Contribution of Afferent Feedback to the Soleus Muscle Activity During Human Locomotion

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Mazzaro, Nazarena, Michael J. Grey, and Thomas Sinkjær. Contribution of afferent feedback to the soleus muscle activity during human locomotion. J Neurophysiol 93: 167–177, 2005. First published September 8, 2004; doi:10.1152/jn.00283.2004. During the stance phase of the human step cycle, the ankle undergoes a natural dorsiflexion that stretches the soleus muscle. The afferent feedback resulting from this stretch enhances the locomotor drive. In this study a robotic actuator was used to slightly enhance or reduce the natural ankle dorsiflexion, in essence, mimicking the small variations in the ankle dorsiflexion movement that take place during the stance phase of the step cycle. The soleus (SOL) and tibialis anterior EMG were analyzed in response to the ankle trajectory modifications. The dorsiflexion enhancements and reductions generated gradual increments and decrements, respectively, in the ongoing SOL EMG. We exercised care to ensure that the imposed ankle movements were too slow to elicit distinct burst-like stretch reflex responses that have been investigated previously. The increased SOL EMG after the dorsiflexion enhancements was reduced when the group Ia afferents were blocked with peripheral ischemia at the thigh, and during high-frequency Achilles tendon vibration. However, neither ischemia nor tendon vibration affected the decrements in the SOL EMG during the dorsiflexion reductions. These findings give evidence of the contribution of afferent feedback to the SOL activity in an ongoing basis during the stance phase. The results suggest that mainly feedback from the group Ia pathways is responsible for the increments in the SOL EMG during the dorsiflexion enhancements. However, the decrements in the SOL activity might be mediated by different afferent mechanisms.

INTRODUCTION

In an attempt to investigate the importance of afferent feedback during human walking, several research groups have measured the muscle response to rapid perturbations of the foot and/or leg (Dietz et al. 1985, 1987; Sinkjær et al. 1996; Yang et al. 1991). Yang et al. (1991) used a pneumatic device to apply rapid dorsiflexion perturbations in the early stance phase. As a result, burst-like stretch reflex responses, defined as a short-latency reflex with a synchronized large-amplitude response, were elicited in the SOL muscle. It was concluded that the observed reflex response contribute to 30–60% of the SOL EMG during the stance phase of human walking. Sinkjær et al. (1996) confirmed this conclusion and extended these observations to show that the SOL stretch reflex response is modulated during the gait cycle, being higher at the mid stance phase. These studies demonstrated the importance of afferent feedback mediating compensatory reflex responses to unexpected external perturbations; however, the extrapolation of these results to normal unperturbed walking must be interpreted cautiously. It might be necessary to differentiate the role of afferent feedback during such corrective reactions, and the contribution of afferent feedback to the ongoing muscle activation during unperturbed walking. During normal unperturbed walking other neural mechanisms might be implicated (Nielsen and Sinkjær 2002; Yang et al. 1991). Recent evidence suggests that the afferent feedback that is generated during unperturbed walking and the afferent feedback that signals an unexpected destabilizing perturbation may be centrally processed differently depending on the frequency components of the feedback signal (Morita et al. 1998).

One way to demonstrate the contribution of afferent feedback to the background SOL EMG during walking is to remove the feedback, and observe the effect of this removal on the EMG activity. Sinkjær et al. (2000) hypothesized that by arresting the ankle dorsiflexion or applying a fast plantarflexion movement during the stance phase, the ankle extensor muscles would be unloaded, and the afferent feedback from these muscles would decrease. Indeed, after the unload movements, a decrease of about 50% is measured in the SOL EMG, suggesting that afferent feedback from group II muscle spindles and/or tendon receptors contribute to the SOL activity during the stance phase of walking.

The aim of the present study was to investigate the afferent feedback–mediated adjustment of the SOL activity in the stance phase of normal human walking. We applied small-amplitude, slow-velocity modifications to the ankle trajectory during the stance phase of the gait cycle to mimic the variations on the ankle movement that normally occur during human walking. The slow ankle trajectory modifications would therefore simulate the modifications in sensory feedback that might occur during unperturbed walking on flat or slightly uneven surfaces. The protocol involved controlled enhancements and reductions of the natural ankle dorsiflexion during the stance phase of the gait cycle. We hypothesized that, if sensory feedback contributes to the enhancement of the plantar flexor muscle activation during walking, the slow dorsiflexion enhancements and reductions would result in coincidental increments or decrements in the ongoing SOL EMG. A semiportable robotic actuator was used to apply the slow ankle trajectory modifications. We exercised care to ensure that the dorsiflexion enhancements were too slow to produce the distinctive burst-like stretch reflex responses that are typical of fast perturbations (Dietz et al. 1987; Sinkjær et al. 1996; Yang et al. 1991). Peripheral ischemia and sustained Achilles tendon vibration were applied together with the ankle trajectory mod-

