Timing-Specific Transfer of Adapted Muscle Activity After Walking in an Elastic Force Field

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Blanchette A, Bouyer LJ. Timing-specific transfer of adapted muscle activity after walking in an elastic force field. J Neurophysiol 102: 568–577, 2009. First published May 6, 2009; doi:10.1152/jn.91096.2008. Human locomotion results from interactions between feedforward (central commands from voluntary and automatic drive) and feedback (peripheral commands from sensory inputs) mechanisms. Recent studies have shown that locomotion can be adapted when an external force is applied to the lower limb. To better understand the neural control of this adaptation, the present study investigated gait modifications resulting from exposure to a position-dependent force field. Ten subjects walked on a treadmill before, during, and after exposure to a force field generated by elastic tubing that pulled the foot forward and up during swing. Lower limb kinematics and electromyographic (EMG) activity were recorded during each walking period. During force field exposure, peak foot velocity was initially increased by 38%. As subjects adapted, peak foot velocity gradually returned to baseline in <125 strides. In the adapted state, hamstring EMG activity started earlier (16% before toe off) and remained elevated throughout swing. After force field exposure, foot velocity was initially reduced by 22% and returned to baseline in 9–51 strides. Aftereffects in hamstring EMGs consisted of increased activity around toe off. Contrary to the adapted state, this increase was not maintained during the rest of swing. Together, these results suggest that while the neural control of human locomotion can adapt to force field exposure, the mechanisms underlying this adaptation may vary according to the timing in the gait cycle. Adapted hamstring EMG activity may rely more on feedforward mechanisms around toe off and more on feedback mechanisms during the rest of swing.

INTRODUCTION

Human locomotion is a stereotyped movement resulting from the timely activation of numerous muscle groups (Carlssø 1972; Winter 1987). Neurophysiological studies have shown that the neural control of this movement involves tight interactions between feedforward (central commands from voluntary and automatic drive) and feedback (peripheral commands from sensory inputs) mechanisms. Both of these mechanisms contribute significantly to the muscle activation pattern expressed during walking (Bo Nielsen 2002; Dietz and Duysens 2000; Gray et al. 2007; Nielsen and Sinkjaer 2002; Patla 1996; Yang et al. 1991). Furthermore, central and peripheral commands to lower limb motoneurons are modulated as a function of the timing in the gait cycle (Bo Nielsen 2002; Capaday and Stein 1987; Duysens et al. 1992; Sinkjaer et al. 1996; Yang and Stein 1990; Zehr et al. 1997). Such modulation must therefore be included among the many variables involved in the neural control of walking.

Based on a protocol originally developed for reaching arm movements (Kawato 1999; Lackner and Dizio 1994; Sainburg et al. 1995; Shadmehr and Mussa-Ivaldi 1994; Wolpert et al. 1995), a few studies have recently investigated how subjects adapt their walking pattern when an experimentally controlled force environment (“force field”) is applied during the swing phase. As subjects started walking in the force field, they initially showed large movement deviations from baseline (Boyer et al. 2003; Emken and Reinkensmeyer 2005; Lam et al. 2006). Over the course of several strides, they then gradually learned to take into account the new force environment, and movement kinematics returned toward baseline due to modifications in the stereotypic muscle activations. When the force field was removed, movement aftereffects were present (Bouyer et al. 2003; Emken and Reinkensmeyer 2005; Lam et al. 2006; Noble and Prentice 2006). The latter resulted from modifications in muscle activation pattern that persisted once the force field was removed. In the absence of the force, these modifications could not be due to augmented feedback resulting from the action of the force field on the limb (feedback mechanism). They rather represented a change in central commands (feedforward mechanism).

Such a change in central commands has a potentially important impact for gait rehabilitation. For example, force field adaptation could be used as an implicit learning tool to train patients. With a properly tailored force field applied during walking, desired locomotor aftereffects could be planned to improve the patient’s gait. Aftereffect “tailoring” is currently supported by experiments performed during reaching in normal subjects (Patton and Mussa-Ivaldi 2004) and by mathematical modeling for walking (Liu and Reinkensmeyer 2004). However, before proceeding with the formal development of locomotor rehabilitation protocols based on aftereffects, an understanding of “how” the adapted muscle activation pattern is transferred to postexposure walking is warranted as the adapted pattern likely results from a combination of feedforward and feedback mechanisms. Indeed in an extreme case, if only feedback mechanisms were to be used in the adaptive process, the adapted muscle activation pattern would be expressed during force field exposure, but it would not be transferred to postexposure walking. To date, only the study of Lam et al. has reported electromyographic (EMG) activity during force field-induced locomotor adaptation (Lam et al. 2006). Using a velocity-dependent resistance applied against hip and knee movement, these authors compared the adapted muscle activity to catch trials (unexpected force removal during 1 stride).

