Ankle extensor proprioceptors contribute to the enhancement of the soleus EMG during the stance phase of human walking

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Abstract: A rapid plantar flexion perturbation applied to the ankle during the stance phase of the step cycle during human walking unloads the ankle extensors and produces a marked decline in the soleus EMG. This demonstrates that sensory activity contributes importantly to the enhancement of the ankle extensor muscle activation during human walking. On average, the EMG begins to decline approximately 52 ms after the perturbation. In contrast, a rapid dorsiflexion perturbation produces a group Ia mediated short-latency stretch reflex burst with an onset latency of approximately 36 ms. The transmission of sensory traffic from the foot and ankle was suppressed in 10 subjects by an anaesthetic nerve block produced with local injections of lidocaine hydrochloride. The anaesthetic block had no effect on the stance phase soleus EMG, the latencies of the EMG responses, or the magnitude of the EMG decline following the plantar flexion perturbation. Therefore, it is more likely that proprioceptive afferents, rather than cutaneous afferents, contribute to the background soleus EMG during the late stance phase of the step cycle. The large difference in onset latencies between the short-latency reflex and unload responses suggests that the largest of the active group Ia afferents might not contribute strongly to the background soleus EMG, although it remains to be determined which of the proprioceptive pathways provide the more important contributions.

Key words: afferent feedback, gait, locomotion, stretch reflex.

Résumé : Une perturbation rapide de la flexion plantaire appliquée à la cheville durant la phase posturale du cycle de la marche chez l’humain décharge les extenseurs de la cheville et produit une diminution importante de l’EMG du soléus. Ceci démontre que l’activité sensorielle contribue de façon importante à l’augmentation de l’activation du muscle extenseur de la cheville durant la marche chez l’humain. En moyenne, l’EMG commence à diminuer environ 52 ms après la perturbation. En revanche, une perturbation rapide de la dorsi-flexion produite une boucle réflexe du groupe Ia à latence courte, avec une période de latence de l’attaque de 36 ms. La transmission du trafic sensoriel à partir du pied et de la cheville a été supprimée chez 10 sujets par un blocage nerveux par anesthésie avec des injections locales d’hydrochloraide de lidocaïne. L’anesthésie n’a pas eu d’effets sur la phase posturale de l’EMG du soléus, sur les temps de latence des réponses EMG ou sur l’amplitude du déclin de l’EMG suite à une perturbation de la flexion plantaire. Ainsi, il est plus probable que les afférences proprioceptives plutôt que les afférences cutanées contribuent à l’EMG de base du soléus durant la phase posturale de cycle de la marche. La grande différence dans les temps de latence des attaques entre le SLR et les réponses de décharge suggèrent que les afférences du groupe Ia ne contribuent pas fortement à l’EMG de base du soléus, quoiqu’il reste à déterminer lequel des sentiers proprioceptifs fournit les contributions les plus importantes.

Mots clés : rétroaction afférente, démarche, locomotion, réflexe myotatique.

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Introduction

Sensory feedback from peripheral afferents contributes to the control of walking by modulating the basic motor programs and (or) the output from these programs, by controlling phase transitions, and by reinforcing the ongoing locomotor muscle activity (Pearson 1993, 2003). In human experiments, the role of afferent feedback in the control of walking has been studied by applying electrical nerve stimulation (Capaday 1997; Pierrot-Deseilligny 1997; Stein et al. 1993; Zehr and Stein 1999) or mechanical joint perturbations (Dietz et al. 1987; Sinkjær et al. 1996; Yang et al. 1991). In general, the rationale for this type of study has been that an electrical or mechanical perturbation would enhance the natural afferent traffic, thus providing insight about the natural afferent feedback during unperturbed walking. Based on the observation that rapid dorsiflexion stretch-evoked short-latency reflex (SLR) responses (e.g., Dietz et al. 1987; Sinkjær et al. 1996; Yang et al. 1991) and electrically evoked H-reflex responses (e.g., Capaday and Stein 1986; Faist et al. 1996) are modulated during walking, it was concluded that the large-diameter spindle afferents contribute significantly to the enhancement of plantar flexor muscle activity during the stance phase of normal walking.