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Modulation of the SOL response in the mid and late stance phase

To study the SOL response to the dorsiflexion enhancements on different parts of the stance phase, in 12 volunteers the same level of dorsiflexion was applied 200–300 ms after heel contact (mid stance phase), and 150–200 ms later (late stance phase), and maintained for 150–250 ms. The amplitude and velocity of the perturbations were chosen as the minimum dorsiflexion level that evoked an increment in the SOL EMG (about +5 deg s\(^{-1}\); ±2 deg).

Ischemic block of the group Ia afferent pathways

In 8 subjects peripheral ischemia was combined with dorsiflexion enhancements or reductions to evaluate the contribution of the group Ia afferent pathways to the SOL EMG. In this protocol, the SLR and the medium-latency stretch reflex (MLR) responses, mediated by the group Ia and II pathways, respectively (Allum and Budingen 1979; Corina et al. 1995; Diener et al. 1983; Dietz et al. 1987; Nasher 1976; Schieppati et al. 1995; Toft et al. 1991), were used to monitor the ischemic block.

Before the application of ischemia, fast dorsiflexion perturbations were applied during walking to elicit a stretch reflex response. Next, the actuator was programmed to apply one level of slow dorsiflexion enhancement or reduction during the stance phase.
Ischemia was applied by positioning a pneumatic cuff about 10 cm above the left knee and inflating it to a pressure of 220 mmHg while the subjects were seated with their knees flexed at 90 deg. During the first 15 min, while the subject remained seated, the progress of the ischemic block was determined by applying 5–10 fast ankle dorsiflexion perturbations every 2 min and measuring the amplitude of the SLR. When the amplitude of the SLR decreased to about 25% of the initial value, the subjects were asked to walk with the pneumatic cuff inflated and still in place. During walking, the monitoring was performed by applying fast dorsiflexion perturbations every 6–8 steps, and measuring the SLR response. When the SLR decreased to about 15% of the initial value, the group Ia fibers was considered blocked, and the same slow dorsiflexion enhancement or reduction applied before ischemia was repeated. In addition fast dorsiflexion perturbations and control steps were recorded randomly.

The experiment was stopped when the subjects had difficulty walking or when the MLR response started to decrease (Grey et al., 2001). Amplitudes of the SLR and MLR responses were restored about 10 min after the cuff was deflated.

Achilles tendon vibration

High-frequency tendon vibration is a powerful stimulus for the group Ia afferents (Burke et al., 1976a; Roll and Vedel, 1982; Roll et al., 1989). In this study Achilles tendon vibration was combined with slow dorsiflexion enhancements and reductions to investigate the contribution of the Ia pathways to the SOL EMG during the stance phase.

Achilles tendon vibration during sitting. It was previously shown that the group Ia-mediated response is depressed during the application of tendon vibration (Schieppati et al., 2001). Therefore we initially evaluated the effect of constant Achilles vibration on the Ia pathways, by comparing the SOL SLR response before and during tendon vibration, in the sitting position. A custom-designed, high-pressure, hydraulic actuator (MTS-Systems 215.35, 230 bar) (Voigt et al., 1999) was used to deliver fast dorsiflexion perturbations during sitting, to elicit a stretch reflex response in the SOL muscle. An in-house–fabricated portable vibrator was used to deliver the vibratory stimulus. The vibrator consists of a DC motor with an eccentric rotating mass, embedded in a plastic tube (2.5 cm diameter, 5 cm long), with a total weight of 150 g. During the experiment the subjects were sitting with the right knee flexed about 20 deg and the right hip flexed about 80 deg. The right foot was firmly strapped to a foot adapter on the hydraulic actuator, ensuring that the anatomical axes of the ankle of the subject coincided with the center of rotation of the actuator. The subjects were instructed to hold a constant 5% maximum voluntary contraction (MVC) plantar flexion against a footplate; visual feedback was provided through an oscilloscope. Initially, a series of 25 fast dorsiflexion perturbations (600 deg s\(^{-1}\); 8 deg) were applied to elicit a control profile of the SLR. Next, the vibrator was positioned transversally to the tendon and tightly fixed by means of an elastic ankle support. The Achilles tendon was constantly vibrated at 110 Hz and the fast dorsiflexion perturbations were repeated.