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They concluded that adapted muscle activation patterns in rectus femoris and tibialis anterior activity during swing were controlled by feedback mechanisms, while modifications in biceps femoris and medial hamstring activity during preswing involved feedforward mechanisms. These results suggest that the mechanisms underlying force field adaptation during locomotion are either different from one muscle group to another and/or depend on the timing in the gait cycle where adaptation occurred.

The present study also investigated adaptation of the muscle activation pattern during force field exposure and its transfer to postexposure walking. Our hypothesis was that transfer of the adapted muscle activation pattern would not be constant throughout a stride, as central and peripheral commands to motoneurons during locomotion are differentially modulated depending on the timing in the gait cycle. To test this hypothesis, a position-dependent force field that modified the force environment over a wide portion of the gait cycle was used. Our specific objectives were to describe the modifications in the walking pattern during and after force field exposure, to relate the muscle activity aftereffects to the modifications that occurred in the presence of the force field, and to evaluate the effects of a delay between force field exposure and post-exposure walking on the expression of aftereffects. Part of these results have appeared in abstract form (Tremblay and Bouyer 2003).

**Methods**

**Subjects**

Ten healthy young adults (3 men) aged between 19 and 26 yr, drawn from the University student population, participated in this study (Table 1). All subjects read and agreed to a formal consent form describing the procedure and their involvement in the study. A general health condition questionnaire ensured that they had no known history of neurological or orthopedic disorders that could affect performance during the experiment. The study received approval from the local ethics committee.

**General protocol**

Each subject came to the laboratory for a single half-day visit. The experiment consisted of walking on a motorized treadmill at comfortable speed (range: 3.3–4.0 km/h) for three periods of 10 min. During each walking period, spatiotemporal gait parameters, right lower limb kinematics, and EMG activity from up to seven muscles were simultaneously recorded. The three walking periods were: before (control), during (adaptation), and after (recovery) exposure to a force field applied to the right lower limb. Between the walking periods, subjects stood still with their feet on either side of the moving belt. At the beginning of each walking period, once the proper belt speed was reached, they stepped on with their left lower limb first. Similarly, at the end of each walking period, they stepped aside before the belt was stopped. This procedure avoided differences in walking speed at treadmill start/stop.

In the presence of the force field, the instruction was “resist the force to walk normally.” Subjects were looking at a target located 12 feet ahead during walking. For 7 of the 10 subjects, postexposure walking occurred immediately after removing the force field. For the three others, postexposure walking was delayed by 10 min, a period during which they sat and avoided lower limb movement.

**Force field generation**

To create the position-dependent force field, a very simple method was used. It consisted of attaching elastic tubing between the foot of the subject and the front of the treadmill (Fig. 1A). This arrangement produced a force field that pulled the foot forward and up when the leg was unloaded but had little effects during the rest of the gait cycle due to limb loading by the subject’s weight.

Force intensity was related to foot antero-posterior position, being largest around toe off. In addition, elastic stretching during stance did not require active work from the subject but resulted from the backward movement of the loaded lower limb with the treadmill belt. Thera-band silver elastic tubing (Hygienic, Akron, OH) was chosen to create this force field for its material characteristics, including a stable force-generating potential for >5,700 cycles (Patterson et al. 2001). Figure 1B presents a typical force-stretching relationship for a 40-cm elastic tubing, i.e., the average elastic length used in the present study. The relationship follows a third-order polynomial (equation in Fig. 1B). Comparing the values obtained during elastic stretching (●) and shortening (○), it can also be seen that this material produces very low hysteresis.

The elastic tubing attachment points are shown in Fig. 1A. Briefly, one end of the tubing is attached to a nylon strap at the level of the metatarsophalangeal joint. The other end is attached to a custom-built anchor point located on the treadmill frame that could be adjusted mediolaterally such that the tubing always remained aligned with the foot in the sagittal plane. Walking with the elastic tubing attached in this configuration produced a force that changed in intensity and direction depending on foot position. This “force field” is represented by the arrows and gray area on Fig. 1.