However, it is becoming increasingly clear that the results from paradigms using electrical nerve stimulation or rapid mechanical joint perturbations must be interpreted very cautiously (Nielsen and Sinkjær 2002a, 2002b). The complexity of the underlying mechanics and neural circuitry are such that it is not straightforward to make conclusions about the control of normal locomotion using techniques that evoke rapid and synchronous bursts of afferent traffic to the spinal cord. It has recently been proposed that the stretch reflex response observed in the ankle extensors after a rapid, unexpectedly imposed dorsiflexion perturbation may represent a neural corrective response or modification of the neural program rather than a “simple” feedback-mediated enhancement of the background extensor muscle activity (Nielsen and Sinkjær 2002a, 2002b; Sinkjær et al. 2001).

An alternative method to investigate the role of afferent feedback during motor control tasks is to remove the feedback. Sinkjær et al. (2000) removed the afferent feedback during the early- to midstance phase of walking by rapidly plantar flexing the ankle with a robot actuator (Andersen and Sinkjær 1995). In response to this perturbation, they observed a transient drop in the soleus EMG of approximately 50%. Because the soleus muscle is monoarticular, such a rapid plantar flexion must unload the Achilles tendon and (or) soleus muscle and temporarily decrease the output of musculotendon proprioceptors. The reduced proprioceptive feedback was thought to produce the transient decline in soleus EMG. The “unload response”, as termed by Sinkjær et al. (2000), is effectively the same as the “unload reflex” that was extensively studied in muscles of the hand and arm (e.g., Angel et al. 1965, 1973; Angel 1987; Angel and Weinrich 1986; Burke et al. 1978; Merton 1951). Angel et al. (1973) reported that the reduction in triceps brachii activity observed after its sudden release during a voluntary contraction was not affected by an anaesthetic block of the biceps brachii. Sinkjær et al. (2000) observed the same result in the soleus muscle following a plantar flexion perturbation with an anaesthetized tibialis anterior muscle. These observations demonstrate that the decline in muscle activity cannot be explained by reciprocal inhibition.

An imposed dorsiflexion stretches the soleus muscle and produces a corrective burst in the soleus EMG that is commonly called the stretch reflex response. The afferent feedback that contributes to this response is well described in the literature. However, the afferent feedback that contributes to the enhancement of the stance phase soleus EMG may be different or be processed differently than the stretch reflex response. This afferent feedback is removed by a rapid plantar flexion that unloads the Achilles tendon and (or) soleus muscle; hence, we use the term “unload response” to describe the drop in soleus muscle activity that follows an imposed plantar flexion perturbation.

While Sinkjær et al. (2000) argued that the unload response could most likely be attributed to an unloading of the ankle extensors, they did not rule out the possibility that the response could be attributed to cutaneous afferents or proprioceptors in the foot. The objective of the present study was to explore which pathway(s) are involved in the afferent-mediated feedback that contributes to the enhancement of ankle extensor muscle activity during the late stance phase of the step cycle. Dorsiflexion and plantar flexion perturbations of the ankle joint were presented in the latter part of the stance phase while able-bodied human subjects walked on a treadmill. These perturbations produced a stretch reflex and unload response, respectively, in the soleus EMG. Previously, we provided evidence that the short-latency and medium-latency stretch reflex responses to a dorsiflexion perturbation receive strong contributions from the spindle groups Ia and II afferents, respectively (Grey et al. 2001, 2002). By directly comparing the onset latency of the stretch reflex and unload responses in the same group of subjects, we can begin to illuminate which afferent pathways might contribute to the enhancement of the ankle extensor muscle activity during the stance phase of the step cycle. To determine if the unload response is affected by cutaneous afferents or proprioceptors in the foot, we compared the response before and after an anaesthetic block of the foot.