Achilles tendon vibration during walking. In subjects in whom the SOL SLR response was decreased during the application of vibration in the sitting position, the test was repeated during walking. Nine subjects were instrumented with a portable ankle actuator and the portable vibrator on the left leg. The ankle actuator was programmed to impose fast dorsiflexion perturbations, and the SOL responses without and during Achilles tendon vibration were compared. If the amplitude of the SLR during vibration was reduced to ≥60% of the value without vibration, the slow dorsiflexion enhancements and reductions were applied without and during constant tendon vibration.

Off-line data analysis

Signal processing and analysis were carried out off-line. The EMG records were rectified and filtered with a 40-Hz first-order, low-pass filter to extract an amplitude envelope. The SOL EMG response to the slow dorsiflexion enhancements and reductions was visually examined for any evidence of SLR response, and all trials that showed such a response were removed from the analysis. In all cases, no more than 5–10 trials were removed from the analysis. The muscle activity was calculated as the area under the EMG signal from the beginning to the end of the perturbations. Changes in SOL EMG, resulting from the dorsiflexion enhancements and reductions, were calculated as the difference in activity between perturbed and control steps, and normalized with respect to the background muscle activity. For the protocol with constant-amplitude enhancements, the muscle activity was calculated as the mean EMG value within the dorsiflexion window. In this case the increments were normalized with respect to the same control value. Similarly, when the enhancements were imposed in the mid and the late stance phases, the increments in SOL EMG were normalized with respect to the same control activity. In this case the control activity was measured from the onset of the mid stance perturbation to the end of the late stance perturbation.

The velocity of the ankle movements were expressed as the difference (increment or decrement) with respect to the normal dorsiflexion velocity in (deg s\(^{-1}\)).

Statistical analysis

Linear regression analyses were used to test the relationship between the changes in the SOL activity and the velocity of the imposed ankle dorsiflexion movements. In this protocol where enhancements with the same and different amplitudes were applied, the slopes of both linear regression analyses were compared. A one-way repeated-measures (RM)–ANOVA test was used to evaluate the response to the enhancements applied in the mid and late stance. Paired Student’s t-tests were applied to compare the responses to the slow perturbations without and during ischemia. A 2-way RM-ANOVA test was used to analyze the effect of ankle perturbations and Achilles tendon vibration in the SOL activity during control steps, stretch reflexes, and slow ankle perturbations. The results of all statistical tests were considered significant at the P < 0.05 level. Results are presented as means ± SD unless otherwise indicated.

Results

The dorsiflexion enhancements and reductions were applied in the mid to late stance phase, about 200–400 ms after heel contact when the natural dorsiflexion increases gradually. The reason that the perturbations were not presented earlier in the stance phase is that shortly after heel contact the ankle joint undergoes a rapid transition from plantarflexion to dorsiflexion. Any dorsiflexion enhancement applied in this early phase of the stance would have to be fast, with the consequence of eliciting a burst-like stretch reflex response, which we were taking care to avoid. The ankle trajectory modifications were almost imperceptible to the subjects, possibly because the imposed changes in the dorsiflexion amplitude and velocity were in the range of the normal variability of the ankle movement during the stance phase of walking.

Relationship between the ankle dorsiflexion and the SOL EMG

The normal ankle dorsiflexion was enhanced or reduced during the stance phase, and the influence in the ongoing SOL EMG was tested. Figure 1 is an example of a typical data record for one subject, with each trace representing an ensemble average of 30 trials. In this case, the subject walked at 3 km h\(^{-1}\) and the natural ankle dorsiflexion was enhanced or reduced...
for a period of 300 ms, starting about 350 ms after heel contact (Fig. 1A). The velocity and amplitude of the natural ankle dorsiflexion within the selected time window were 33 deg s⁻¹ and 10 deg, respectively. Although 8 different ankle trajectories modifications were applied to this subject, only 4 sets are shown for clarity. The dorsiflexion enhancements and reductions that are shown have approximately the following velocities and amplitudes, respectively: ±6 deg s⁻¹; ±2 deg and ±16 deg s⁻¹; ±5 deg. After the perturbation, the ankle was released and it returned to its normal trajectory after about 100 ms. Figure 1B shows the corresponding ensemble average of the rectified and filtered SOL EMG record. Before the perturbations, the SOL EMG of the control and modified steps were similar, but during the perturbations, the EMG increased or decreased when the natural dorsiflexion was enhanced or reduced, respectively. The SOL EMG during the dorsiflexion enhancements and reductions changes smoothly and there are no abrupt changes similar to that observed with rapid perturbations. The TA EMG was unchanged in the perturbed trials compared with the controls (Fig. 1C), and thus it was not further analyzed. Figure 1D illustrates the percentage changes in SOL EMG in response to the 8 different perturbations applied to this subject.