In an effort to normalize the level of difficulty of the task across subjects, peak force field intensity was calibrated for each subject to ~40% of the maximal force produced voluntarily while standing on the left leg and pulling back as hard as possible with the right (experimental) leg off the ground. Foot position on the treadmill at toe off and force-stretching equations (e.g., Fig. 1B) were used to determine elastic length. Finally, to minimize fore-aft movements of the body (which could affect the force produced by the elastic), subjects were instructed to lightly touch the front handlebar of the treadmill with the tip of their fingers at all times.

**Recordings**

A load cell (Honeywell International) placed in series with the elastic recorded force intensity applied to the foot during the walking period in the force field. Custom-made foot switches were placed under the big toe and the heel of each foot to obtain the exact moment of heel contact and toe off. Three-dimensional right lower limb movements were measured using an Optotrack 3020 system (Northern Digital). Triads of small infrared emitting diodes (IRED) were placed on the pelvis and on the right thigh, shank, and foot. In addition, the

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**Table 1. Subject characteristics**

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Category</th>
<th>Age, yr</th>
<th>Gender</th>
<th>Weight, kg</th>
<th>Height, cm</th>
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<tr>
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<td>M</td>
<td>66.4</td>
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</tr>
<tr>
<td>S2</td>
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<tr>
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<tr>
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</tr>
<tr>
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<tr>
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<tr>
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<td>F</td>
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<td>165.0</td>
</tr>
<tr>
<td>S9</td>
<td>ND</td>
<td>22</td>
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<td>F</td>
<td>66.0</td>
<td>173.0</td>
</tr>
</tbody>
</table>

Subject characteristics (age, gender, weight, and height). Subjects that waited 10 min between force field and post-exposure walking are identified as “delayed” (D), while the others are labelled “non-delayed” (ND).
polynomial as detailed in the equation in elastic length used in the present study. This relationship follows a 3rd-order $B^3$ force-stretching relationship for a 40-cm elastic tubing i.e., the average their fingers during walking to prevent fore-aft movements of the body. were asked to lightly touch the front handlebar of the treadmill with the tip of backward movement of the loaded lower limb with the treadmill belt. Subjects stretching during stance did not require active work, but resulted from the produced by elastic tubing attached between the right foot and the frame of the treadmill. This arrangement generated a force that pulled the foot forward and up as shown schematically by the arrows. Force intensity was related to foot inter-electrode distance was ~2 cm. EMG signals were amplified and band-pass filtered (10–500 Hz) before being digitized on-line at 1,000 sample/s for each muscle for off-line analysis.

Data analysis

As each subject was tested before, during, and after exposure to the force field, they served as their own control. This experimental design was optimized to identify changes in the gait pattern during and after force field exposure. Force intensity at toe off was measured for each stride of the force field walking period. Mean and coefficient of variation were then calculated for each subject. Custom software written in MATLAB (MathWorks) was used to identify the exact moment of heel strike and toe off for each step based on footswitch signals. For each stride, peak foot velocity during swing was calculated by taking the modulus of the rate of foot displacement in the sagittal plane (i.e., the resultant from vertical and horizontal rate of displacement). Velocity data were then normalized to the mean baseline value. Using landmark coordinates, the relative angular positions at the hip, knee, and ankle were reconstructed off-line by the KINGAIT software (Mishac Kinetics). Data were synchronized on heel strike and time-normalized separately for stance (60% of stride duration) and swing (40% of stride duration). The gait cycle was then divided into 50 bins of equal width (2% of stride duration).

EMG signals were digitally filtered off-line with a zero lag fourth-order Butterworth filter (band-pass 20 – 450 Hz), rectified, and separated into individual strides synchronized on heel strike. To allow comparison across strides and walking periods, time normalization (60% stance, 40% swing) was applied to the data. EMG signals for each muscle were then divided into 50 bins of equal width (2% of stride duration) and mean amplitude over each bin reported. To allow comparison across subjects, amplitude normalization was performed by dividing each bin by the highest bin obtained during baseline walking (mean of last 100 strides). Zones of consistent changes across subjects were identified post hoc by superimposing group means for all three walking periods in each muscle. More detailed analyses were then performed over the identified zones (see Results).

Statistics

Statistical analyses were performed on amplitude and time-course data.

AMPLITUDE COMPARISONS. The following five epochs in the experiment were defined: 1) last 100 strides of the control walking period: Baseline ("B"); 2) first stride of the walking period in the force field: Initial effects ("IE"); 3) last 100 strides of the walking period in the force field: Adapted state ("AS"); 4) first stride of the walking period after force field removal: Initial aftereffects.
("AE"); and 5) last 100 strides of the walking period after force field removal: Baseline post ("BP").

