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# Soleus Stretch Reflex During Cycling

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The modulation and strength of the human soleus short latency stretch reflex was investigated by mechanically perturbing the ankle during an unconstrained pedaling task. Eight subjects pedaled at 60 rpm against a preload of 10 Nm. A torque pulse was applied to the crank at various positions during the crank cycle, producing ankle dorsiflexion perturbations of similar trajectory. The stretch reflex was greatest during the power phase of the crank cycle and was decreased to the level of background EMG during recovery. Matched perturbations were induced under static conditions at the same crank angle and background soleus EMG as recorded during the power phase of active pedaling. The magnitude of the stretch reflex during the dynamic condition was not statistically different from that during the static condition throughout the power phase of the movement. The results of this study indicate that the stretch reflex is not depressed during active cycling as has been shown with the H-reflex. This lack of depression may reflect a decreased susceptibility of the stretch reflex to inhibition, possibly originating from presynaptic mechanisms.

***Key Words***: ankle, cycling, human, soleus, stretch reflex

## Introduction

Several recent studies have employed a pedaling paradigm to assess reflex modulation during cyclic movement in normal human subjects (Boorman et al., 1992; Brooke et al., 1992; Cheng et al., 1995; Collins et al., 1993; Misiaszek et al., 1995) and in pathophysiological subjects (Boorman et al., 1992). Similar to walking, pedaling requires a reasonably complex rhythmic pattern of motor control. Pedaling on an ergometer affords a good technique for the investigation of reflex modulation because a relatively constant movement pattern is enforced by the mechanical constraint of foot motion along a circular path, compared with walking where the foot is free to move in three dimensions. In addition, subjects are supported, thus decreasing the need to control balance. Jorge and Hull (1986) showed that muscle activation patterns are relatively invariant from one revolution to the next as long as ergonomic factors such as seat height and load remain invariant.

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Boorman et al. (1992) reported that the soleus Hoffman reflex (H-reflex) is modulated throughout the crank cycle in normal subjects. The H-reflex is increased during the down-stroke (power phase) and decreased during the up-stroke (recovery phase) of the cycle in parallel with soleus activation. Brooke et al. (1992) compared the modulation of the soleus H-reflexes over the crank cycle with that during static conditions with matched leg joint angles and soleus muscle activity. They reported a pattern of H-reflex modulation during active pedaling similar to that observed by Boorman et al. (1992). In addition, they showed that the H-reflex is generally depressed during movement as compared to static controls and that it is markedly depressed during the later part of the pedal cycle, following the power phase of the movement. These results are comparable to earlier reports on H-reflex modulation during walking (e.g., Capaday & Stein, 1987a; Crenna & Frigo, 1987). Boorman et al. (1992) suggested that the depressed H-reflex observed during pedaling is due to presynaptic inhibition via central and/or peripheral pathways as was previously proposed by Capaday and Stein (1986) for walking. This led to the conclusion that the stretch reflex may not play a significant role during cycling. A key difference between walking and pedaling is that the soleus muscle activation during pedaling is almost entirely concentric. In contrast, during the stance phase of walking, the soleus muscle undergoes an eccentric contraction. Therefore, one could predict that the stretch reflex during cycling would be less than that during walking.

The H-reflex is a useful tool for investigation the central gain of the reflex. However, the use of the H-reflex to infer properties of the stretch reflex during gait has been questioned by several researchers (Burke, 1985; Dietz, 1997; Sinkjaer et al., 1996). The H-reflex bypasses the fusimotor system as well as the mechanical stimulus of the muscle spindles. It produces a very synchronous afferent volley that is quite different in strength and shape from the afferent mediated response resulting from a muscle stretch induced by a mechanical perturbation. In addition, the H-reflex is strongly affected by presynaptic inhibition (Nielsen & Kagamihara 1993), compared with the less sensitive stretch reflex (Morita et al. 1998). This may account for the observation that H-reflexes are depressed during the stance phase of walking (Capaday & Stein 1986) whereas stretch reflexes are not (Sinkjaer et al. 1996).

To our knowledge, the stretch reflex has not been investigated directly during cycling. Although H-reflex studies have led to an understanding of the excitability of the central pathways of the Ia afferent mediated reflex during pedaling, the contribution of afferent feedback to the reflex has not been determined. It is unknown whether a difference between the H-reflex and stretch reflex like that reported during walking exists in other tasks involving an automatic locomotor process. The purpose of this study was to characterize the modulation and strength of the soleus stretch reflex during cycling. The stretch reflex was elicited with a mechanical perturbation of the ankle during active cycling, and the response compared to that under static conditions with matched autogenic muscle contraction and ankle angle.