Figure 1E shows the linear regression analysis for each subject (gray dotted lines) and across all subjects (black solid line, n = 12), between the percentage changes in SOL EMG and the velocity of the imposed dorsiflexions. For every subject, the slope of the linear regression was significantly greater than zero. The regression slope of the group analysis (0.87% per deg s⁻¹, r = 0.85) is significantly different from zero (P < 0.0001), indicating that the variation in the SOL EMG is linearly related to the velocity and/or amplitude of the perturbations. The point corresponding to the control step (0%, 0 deg s⁻¹) was not included in the regression analysis but was tested to be within the 95% confidence interval, as would be expected.

The increment in SOL EMG is primarily velocity sensitive

Two different sets of dorsiflexion enhancements were applied to explore the amplitude and velocity sensitivity of the observed increments in the SOL EMG. Figure 2 shows example data for a single subject. In this case the subject walked at 3.4 km h⁻¹; 12 ankle perturbations were applied, but only 6 are shown for clarity. Figure 2A shows averaged data of the ankle trajectory during control steps and dorsiflexion enhancements.

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**FIG. 1.** Example of an averaged data record (30 trials) for a typical subject during control steps and dorsiflexion enhancements and reductions. A: ankle angular position during control steps (thick line) and dorsiflexion enhancements and reductions (thin lines) are shown superimposed. The velocity and amplitude of the ankle dorsiflexion during control steps were 33 deg s⁻¹ and 10 deg, respectively. Dorsiflexion enhancements and reductions were applied 350 ms after heel contact and maintained for 300 ms. Imposed perturbations had approximately the following velocities and amplitudes: ±6 deg s⁻¹; ±2 deg and ±16 deg s⁻¹; ±5 deg. B: rectified and filtered SOL EMG during the corresponding control and perturbed steps. C: TA EMG during the control and perturbed steps. D: percentage changes in SOL EMG for the 8 perturbations applied to this subject. Hatching patterns in A–D correspond with each other. E: individual (gray dotted lines) and group linear regression analyses (n = 12, black line) between changes in the dorsiflexion velocity and percentage changes in the SOL activation.
The velocity and amplitude of the normal ankle dorsiflexion within the selected time window were 17 deg s$^{-1}$ and 5 deg, respectively. The dorsiflexion enhancements were applied 310 ms after heel contact and maintained for 200–280 ms. In the series shown on the left have different velocities and amplitudes: +6 deg s$^{-1}$, +2 deg; +13 deg s$^{-1}$, +4 deg; and +26 deg s$^{-1}$, +8 deg, respectively. In the series shown on the right, the final amplitude of all dorsiflexions is 6 deg, but the velocities are different: +20, +26, and +30 deg s$^{-1}$. B: corresponding rectified and filtered SOL EMG. Hatching patterns in A and B correspond with each other. C: regression analyses of both protocols superimposed. Symbol (●) corresponds to dorsiflexion enhancements with different amplitudes and velocities, and (○) corresponds to dorsiflexion enhancements with different velocities but same amplitude. Slopes of the regressions were not statistically different from each other ($P = 0.6$).

The velocity and amplitude of the normal ankle dorsiflexion within the selected time window were 17 deg s$^{-1}$ and 5 deg, respectively. The dorsiflexion enhancements were applied 310 ms after heel contact and maintained for 200–280 ms. In the series shown on the left the velocity and amplitude of the dorsiflexions are +6 deg s$^{-1}$, +2 deg; +13 deg s$^{-1}$, +4 deg; and +26 deg s$^{-1}$, +8 deg, respectively. In the series shown on the right, the final amplitude of all dorsiflexions is 6 deg, but the velocities are different: +20, +26, and +30 deg s$^{-1}$. When the ankle was released it returned to its normal trajectory within about 100 ms. Figure 2B shows the corresponding ensemble average of the rectified and filtered SOL EMG. Before the dorsiflexions, the SOL EMG of control and modified steps are similar, although the SOL EMG increased when the ankle dorsiflexion was enhanced. After the ankle was released the EMG slowly decreased and returned to the level of the control EMG. In Fig. 2C, the linear regression analyses for the same subjects of both protocols are plotted superimposed. The regression lines show the relationship between the percentage change in SOL EMG and the velocity of the perturbations. For this subject the dorsiflexions with different amplitudes and velocities had a regression slope of 0.78% per deg s$^{-1}$ ($r = 0.92$), and the protocol of dorsiflexions with the same amplitude but different velocities had a regression slope of 0.60% per deg s$^{-1}$ ($r = 0.95$).