Paired comparisons were made between baseline (B) and the four other epochs (IE, AS, AE, and BP) using nonparametric statistics (Wilcoxon signed-rank test) with each subject serving as its own control.

TIME-COURSE DETERMINATION. Time course was calculated based on an 11-point moving average. It was defined as the number of strides taken to reach the 95% confidence interval of the last 100 points collected during force field exposure for adaptation, and after force field removal for recovery.

RESULTS

Force intensity

The elastic calibration process showed a large intersubject variability in maximal force output (range: 193–297 N). As our goal was to create a force field applying ~40% of this maximal force with the elastic tubing, the force intensity therefore applied to the foot at toe off varied between 64 and 154 N depending on the individual. For a given subject however, force intensity variation at toe off over the 10-min exposure to the force field was minimal, as shown by coefficients of variation ≤3.9%.

Spatiotemporal gait parameters

Force field exposure did not affect stride duration during walking (B vs. IE: P = 0.193; B vs. AS: P = 0.432; B vs. AE: P = 0.432; B vs. BP: P = 0.322). However, proportions of swing and stance were modified in the presence of the force field. Swing proportion increased from 36% of the gait cycle during baseline to 42% (P = 0.002) during the first stride in the force field. By the end of force field walking, swing proportion had returned toward baseline (38% of gait cycle duration), but the difference remained statistically significant (P = 0.004). After removing the force field, there was no difference in swing proportion when compared with baseline (B vs. AE: P = 0.846; B vs. BP: P = 0.820). To prevent the changes in swing/stance proportion between some epochs from affecting other measurements, a time normalization to 40% of the gait cycle duration for swing and to 60% for stance was applied to the data presented in the following text.

Foot kinematics

INITIAL EFFECT AND ADAPTATION TO THE FORCE FIELD. Figure 2A shows the peak foot velocity during swing for each stride of the three walking periods in a representative subject (subject 1). First, it can be seen that stepping on the already moving treadmill belt did not affect peak foot velocity during swing in the baseline period, even for the first stride. During the force field walking period, however, peak foot velocity was initially augmented and then gradually returned toward control values. Figure 2B focuses on the five epochs analyzed and presents foot velocity separately for each subject. Comparing the first stride taken in the presence of the force field to baseline, the main effect for 9 of the 10 subjects was a significant increase in peak foot velocity during swing (range: 21–73%; P = 0.004). The 10th subject showed an initial foot velocity reduction of 19% resulting from a reduction in hip and knee excursion (data not shown). Once the subject realized that this reaction was inappropriate to produce normal walking, the strategy was abandoned. By the fourth stride, more natural angular excursions returned, and an increase in foot velocity of 12% was observed (data not shown).

By the end of the force field walking period (Fig. 2B; AS), foot velocity had returned to baseline (B vs. AS: P = 0.432). While all subjects adapted to the presence of the force field, strategies and time courses to reach the adapted pattern varied. Some subjects showed a systematic gradual decrease in foot velocity, while others presented more “exploratory” strategies that included a mixture of foot velocities above and below baseline. Considering foot velocity error in absolute terms, Table 2 shows that the number of strides to reach the adapted state was between <5 and 125 (5 strides being the resolution limit of the moving average method used to determine time courses; see METHODS).

AFTEREFFECT AND RECOVERY AFTER FORCE FIELD EXPOSURE. The step-by-step recovery in peak foot velocity (subject 1) can be observed in Fig. 2A when focusing on the data obtained during postelastic walking. After removing the force, Fig. 2B (AE) shows that the first step taken presented aftereffects, i.e., a significant decrease in peak foot velocity during swing. This decrease was observed in all subjects (range: 9–32% of baseline; P = 0.002). In addition, recovery strategies were similar across subjects, all showing a gradual return towards baseline. Number of strides to reach the postexposure baseline varied from 9 to 51 (Table 2).

DELAY VERSUS NO DELAY. Statistics were not used to compare subjects with and without delay between force field exposure and postexposure walking periods due to the limited sample size. However, results from the three subjects with a delay show that it took between 9 and 35 strides (Table 2) for peak foot velocity to return to baseline. These values were quite similar to those of subjects without a delay, ranging from 11 to 51 strides (Table 2). Furthermore, delayed subjects showed initial aftereffect amplitude within the inter-subject variability of the nondelayed subjects (Fig. 2B; AE).

