equilibrium position of the system within well-defined forward and backward boundaries.

Initially, the level of co-activation of agonist and antagonist muscles of the nonperturbed arm at a boundary equilibrium position could be substantial (see Fig. 7E, EMGs of LAD and LPD). However, the same figure shows that before the swinging resumed, the activity of these muscles was reduced to a relatively low level while the position was maintained. This implies that the boundary equilibrium positions were close to the appropriate threshold positions of the arms at which all muscles might be silent in the absence of co-activation of agonist and antagonist muscles. Thus rhythmic resetting of the equilibrium configuration of the arms could result from central shifts in the reflex thresholds. The residual level of muscle activation at the boundary equilibrium positions (Figs. 6 and 8) appeared sufficient to balance the comparatively low torque of the arms at these positions until regular oscillations resumed.

The notion of threshold control implies that neural control levels only indirectly influence the EMG activity and muscle forces. Specific values of these variables emerge depending on the differences between the threshold and the actual positions of the arms (Archambault et al. 2005). The notion of threshold configuration is applicable to other multi-joint movements such as jumps and walking (Günther and Ruder 2003; St-Onge and Feldman 2004). In particular, Günther and Ruder (2003) simulated human walking in a planar model based on the transition between two threshold configurations of the body. Natural walking may also include a change in the threshold body configuration responsible for the transfer of weight from one side of the body to the other in each step.

One prediction of the threshold control hypothesis is that, in some movements, the actual and the threshold configurations of the body or its segments may match each other near the points of movement reversals. The matching will result in a minimization of the EMG activity of arm muscles near the points of movement reversal as observed for jumping and walking in place (St-Onge and Feldman 2004) and, in the present study, during both synchronous and reciprocal patterns of swinging (Figs. 7 and 8). The depth of this minimization likely depends on the residual level of co-activation of the opposing muscle groups.

In conclusion, our results show that a central generator of rhythmic arm movement produces transitions between two equilibrium arm positions without destabilizing either of them. These positions appear to be natural points at which the system may modify its behavior: to cease and either resume the oscillations or initiate a new motor action. One can hypothesize that the equilibrium positions, frequency, spatial boundaries, and bilateral coordination during arm oscillations may be controlled by changing the rate, extent, and the pattern of shifts in the threshold configuration of the bilateral system without direct involvement of neural control levels in the specification of muscle activity and forces. Our findings may be relevant to locomotion and suggest that walking may also be generated by transitions between several threshold configurations of the body, possibly accomplished by modulation and gating of proprioceptive and cutaneous reflexes.

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