Across all subjects ($n = 12$), the dorsiflexions with different amplitudes and velocities had a slope 1.2 ± 0.4% per deg s$^{-1}$ ($P = 0.004$), and the dorsiflexions with the same amplitude but different velocities had a slope 0.71 ± 0.2% per deg s$^{-1}$ ($P = 0.0006$). A comparison between the 2 slopes showed that they were not significantly different from each other ($P = 0.1$).
Equal SOL EMG response in the mid and late stance phases

Slow dorsiflexion enhancements were applied in the mid and late phases of the stance to investigate whether the contribution of afferent feedback to the SOL EMG is modulated during different parts of the stance phase. Figure 3 is a typical data record for a subject walking at 3.2 km h\(^{-1}\). In this case the slow enhancements were applied 330 and 500 ms after heel contact (mid and late stance phases), and maintained for 180 ms. Within the corresponding time windows the normal amplitude and velocity of the ankle dorsiflexion were 4 deg and 22 deg s\(^{-1}\), respectively. The applied dorsiflexion enhancement had amplitude of 3 deg, and a velocity of 16 deg s\(^{-1}\) (see Fig. 3A).

Figure 3B shows the corresponding ensemble averaged and filtered SOL EMG signals. The SOL EMG during the perturbations increased with respect to the control EMG in both cases, the mid and late stance phases.

Figure 3C shows the percentage increments in SOL EMG for mid (23.6 ± 7%) and late (22.6 ± 7%) stance phases across all subjects (\(n = 12\)). In both cases the increments in SOL EMG were greater than zero, but not significantly different from each other (one-way RM-ANOVA; \(P = 0.58\)).

Contribution of the group Ia to the SOL EMG

Peripheral ischemia was combined with slow dorsiflexion enhancements and reductions to evaluate the contribution of group Ia afferents to the SOL EMG. Figure 4A shows typical data of one subject of the ankle trajectory and SOL EMG during control steps and fast dorsiflexion perturbations, before and during the ischemic block. Note that before the ischemic block, the onset of the SLR was evident and clearly defined, appearing 50 ms after the stretch and lasting about 20 ms. Typically the amplitude of the SLR started to decrease about 15 min after the cuff inflation, and it decreased to <15% of the initial value about 18–20 min after the cuff inflation. Figure 4B shows the amplitudes of the SLR and MLR responses across all subjects (\(n = 8\)) before and during ischemia. A paired t-test showed a significant difference (\(P = 0.027\)) in the amplitude of the SLR before and during the ischemic block (79.5 ± 14 and 27.8 ± 7 \(\mu\)V, respectively). However, the MLR response was not significantly different before and during the block (93 ± 13 and 56.5 ± 10 \(\mu\)V, respectively, \(P = 0.1\)). Figure 4, C–F, shows typical data of 2 subjects of the ankle trajectory and SOL EMG during control steps and dorsiflexion enhancement and reductions, before and during ischemia. Figure 4C shows the ankle trajectory profile when ischemia was combined with dorsiflexion enhancements. In this example the subject walked at 2.8 km h\(^{-1}\). The velocity and amplitude of the normal ankle dorsiflexion within perturbation window were 32 deg s\(^{-1}\) and 8 deg, respectively. The enhancement (+20 deg s\(^{-1}\); +5 deg) was applied 430 ms after heel contact and maintained for 250 ms. The SOL EMG corresponding to the control and perturbed steps are shown in Fig. 4E. The SOL EMG increased after the dorsiflexion before the application of ischemia. However during ischemia there was no increase in the SOL activity.

Figure 4G shows percentage increments in the SOL EMG across all subjects (\(n = 8\)) during the dorsiflexion enhancements. A Student’s paired t-test showed that the responses to the dorsiflexion enhancements before (30.3 ± 7%) and during ischemia (3.5 ± 3%), were significantly different from each other (\(P = 0.007\)).

Figure 4D shows a case where ischemia was combined with dorsiflexion reductions. In this example the subject walked at 3 km h\(^{-1}\), and the normal dorsiflexion velocity and amplitude within the perturbation window were 36 deg s\(^{-1}\) and 9 deg, respectively. The dorsiflexion reduction (−20 deg s\(^{-1}\); −5 deg) was applied 210 ms after heel contact and maintained for 250 ms. Figure 4F shows that the SOL EMG activity decreased during the dorsiflexion reductions in both cases, before and during the ischemic block.