Lower limb joint kinematics

Contrary to foot velocity, initial effect of the force field on lower limb joint kinematics varied across subjects. However, they all reached a similar adapted pattern. Figure 3 presents the relative angular positions across the hip, knee, and ankle joints (mean of all subjects) in the adapted state. Peak hip flexion during swing was similar to baseline (P = 0.301). During swing, peak knee flexion was reduced for 8 of the 10 subjects by a mean of 8° (P = 0.014). This peak occurred at the same timing in the gait cycle as during baseline walking, however. At the ankle, subjects reached a peak plantarflexion ~2° earlier. The amplitude of this peak was reduced by a mean of 14° (P = 0.002). In addition, an 11° dorsiflexion appeared at ~70% of the gait cycle. During the last 15% of swing, ankle position was back to baseline. The small error bars in Fig. 3 shows that these effects were systematic across subjects.

EMG activity

INITIAL EFFECT AND ADAPTATION TO THE FORCE FIELD. Initial effects of force field exposure on EMG activity varied across
subjects (data not shown). Yet they all converged to a similar adapted pattern as will now be described. Figure 4 shows the details of the adapted muscle activation pattern in comparison to baseline (mean of all subjects). The main and most consistent change across subjects occurred in medial and lateral hamstring between 42 and 80% of stride, i.e., from 20% before toe off to 20% after. In the adapted state, hamstring activity started before the end of the stance phase i.e., 40% earlier than during baseline walking, and reached a plateau near toe off. The amplitude of this plateau is similar to the peak activation during baseline walking for MH and slightly higher for LH. This elevated activity was maintained until the end of the baseline locomotor burst.

Figure 5 focuses on the mean EMG amplitude of LH and MH between 42 and 80% of stride. The increase in activity in this section of the gait cycle was large and significant (B vs. AS; LH: $P = 0.016$ and MH: $P = 0.008$). It must be noted that increased hamstring activity was already observed in the first step taken in the force field but to varying degrees across individuals (data not shown). Detailed observation of both panels in Fig. 5 shows that the increase in activity at AS was observed in all subjects recorded and was not necessarily similar in these two agonists for a given subject. Returning to Fig. 4, it can be seen that this hamstring activation was not accompanied by a concomitant increase in antagonist muscles such as VL and RF. However, increases in VL and RF EMG were sometimes observed at the end of stance (30–60% of stride). These modifications were less consistent across subjects, however, and were therefore not further analyzed.

Regarding muscles acting mainly around the ankle joint, significant changes were absent in TA and small in MG and SOL. A small burst of activity appeared in MG during early swing (56–74% of stride). In SOL, a decrease in peak amplitude was also observed near the end of stance (44–48% of stride).

INITIAL AFTEFFECT FOLLOWING FORCE FIELD EXPOSURE. Once the force field was removed, hamstring EMG did not immediately return to baseline. Figure 6A shows that in the first stride after force field removal, an additional burst of activity appeared for both of these muscles within the zone of interest.
activity was not maintained until the end of the normal burst. Instead, a significant decrease (Fig. 8; *) was observed during swing starting at 74% of stride for LH and 62% of stride for MH.

**DISCUSSION**

In the present study, individual locomotor patterns were recorded before, during and after a 10-min exposure to a force field applied to the foot. By comparing muscle activation patterns obtained at different epochs in this experimental protocol, some of the mechanisms underlying the neural adaptation to the presence of the force field and its transfer to postexposure walking can be inferred.

**Initial exposure to the force field**

When subjects took their first step with the elastic tubing attached to their right foot, the result was a perturbed swing movement, presenting a large increase in peak foot velocity. Comparing EMG activity between initial effect and baseline showed that hamstrings activation was augmented (B vs. IE; LH: \(P = 0.016\) and MH: \(P = 0.008\)). Therefore as early as on the first step, subjects had already changed their muscle activation pattern.

**TABLE 2. Time courses of changes in peak foot velocity and hamstring EMG**

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Category</th>
<th>Lateral Strides</th>
<th>Medial Strides</th>
</tr>
</thead>
<tbody>
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</tr>
<tr>
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<td>10</td>
</tr>
<tr>
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<td>ND</td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td>S4</td>
<td>D</td>
<td>66</td>
<td>14</td>
</tr>
<tr>
<td>S5</td>
<td>D</td>
<td>38</td>
<td>9</td>
</tr>
<tr>
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<td>ND</td>
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</tr>
<tr>
<td>S7</td>
<td>ND</td>
<td>40</td>
<td>9</td>
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<td>&lt;5</td>
<td>59</td>
</tr>
</tbody>
</table>

Individual time courses were calculated using an 11-point moving average. Values represent the number of strides before reaching the 95% confidence interval calculated on the last 100 points collected during force field exposure for adaptation, and after force field removal for recovery. D: delayed; ND: non-delayed; EMG, electromyography; Missing data for lateral hamstring: S4, S5, S8 and for medial hamstring: S3, S4, S7.