Methods

Thirty healthy volunteers (20 males and 10 females) between the ages of 22 and 38 with no history of neuromuscular disorder participated in this study. The subjects gave their informed consent prior to their participation and the local ethics committee approved the experimental protocol. The experiments were conducted in accordance with the Declaration of Helsinki.

Apparatus and instrumentation

The subjects were instrumented with bipolar surface EMG electrodes (Neurileve 720; Medicotest A/S, Denmark) over the soleus and tibialis anterior muscles of the left leg. The EMG signals were amplified and bandpass filtered from 20 to 1000 Hz. Dorsiflexion and plantar flexion perturbations were imposed while the subjects walked on a treadmill (Powerjog EG30; Sport Engineering Ltd., Great Britain) using a custom-built robotic actuator. Essentially, the device is a functional joint connected to a powerful AC servomotor.
via flexible Bowden cables. The functional joint was tightly fixed to the subject’s left leg with a polypropylene plaster cast, aligned coaxially with the ankle joint. The robot was operated under PID control through a personal computer to provide a freely moving interface during unperturbed walking and an imposed dorsiflexion or plantar flexion rotation at any desired time during the step cycle. Complete details of the mechanics of this device are reported elsewhere (Andersen and Sinkjær 1995). The ankle angular position was measured with an optical encoder incorporated within the functional joint. The ankle angular velocity was determined offline by differentiating this record. All data were sampled at 2 kHz.

Protocol
Prior to data collection, each subject walked at a comfortable self-selected speed (typically 3.5–4 km/h) for an adaptation period of approximately 5 min. The robotic actuator was then programmed to deliver a rapid plantar flexion or dorsiflexion ramp and hold perturbations (6°, 500°/s, 150-ms hold time) to the ankle during the later half of the stance phase. The delivery of the perturbations was timed so that the desired electromyographic responses occurred at about the time that the soleus muscle activity was greatest, approximately 60% into the stance phase. Data were recorded for 1200 ms starting 600 ms before the perturbation. Dorsiflexions, plantar flexions, and control steps were presented pseudo-randomly (every four to seven steps) until 25–30 trials were recorded for each condition.

In 12 of these subjects, the transmission of cutaneous afferents from the foot and ankle was blocked by an injection of lidocaine hydrochloride solution (250–500 mg) around the nerves supplying the skin of the foot and ankle (saphenous, deep peroneal, superficial peroneal, sural, and posterior tibial). The efficacy of the block was evaluated by testing the subjects’ sensation from the skin as well as somatosensory-evoked potentials (SEPs). Sharp and dull skin sensation was tested with the point and the hub of the needle. When the subject could no longer sense touch with the needle, stroking, or moderate pressure, the SEP procedure was performed. Surface electrical stimulation was applied to 3 areas of the foot (anterior, mediodorsal, and laterodorsal aspects) covering the 3 main sensory areas of the foot. A series of 500 stimuli were delivered at 2 Hz to each of these areas at an intensity corresponding to 2 times the paraesthetic threshold. The EEG was recorded using a single vertex electrode (bandpass 0.05–500 Hz, sampling rate 2 kHz, bilateral ears-referenced). SEPs were recorded for each of the 3 stimulation sites and compared with pre-block measures. The block was considered effective when the subject lost sensation from all areas of the skin on the foot and ankle and the SEPs were depressed. The subject was then instructed to walk on the treadmill and the ankle was perturbed as before. Data records were recorded before and after the nerve block to ensure the efficacy of the anaesthesia throughout the data collection.