Figure 4H shows percentage decrements in the SOL EMG during dorsiflexion reductions across all subjects (\(n = 8\)). A Student’s paired t-test showed that the percentage reductions in EMG before (−16.3 ± 5%) and during ischemia (−11.5 ± 4%) were not statistically different from each other (\(P = 0.1\)).

Constant Achilles tendon vibration was combined with dorsiflexion enhancements and reductions to evaluate the contribution of group Ia afferents to the SOL EMG. Initially the effect of tendon vibration on the group Ia was evaluated during sitting. Figure 5A shows typical raw data of a subject of the
FIG. 4. Example of an averaged data record for a typical subject during fast dorsiflexion perturbations and slow dorsiflexion enhancements and reductions, before and during peripheral ischemia. A: ankle trajectory and SOL EMG during control steps and fast dorsiflexion perturbations before (black) and during (gray) ischemia. B: peak-to-peak increment in the SOL SLR and MLR responses before and during ischemia. The SLR was decreased during ischemia (\(P = 0.027\)), although the MLR was not significantly affected (\(P = 0.1\)). C and D: ankle trajectory during control steps and dorsiflexion enhancements (\(+20 \text{ deg s}^{-1}; +5\) deg) and reductions (\(-20 \text{ deg s}^{-1}; -5\) deg) before and during ischemia. E and F: SOL EMG during control steps (thick black line) and dorsiflexion enhancements and reductions (thin gray lines), before and during ischemia. G and H: percentage change in SOL EMG after dorsiflexion enhancements and reductions, before and during ischemia across all the subjects (\(n = 8\)). Ischemia significantly affected the response to the dorsiflexion enhancements (\(P = 0.007\)) but not to the dorsiflexion reductions (\(P = 0.1\)). Hatching patterns in C–H correspond with each other.
ankle trajectory and SOL SLR during sitting without and during Achilles tendon vibration. In 9 subjects the amplitudes of the SLR without and during vibration (0.8 ± 0.2 and 0.35 ± 0.2 mV, respectively) were significantly different (P = 0.002). A 2-way RM-ANOVA test showed that during walking, both the ankle perturbations and tendon vibration had a significant effect on the SOL EMG (P < 0.01), and there was a significant interaction between both factors. The SOL SLR responses without and during vibration (54.3 ± 12 and 32.9 ± 8 μV, respectively) were significantly different from each other (P = 0.003). However, the MLR responses without and during vibration (54.6 ± 11 and 44.8 ± 6 μV, respectively) were not significantly different (P = 0.2) (Fig. 5B).

Raw data of 2 subjects of the ankle trajectory and SOL EMG during control steps and dorsiflexion enhancements and reductions without and during Achilles tendon vibration are shown in Figure 5, C–F. The subject shown in Fig. 5C walked at 2.8 km h⁻¹, and the velocity and amplitude of the normal ankle dorsiflexion within the defined time window were 32 deg s⁻¹ and 8 deg, respectively. The dorsiflexion enhancements (+7 deg s⁻¹; +2 deg) were applied 330 ms after heel contact and maintained for 270 ms. For the case shown in Fig. 5D the dorsiflexion reductions (−18 deg s⁻¹; −4 deg) were applied 400 ms after heel contact and maintained for 210 ms. Figure 5G shows that the SOL EMG during control steps with and without tendon vibration (31.7 ± 7 and 35 ± 7 μV s⁻¹, respectively) were not significantly different from each other (P = 0.125). During the dorsiflexion enhancements, the SOL EMG without and during tendon vibration were significantly different from each other (14.4 ± 4 and 10.5 ± 3%, respectively, P = 0.023) (see Fig. 5H). However, the decrements in the SOL EMG during dorsiflexion reductions applied without and during tendon vibration were not significantly different from each other (−12.5 ± 6 and −12.6 ± 7%, respectively; P = 0.912) (Fig. 5I).

D I S C U S S I O N

The aim of this study was to investigate the afferent feedback–mediated adjustments of the SOL muscle activation during the stance phase of human walking. Our experimental paradigm was designed to modify the proprioceptive feedback from ankle extensor muscles by slightly enhancing and decreasing the normal ankle dorsiflexion during the stance phase, thus mimicking the normal variability of the ankle movement during walking on flat or slightly irregular surfaces. The imposed dorsiflexion enhancements and reductions generated gradual increments and decrements, respectively, on the ongoing SOL EMG. The SOL EMG increments resulting from the dorsiflexion enhancements were more sensitive to the velocity of the ankle movement than to its amplitude, and were similar in the mid and late stance phases of the step cycle. In addition ischemia and Achilles tendon vibration depressed the increments on the SOL EMG during the dorsiflexion enhancements, but did not affect the decrements on the SOL activity in response to the dorsiflexion reductions. This study provides evidence that afferent feedback contributes to generate adaptive modifications on the SOL activity during walking. Moreover the results from ischemia and Achilles tendon vibration indicate that increments on the SOL activity may be sensitive to feedback from the group Ia pathways, whereas different afferent mechanisms might mediate the decrements during the dorsiflexion reductions.