(42–80% of the gait cycle). This extra burst in LH reached a peak amplitude at 64% of the gait cycle. For MH, the peak amplitude was located at 54% of the gait cycle. Figure 6B shows that the difference with baseline between 42 and 80% of the gait cycle was significant (B vs. AE; LH: \(P = 0.016\) and MH: \(P = 0.008\)). Once again, increases in activity were not necessarily similar in these two agonists for a given subject.

**RECOVERY AFTER FORCE FIELD EXPOSURE**

MH EMG activity for the first 50 strides after force field removal is plotted in Fig. 7 for one representative subject (S7). It can be seen that aftereffects persisted for several strides.

Aftereffects time courses for LH and MH are presented for all subjects in Table 2. Number of strides before reaching the baseline post ranged between 8 and 65 strides for LH and between 9 and 59 strides for MH. Interestingly, the amplitude of initial aftereffects and number of strides before returning to a baseline were not necessary similar for lateral and medial hamstrings in a given subject (Table 2).

**DELAY VERSUS NO DELAY**

EMG activity from LH and MH were not always available from the same subject due to technical problems during the experiment. Nevertheless, it can be seen from the limited data that initial aftereffect amplitude and aftereffect time course for the subjects with delay were within the range of non-delayed subjects. These results suggest that the delay did not modify the aftereffects. This information complements the foot velocity data obtained in all three delayed subjects (see preceding text).

**COMPARISON BETWEEN ADAPTED STATE AND INITIAL AFFTEREFFECT**

To compare initial aftereffect to the adapted EMG, Fig. 8 shows AS and AE hamstring activations superimposed. Aftereffect in hamstring EMG consisted of a large burst that began at the same timing as the adapted burst. This aftereffect burst (44–72% of stride for LH and 44–60% of stride for MH) was not statistically different from the adapted burst at the same timing. Contrary to the adapted burst, however, the elevated
Considering that the instruction to the subjects was to "resist the force to walk normally," several neural mechanisms could have influenced this immediate increase in EMG activity. First, as subjects were aware of the presence of the elastic tubing, feedforward control could have been modified based on naïve expectation of how the new forces should be applied on the lower limb. Second, by pulling on the foot, the elastic tubing modified the sensory afferent pattern produced during walking. For example, by pulling the foot forward, the force field changed the expected muscle elongation/shortening pattern. This likely changed spindles afferent discharge (Taylor et al. 2006). In addition, by pulling the foot up, the force field tended to reduce loading of the experimental leg and to shift body weight to the other leg more rapidly at the end of stance. This likely changed Golgi tendon organ and plantar skin afferent discharge (Dietz and Duysens 2000; Loeb et al. 1977). Knowing that the neural control of locomotion involves feedback mechanisms (af Klint et al. 2008; Dietz and Duysens 2000; Gray et al. 2007; Nielsen and Sinkjaer 2002; Yang et al. 1991), this modified afferent pattern likely participated in the reshaping of muscle activations through positive feedback loops (Gray et al. 2007; Prochazka et al. 1997a,b; Yang et al. 1991).

However, while the EMG data indicate a rapid modification in muscle activation pattern as soon as the force field was introduced, such mechanism did not cancel out the perturbation, as shown by the large increase in peak foot velocity present during swing (B vs. IE: P = 0.004).