Data analysis
Signal processing and analysis were carried out offline. The EMG records were rectified and filtered with a 40-Hz first-order low-pass filter to extract an amplitude envelope. The individual data for a particular trial were then ensemble averaged to produce a single record for each subject and type of perturbation. Onset latencies of the soleus electromyographic responses were determined by visual inspection using a moveable cursor on the display. The onset latency was defined as the first major deflection in the EMG record within a specific window for each response: SLR 30–45 ms, medium-latency reflex (MLR) 60–80 ms, and unload response 30–80 ms. Data were excluded from the analysis if the onset responses were not clearly identifiable within ±2 ms. All statistical tests were conducted with a significance level of 0.05 and all results are shown as means ± SD.
Fig. 2. Stretch reflex and unload response onset latencies produced by dorsiflexion and plantar flexion perturbations, respectively. (A) Mean onset times for the short-latency reflex (SLR), medium-latency reflex (MLR), and unload responses across all subjects; (B) the individual data for each subject indicate that the unload response onset latency (triangles) occurs between that of the SLR (solid circles) and MLR (open circles) responses ($p < 0.001$).

Results

A typical set of ensemble-averaged data (27 records) for one subject is shown in Fig. 1. In this case, the subject was walking at 3.5 km/h and the perturbations were applied at 360 ms following heel contact. The control step (thick line) is shown superimposed over the perturbed steps (thin lines). The perturbation onset is defined as time zero and is indicated with a vertical line through each record. The ramp times were 29 and 30 ms, respectively, for the dorsiflexion and plantar flexion perturbations. In both cases, the ankle was held for 200 ms and then released, returning to its natural position in the step cycle within 150 ms (Fig. 1a).

The dorsiflexion perturbation produced SLR and MLR responses in the soleus EMG (Fig. 1b) with onset latencies of 32 and 78 ms, respectively. No response is evident in the tibialis anterior EMG following the dorsiflexion perturbation. In contrast, the plantar flexion produced a depression in the soleus EMG with an onset latency of 55 ms and a very small response in the tibialis anterior EMG approximately 30 ms after the perturbation (Fig. 1c).

As noted in the Methods section, a strict tolerance of ±2 ms was used in the determination of the onset latencies.

This strict inclusion criterion was met for all but 4 subjects. The mean onset latencies for the soleus dorsiflexion and plantar flexion responses of the remaining 26 subjects are shown in Fig. 2a and summarized in Fig. 2b. The onset latency of the unload response occurred between that of the SLR and MLR onsets for all but one subject. Across all subjects, the dorsiflexion perturbation produced SLR and MLR responses with mean onsets of $37 \pm 5$ and $74 \pm 6$ ms, respectively, while the plantar flexion produced a drop in the soleus EMG at $54 \pm 11$ ms. A one-way repeated-measures analysis of variance test indicated that the difference between the onset latencies of the 3 responses was significant ($p < 0.0001, F_{2,25} = 224.84$). Scheffé’s multiple-comparison test showed that all of the response latencies were different from one another ($p < 0.001$).

Lidocaine hydrochloride was used in 12 subjects to block the neural traffic from the cutaneous afferents of the foot and ankle. In 2 subjects, the anaesthetic did not produce an effective block. In both cases, the sensation to touch was lost but the SEP was not significantly depressed; consequently, we did not proceed with the postblock recordings for these subjects. Across the remaining 10 subjects, the anaesthesia produced a depression in the SEPs of $68\%$ ($p < 0.001$), indicating that the afferent transmission from the foot was strongly depressed. All subjects reported that they felt they were able to walk normally. As shown in Fig. 3, the anaesthesia had no effect on the ankle trajectory or muscle activation patterns.

The effect of the anaesthesia on the unload response is shown in Fig. 4. Despite the effectiveness of the anaesthetic block, there was no change in either the onset latency or the amplitude of the electromyographic response.

Discussion

We have shown that a rapid plantar flexion perturbation produces a decrease in the soleus EMG with an onset latency that is between the SLR and MLR responses that are induced by a rapid dorsiflexion perturbation. These data suggest that the afferent feedback contributing to the late stance phase enhancement of the extensor locomotor EMG might be different, or be processed differently, from the afferent feedback that contributes to the reflex-mediated response to an imposed dorsiflexion perturbation. Moreover, we have shown that the unload response is not contributed to by cutaneous afferents or proprioceptors in the foot or ankle. This result provides good evidence that proprioceptors contribute to the afferent-mediated enhancement of the stance phase locomotor EMG.