## Methods

### *Subjects*

Eight healthy male subjects (age, 20–32) with no history of neuromuscular disorder were tested. The subjects had a wide range of cycling abilities: Two were elite

183

triathletes, four were regular cyclists, and two were noncyclists. All subjects gave informed consent prior to participating in the experiment.

## Apparatus and Instrumentation

Subjects were seated on a motorized bicycle ergometer for the duration of the experiment. The handlebar and seat were adjusted to positions of comfort for each subject prior to the beginning of the experiment. Standard full shank cycling shoes were worn and fixed securely to the crank arms with clipless pedals (Ritchey WCS). The crank arms were linked to a torque motor (NSK RS 1010 MegaTorque Motor) through a modified bicycle drivetrain adjusted to eliminate backlash between the crank arms and motor shaft. The ergometer was controlled with a PC computer. Crank position was measured with a resolver integrated with the motor shaft. The angular position information was digitized over 2.4° segments with a 10-bit resolver to digital converter providing a resolution of 0.00234°.

Subjects were instrumented with bipolar surface EMG electrodes (Neuroline) on their left soleus and tibialis anterior muscles. EMG signals were amplified and bandpass filtered from 20 Hz to 2 kHz. The ankle angle was measured with a custom built goniometer manufactured from a linear precision potentiometer, lexan arms, and a linear, low-noise ±5 V power supply. All data were sampled at 2.5 kHz with a 16-bit A/D data acquisition board (National Instruments AT MIO 16X). The crank position and ankle angle signals were differentiated offline to provide crank and angular velocity records.

## Protocols

The experiment was divided into two phases: dynamic (active pedaling) and static controls. For the dynamic phase, subjects pedaled at 60 rpm against a 10-Nm load. Visual and audible feedback were provided to assist the maintenance of the correct cadence. Stretch reflexes were elicited by applying small amplitude (3–5°) dorsiflexor perturbations to the ankle with torque pulses of 15–30 ms duration. The ankle angle and soleus EMG for each trial were recorded on an oscilloscope triggered by the perturbation command to the motor. Perturbations were presented on pseudo-random cycles, with an average of one perturbation in five revolutions. Subjects were instructed not to react to the perturbation and to continue cycling following the displacement. Twenty to 30 perturbations were elicited and recorded at each crank angle under both dynamic and static conditions.

Following this dynamic phase, the static protocol was performed. The motor was servo-controlled to maintain the crank in the same position as in the dynamic case. Using the stored traces on the oscilloscope as feedback, the subjects matched the ankle angle and level of soleus EMG activity that they had produced immediately prior to the perturbation while pedaling. The ankle was then perturbed with a torque pulse adjusted to produce an ankle displacement and angular velocity that matched the trajectory of the applied perturbation in the dynamic phase.

Stretch reflexes were elicited from the soleus muscle in all subjects at crank angles of 30°, 60°, 90°, 120°, and 150°, with the top dead center position of the crank arm defined as zero. In order to determine the stretch reflex modulation over the remainder of the crank cycle, perturbations were also applied at 210°, 240°, 270°, 300°, and 330° in 3 subjects. The duration of a typical session was approximately 2 hours; and it increased to 3 to 4 hours when the full cycle was collected.



Therefore, the order of crank positions was chosen randomly in order to prevent potential systematic bias due to fatigue. However, the static trials always followed the dynamic trials for a particular crank angle in order to match the pre-stretch soleus EMG and ankle angle. Data were recorded for the dynamic condition starting 2 s prior to the onset of the perturbation and ending 750 ms after the perturbation command was sent to the motor. This ensured that each record contained two unperturbed cycles followed by a perturbed cycle. For the static condition, data were recorded 500 ms before and 750 ms after the onset of the perturbation.

## Analysis

Signal conditioning and analysis were carried out offline. The EMG records were high pass filtered at 5 Hz with a fourth order Chebychev filter to remove any movement artifact. The EMG records were then full wave rectified and low pass filtered at 20 Hz with a first order filter to extract an amplitude envelope. The data records were aligned in time to a common reference point at the start of the stretch defined by the perturbation command to the motor. The background level of soleus EMG for each trial was determined by calculating a mean rectified value immediately before the stretch. A 30-ms window was chosen for the dynamic condition, and a 50-ms window for the static condition. The shorter interval used for the dynamic condition was chosen to ensure that the EMG was more representative of the activity near the desired crank position. The stretch amplitude was calculated from the difference between the peak displacement of the perturbation and the ankle angle at the onset of stretch. Stretch velocity was calculated from the slope of the ankle displacement. At a particular crank angle, the background soleus EMG, perturbation amplitude and perturbation velocity were matched between the dynamic and static conditions. Trials that deviated by more than 20% from the grouped mean in any of these criteria were rejected from further analysis.