**Adaptation to the force field**

With repeated stepping in the force field, subjects adapted as shown by a gradual return of swing phase foot velocity to baseline (B vs. AS: P = 0.432). As the force intensity variation at toe off was minimal over the 10-min exposure (CV ≤ 3.9%), the reduction in foot velocity therefore had to involve a change in the muscle activation pattern. Adaptation took ≤125 strides (Table 2). While, to our knowledge, adaptation to position-dependent force fields during locomotion has not been previously investigated, this time course is similar to what has been reported in comparable studies that used velocity-dependent (Emken and Reinkensmeyer 2005; Lam et al. 2006) or acceleration-dependent (Noble and Prentice 2006) force fields.
While foot velocity returned to baseline, from a global lower limb kinematic perspective, the adapted and control patterns were not identical. Indeed, in the adapted state, the ankle joint was kept dorsiflexed throughout swing, instead of reaching a peak around 15° of plantarflexion after toe off and then gradually returning toward a neutral position around heel strike (cf. Fig. 3). The fact that ankle angular position did not return to baseline may be due to a movement optimization to the force field environment. In reaching experiments, it has been shown that adapted movement trajectories can be predicted using a minimum cost algorithm, suggesting that subjects are trying to minimize movement cost rather than to completely cancel out movement deviation from baseline (Isawa et al. 2008). Similarly, in a study focusing on constrained walking that used several treadmill speed–step frequency combinations, Bertram (2005) showed that minimization of metabolic cost appears to dominate walking control in such a situation. The latter suggests that movement optimization principles would also apply.

![Figure 6](http://jn.physiology.org/)

**FIG. 6.** Initial aftereffect for LH and MH EMG activity. **A:** initial aftereffect (thick gray line) compared with baseline (thin black line) on rectified and filtered LH (n = 7) and MH (n = 7) EMG activity. Error bars correspond to SE. Data were time-normalized to 60% for stance and 40% for swing. Data were also amplitude-normalized to the peak locomotor EMG measured during baseline. **B:** initial aftereffect compared with baseline on LH and MH mean EMG amplitude between 42 and 80% of the gait cycle for each subject. Opened-gray symbols represent subjects who sat and avoided lower limb movement for 10 min between force field and postexposure walking. Missing data for LH: S4, S5, S8 and for MH: S3, S4, S7. Asterisk, P < 0.05.

![Figure 7](http://jn.physiology.org/)

**FIG. 7.** Stride-by-stride aftereffects in MH EMG activity. Rectified and filtered MH EMG activity for the 1st 50 strides after force field removal (subject S1). Data were time-normalized to 60% for stance and 40% for swing. Left vertical line corresponds to toe off, and right to heel strike.

![Figure 8](http://jn.physiology.org/)

**FIG. 8.** LH and MH EMG activity comparisons between adapted state and initial aftereffect. Initial aftereffect (thick gray line) compared the adapted state (thick black line) on rectified and filtered LH (n = 7) and MH (n = 7) EMG activity. Error bars correspond to SE. Data were time-normalized to 60% for stance and 40% for swing. Data were also amplitude-normalized to the peak locomotor EMG measured during baseline. Asterisk, P < 0.05.
to walking movement control. In the present study, canceling out ankle angular position deviations from baseline during force field exposure would have required activation of ankle plantarflexors during swing to actively resist the force and bring the ankle into plantarflexion. Such a strategy would have been more costly from a metabolic point of view.

By comparing adapted EMG activity to baseline, Fig. 4 shows that hamstrings (hip extensor/knee flexor) were by far the most modified of the seven recorded muscle groups. EMG analysis therefore focused on these muscles. Hamstring muscle activity, normally present only around heel strike, now started before toe off and was maintained until the end of the baseline burst. This extra activity was not accompanied by a coactivation of the antagonists VL and RF. As the hip is flexing and the knee extending during mid to late swing (cf. Fig. 3), part of this increased hamstring EMG activity likely produced an eccentric contraction used to reduce hip flexion and/or knee extension velocity. Further studies will have to include a biomechanical analysis and a mathematical model to better understand how hamstrings are involved in the fine dynamic control of the hip and knee angular excursions during force field walking. This was beyond the scope of the present study.

Contrary to the first stride in the force field that showed different initial effects across individuals, once adapted, all subjects used similar kinematic and EMG patterns (Figs. 3 and 4). This “single solution” adopted in the adapted state suggests that there is an important benefit to the use of such a pattern and could be supported by the movement cost optimization theory.

**Initial effects following force field removal**

When subjects took a first step after the force field was removed, aftereffects were observed, expressed as a significant decrease in peak foot velocity during swing when compared with baseline (Fig. 2). Comparison of initial aftereffect in hamstring EMG activity with baseline showed a new burst of activity around toe off. Considering that walking occurred in the same force environment as at baseline, the muscle activation pattern reshaping could not be due here to a modified afferent input pattern produced by the force field. However, the afferent input pattern could potentially have been modified as a result of muscle spindle habituation. Indeed in the present experiment, repetitive eccentric contractions of hamstrings were produced by the subjects during force field walking. This oversolicitation of the muscle could have produced spindle habituation (Jackson et al. 2009). In turn, habituation could cause aftereffects due to the temporarily altered firing pattern produced by the habituated receptors. If this was the case, time between force field exposure and postexposure walking would become an issue. With a delay of 5–7 min between force field and postexposure walking, spindles would have enough time to recover (Jackson et al. 2009); as a result, no aftereffects would be observed in subjects having been delayed.