Assuming that the electromyographic responses to dorsiflexion and plantar flexion perturbations converge on the same motoneurons, the difference in their onset latencies can only be attributed to different mechanics that produce the cessation or increase in sensory output, different conduction velocities of the afferent fibers, or different delays within the spinal cord circuitry. At this time, it is not possible to directly measure the input–output properties of proprioceptors in response to rapid ankle perturbations while able-bodied human subjects walk on a treadmill. However, we do know from pioneering work in the cat (Matthews 1964; Matthews and Stein 1969, 1972) and human micro-
neurography studies (e.g., Burke et al. 1978) that rapid muscle shortening produces an “almost immediate” temporary cessation of the firing rate of the primary endings of the muscle spindle. In the individual data presented in Fig. 2b, the onset latency of the unload response was more than 20 ms greater than the onset latency of the short-latency stretch reflex response for half of the subjects. While we believe that it is unlikely that a delay of this magnitude can be accounted for solely by mechanical properties of the proprioceptors, the precise delay between an ankle plantar flexion and the decrease in the cessation of spindle firing remains to be determined.

There is a considerable body of evidence to support the view that the onset and initial part of the SLR response can be attributed to the group Ia afferent pathway (Matthews 1991; Taylor et al. 1985). Furthermore, we have previously elicited a SLR in the midstance phase of walking and demonstrated that this burst can be abolished with peripheral ischemia (Grey et al. 2001). We have also observed that this burst is depressed in the presence of tendon vibration at a frequency known to depress the sensitivity of primary spindle afferents (Mazzaro et al. 2004). The clear difference between the onsets of the SLR response and the unload response observed in the present study suggests that the fastest of the active group Ia afferents might not contribute to the enhancement of the locomotor extensor muscle activity. However, it is possible that slower diameter group Ia afferents make a significant contribution in this regard.

There is good evidence to suggest that the medium-latency stretch reflex response is most sensitive to the output of group II afferents originating on the secondary endings of the muscle spindle (Grey et al. 2001, 2002). In this study, the MLR burst was strongly suppressed following the ingestion of tizanidine, an α2-adrenergic receptor agonist known to selectively depress transmission in the group II afferent pathway (Bras et al. 1990; Hammar and Jankowska 2003; Jankowska et al. 2000; Skoog 1996). The observation in the
present study that the unload response occurs at a latency that is considerably shorter (by approximately 20 ms) than that of the medium-latency stretch reflex response cannot be used to rule out a contribution from the group II pathway, but it does strongly suggest that a faster pathway contributes to the response.

This leads to the possibility that the onset of the unload response could result from the cessation of Golgi tendon organ output via group Ib afferents. Both Achilles tendon force and rate of force increase during the stance phase of the step cycle; therefore, group Ib output must also increase during the latter part of the stance phase in a manner that it is well suited to provide a positive force feedback signal that would enhance plantar flexor muscle activity. Such positive force feedback through a group Ib pathway has been shown to enhance plantar flexor muscle activity in the cat (Pearson and Collins 1993), and it has been postulated by several groups that a similar mechanism might exist in humans (e.g., Dietz and Duysens 2000; Duysens et al. 2000).

The unload response is potentially a very useful tool that can be used to illuminate afferent feedback contributions to the enhancement of extensor muscle activity during the stance phase of human walking. The results of the present study do not explicitly rule out cutaneous afferents or proprioceptors in the foot as contributors to the enhancement of stance phase plantar flexor muscle activity. However, these results clearly demonstrate that plantar flexor proprioceptors provide an important source of positive feedback that enhances the locomotor muscle activity. How the sensory information is modulated throughout stance phase and how this sensory information is affected by disease remain to be determined.

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References