Slow dorsiflexion enhancements and reductions

In this study, slow dorsiflexion enhancements and reductions (±40 deg s⁻¹) generated gradual increments and decrements on the SOL EMG of ±45%. In previous studies fast dorsiflexion perturbations have been applied during the stance phase of the step cycle, to analyze the contribution of afferent feedback to the SOL activity during walking. For example, Yang et al. (1991) applied dorsiflexion perturbations of ±100 deg s⁻¹ in the early stance phase and reported increments of 30–60% on the SOL EMG. Later, Sinkjær et al. (1996) applied faster dorsiflexion perturbations (250 deg s⁻¹) in different parts of the gait cycle and reported increments of 50–90% on the SOL EMG during the stance phase. In both studies, the rapid ramp-shaped perturbations elicited distinct burst-like responses in the SOL EMG. In contrast, the slow dorsiflexion enhancements used in the present study generated gradual increments in the SOL activity that extended to the entire range of the perturbation.

It is likely that the afferent feedback, associated with small-amplitude, slow-velocity variations of the normal ankle trajectory, mediate adaptations in the muscle activity differently than during fast and large unexpected perturbations (Nielsen and Sinkjær 2002). The slow dorsiflexion enhancements and reductions applied in the present study may modify the sensory feedback in a similar manner to what might happen during normal walking over flat or uneven ground. In contrast, the fast dorsiflexion perturbations that have been used in previous studies might generate an “error-like” signal mediating corrective reflexive responses to these unexpected external events. Morita et al. (1998) proposed that afferent information is processed differently depending on the frequency of the sig-
nals. Therefore the low-frequency sensory information during normal walking might be processed differently by the CNS than the high-frequency bursts of signal associated with fast and large unexpected perturbations. These two inputs might thus generate different outputs despite that the same pathways might be involved.

**Afferent pathways mediating the SOL EMG adaptations**

The dorsiflexion enhancements and reductions, respectively, increase and decrease the natural lengthening of the ankle extensor muscles during the stance phase of the step cycle. It is thus very likely that the output from muscle spindle and Golgi tendon organs increased and decreased, respectively, compared with a normal step, contributing to the observed changes in the SOL activity.

The response to the dorsiflexion enhancements was not modulated, at least in the mid and late stance phases of the gait cycle. In contrast it is known that the SOL stretch reflex (Dietz et al. 1985; Sinkjær et al. 1996; Yang et al. 1991) and the H-reflex (Capaday and Stein 1987) are modulated during the stance phase of the step cycle. For example, Capaday and Stein (1987) reported that the H-reflex is relatively low at the time of heel contact, increases progressively during the stance phase, and reaches its maximum amplitude late in the stance phase. Sinkjær et al. (1996) reported that the stretch reflex reaches its maximum in the mid stance, and decreases at the late stance.

Morita et al. (1998) proposed that the stretch reflex is less sensitive to presynaptic inhibition than is the H-reflex and thus the 2 mechanisms are modulated differently. They suggested that the motoneuronal pool might react differently to a highly synchronized input, such as the electrical stimuli used to elicit an H-reflex response, compared with a less-synchronized input, such as the mechanical perturbation needed to elicit a stretch reflex response. A similar justification can explain our observation that the response to the slow enhancements was not modulated during the stance phase of the gait cycle. Feedback associated with the slow trajectory modifications applied in the present study is less synchronized than feedback associated to fast perturbations; thus these 2 responses might be modulated differently.

The regression curves for the dorsiflexions with different amplitudes and velocities, and the constant amplitude dorsiflexions were not significantly different. This result suggests that, although the velocity of the imposed dorsiflexion enhancements was slow, the increment in the SOL EMG was more sensitive to the velocity than to the amplitude of the imposed dorsiflexions. Although it is well known that the spindle primary endings are more sensitive to velocity than are the spindle secondary endings, both endings respond to amplitude and velocity of the muscle stretch and have the potential to contribute to the observed response (Matthews and Stein 1969).