To evaluate if spindle habituation was an important component of our aftereffects, the present experimental design included three subjects that were purposely delayed by 10 min before they were allowed to start the postexposure walking. Results showed the presence of aftereffects in foot velocity and EMG activity for the three delayed subjects. The initial amplitude of aftereffects was similar to that observed in nondelayed subjects. A delay of 10 min was chosen here based on the fact that the nondelayed group had all returned to baseline by that time. If habituation or other unidentified time-related processes were involved in the generation of aftereffects, they would have been washed out by then. The presence of aftereffects even after a delay between force field exposure and postexposure walking cannot therefore be explained by sensory habituation or other time-related processes. These results rather suggest that aftereffects resulted from a modification in feedforward mechanisms, i.e., from central movement recalibration (e.g., Kawato 1999; Lackner and DiZio 2000).

Furthermore, considering that the instruction to the subjects was to “resist the force to walk normally,” the fact that such aftereffects were present even when participants were aware of elastic removal suggests that the feedforward control of walking has been modified beyond volition by the 10-min force field exposure.

**Recovery following force field removal**

With repeated stepping after force field removal, foot velocity gradually returned to baseline (range: 9–51 strides; Table 2). This time course of recovery is comparable to what others have obtained in similar studies (Lam et al. 2006; Noble and Prentice 2006). In addition, for the three delayed subjects, time course of recovery was similar to that observed in nondelayed subjects.

Considering now the muscle activation pattern, the extra activation around toe off gradually disappeared in 8–65 strides for LH and in 9–59 strides for MH, demonstrating here also that aftereffects were not simply due to stepping onto the treadmill but to an actual movement recalibration that now had to be recalibrated back to the baseline pattern. These EMG time courses are similar to those reported in another study looking at the recovery after velocity-dependent force field exposure (Lam et al. 2006). In the latter study, baseline was reached after 40 strides for biceps femoris (here referred to as lateral hamstring) and after 30 strides for MH. It can be noted that these authors also reported differences in time courses between LH and MH.

**Timing-specific transfer of the adapted hamstring activation pattern**

The fact that the same subjects were tested before, during and after force field exposure allowed another comparison between muscle activation patterns to be made: adapted state with initial aftereffects (AS-AE). In this comparison, the contribution of modified feedforward control and of positive feedback to the adapted muscle activation pattern can be inferred. Similarities between AS and AE muscle activation patterns are considered as transfers of the adapted state, originating from modifications in feedforward control. Conversely, differences between AS and AE muscle activation patterns are considered to be associated with feedback contribution. Comparison between these two epochs is illustrated in Fig. 8 and shows that the initial aftereffect in hamstring EMG activity was not statistically different from the adapted burst between 44 and 72% of the gait cycle for LH and between 44 and 60% for MH. In this part of the gait cycle, feedforward mechanisms therefore dominated.
For the remaining of the swing phase, AS and AE muscle activation patterns were significantly different. AS activity did not therefore completely transfer to AE. As the main difference between initial aftereffect and adapted state is the presence/absence of the force field, the lower transfer of activity suggests that feedback mechanisms dominated for this muscle in mid to late swing. This difference in transfer as a function of timing occurred despite similar level of muscle activation in the adapted state, thereby eliminating differences due to motoneuron excitability between these two parts of the gait cycle as a possible cause for the difference.

Taken together, these results suggest that the adapted hamstring muscle activation pattern during walking in the elastic force field resulted from timing-specific modifications in motor command, involving more feedforward contribution around toe off and more feedback contributions during mid- to late swing.

Conclusion

Our results suggest that for hamstrings, the transfer of adapted muscle activity varies as a function of timing in the gait cycle. The impact of this finding goes beyond increasing our knowledge of the neural control of locomotion: functionally, understanding the rules of muscle activation transfer is extremely important if efficient gait rehabilitation protocols based on force field aftereffects are to be designed. Further experiments will now be required to better understand, at least for all key locomotor muscles across the lower limb, the relationship between transfer capacity and timing in the gait cycle.

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