Peripheral ischemia and Achilles tendon vibration were applied to investigate the contribution of the group Ia pathways to the observed changes on the SOL EMG during the slow perturbations. Surprisingly, both maneuvers affected the response to the dorsiflexion enhancements but not to the reductions, suggesting that these adaptations in the muscle activity may be mediated by different afferent mechanisms.

During the imposed dorsiflexion enhancements the amplitude and velocity of the muscle-tendon complex stretch is greater than during a normal step; thus it is expected that muscle spindle and Golgi tendon organs (GTO) from the stretched muscles increase their firing rate. However, it has been shown that spindle primary afferent pathways (group Ia) are more sensitive to muscle stretch than are spindle secondary and GTO afferent pathways (Burke et al. 1976a,b). Therefore the observed increments in the SOL EMG may be mostly mediated by feedback from the group Ia pathways. Consequently, when peripheral ischemia and Achilles tendon vibration were applied, this response was largely affected. However, because peripheral ischemia differentiates afferent fibers based on their diameter and in humans there is very likely considerable amount of overlap in the diameter of the groups Ia and Ib fibers (Burke et al. 1983), it is not possible to separate the contribution of these 2 fiber types with this technique. Moreover, because smaller fibers are blocked with time we cannot exclude the possibility that the largest of the group Ib and II fibers were also blocked at the end of the protocol, and that they also contribute to the observed increment in the SOL EMG.

During the dorsiflexion reductions, however, the robotic actuator slowed down the natural ankle joint movement and reduced the stretch of ankle extensor muscles. It has been shown that during small-amplitude, slow-velocity sinusoidal muscle stretch, the Ia pathways are more sensitive than the group II pathways during the stretch phase of the imposed movement, although they become completely silent during the unloading phase (Kakuda 2000). The group II fibers, however, follow the sinusoidal load increasing and decreasing its firing rate during all the cycle, even during the unloading phases when the Ia pathways are silent. During the dorsiflexion reductions applied in the present study, although the ankle extensors were still eccentrically stretched, the perturbation feedback from the other pathways may become more relevant during this response. As a result, peripheral ischemia and Achilles tendon vibration did not affect the response to the dorsiflexion reductions. This finding is consistent with the results of Sinkjær et al. (2000) that the decrease in the SOL EMG when the ankle dorsiflexion was arrested during the stance phase of the gait cycle was not affected by peripheral ischemia.

Achilles tendon vibration did not affect the SOL activation during control steps. A similar result was reported by Courtainé et al. (2001), by applying bilateral Achilles tendon vibration during over ground walking. These results suggest that feedback from the group Ia afferent pathways may not contribute importantly to the background SOL activation, whereas feedback from other pathways may play a more significant role.

With the techniques applied in this study, we cannot rule out whether other than the group Ia afferents are involved in the regulation of the background SOL EMG during walking. For example, it has been proposed that load feedback from Ia afferent pathways reinforces the activation of muscles during walking in humans (Dietz and Duysens 2000; Duysens et al. 2000), and cats (Conway et al. 1987; Gossard et al. 1994; Hiebert and Pearson 1999; Pearson et al. 1992). It is also possible that feedback from cutaneous receptors could contrib-
ute to the responses observed in this study. However, cutaneous affere
nts have not been shown to contribute to the stretch reflex response produced by a rapid dorsiflexion perturbation (Grey et al. 2001) or the unload response produced by a rapid plantar flexion perturbation (M. J. Grey, M. Ladoucette, J. B. Andersen, J. B. Nielsen, and T. Sinkjær, unpublished observations). Despite the results suggest that the observed adaptive modifications in the SOL activity are very likely mediated by proprioceptive input rather than cutaneous input, the role of feedback from cutaneous receptors should be assessed in future studies.

In conclusion, previous studies have demonstrated that when an unexpected fast dorsiflexion perturbation disrupts the normal ankle trajectory during the stance phase, a burst-like short-latency reflex response is elicited in the SOL muscle (Dietz et al. 1987; Sinkjær et al. 1996; Yang et al. 1991). We suggest that feedback signaling this kind of extreme fast and large unexpected perturbations is processed differently than the expected proprioceptive information during normal walking, and thus the latter must be investigated differently. The application of slow and small ankle trajectory modifications, almost imperceptible to the subject, may be a better technique to study the importance of afferent feedback during normal human walking. The slow dorsiflexion enhancements and reductions applied in the present study slightly deviate the ankle from its average trajectory, generating small variations in the background SOL EMG. The results suggest that continuous contribution of afferent feedback to the muscle activity may automatically adjust the muscle activation to meet external demands of the walking surface.

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