The remaining trials were ensemble averaged to produce a single record for each subject, crank position, and condition. The peak amplitude of the soleus short latency stretch reflex was calculated from the ensemble soleus EMG records in a 30–70 ms window following the onset of the perturbation (Stein & Kearney, 1995). The onset latency of the stretch reflex was determined by visual inspection using a cursor on the display to detect the first major deflection in the EMG record within this window. The results were averaged to determine the magnitude and onset latency of the stretch reflex for the dynamic and static conditions at each of the tested crank positions.

A two-way repeated measures ANOVA (crank angle × movement condition) was used to compare the effect of the dynamic and static conditions as they changed with the crank angle. An alpha level of .05 was chosen for statistical significance.

# Results

## General Features

A typical recording for one trial is shown in Figure 1. Vertical lines have been superimposed over the second revolution to show the crank position at particular times during the cycle. After relatively little practice, all subjects were able to maintain the required cadence with the aid of a metronome. Although some subjects preferred to maintain a consistent crank velocity profile deviating no more than ±20°/s from the average 360°/s, others deviated as much as ±80°/s. However, it is notable that the variance in the stretch reflex magnitude was mostly due to inter-subject variability (80% variance accounted for in the repeated measures ANOVA). In comparison, no significant correlation was found between crank velocity and stretch reflex magnitude. In the case of the subject whose data is shown in Figure 1A, the deviation in crank velocity was approximately ±60°/s.

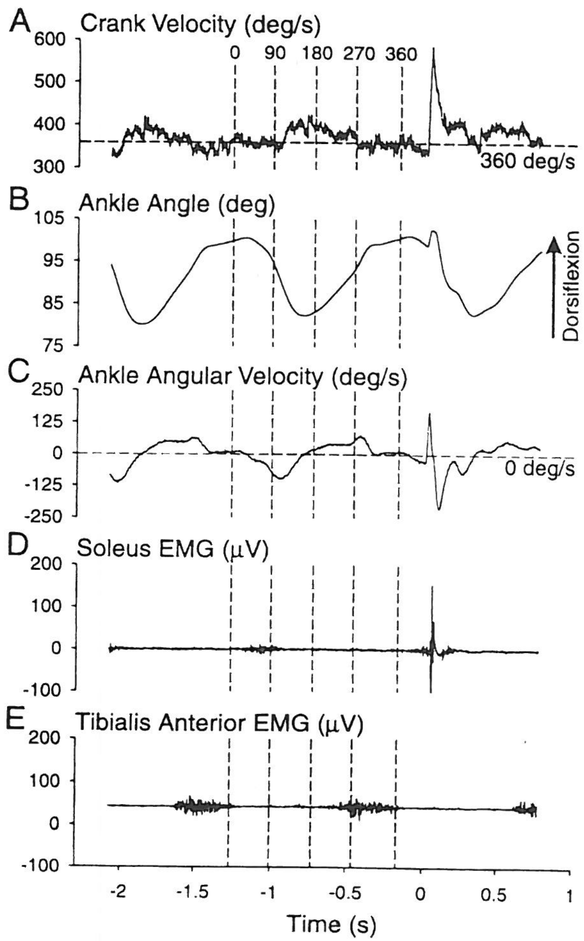


**Figure 1 — A typical data record for a single subject. Three crank cycles are shown with a perturbation at a crank angle of 60° in the last cycle. The traces are aligned at time zero, defined as the onset of the perturbation. Vertical dashed lines superimposed over the second cycle indicate the position of the crank arm, where zero is top dead center. A. Crank velocity determined from the crank position record. B. Ankle joint angle. C. Ankle joint angular velocity determined from the ankle joint ankle. D. Soleus muscle EMG. E. tibialis anterior EMG bandpass filtered from 20 Hz to 2 kHz.**



The power stroke was initiated with a burst of soleus EMG activity lasting approximately 200 ms. In all cases, this burst ended before the crank angle reached 150°. During this phase, the soleus muscle was active and the ankle was plantar flexing. For the data shown in Figure 1B, this occurs between crank angles of approximately 40° to 150°. During the power phase, the ankle velocity reached a maximum near 120° and dropped to zero near 150° (Figure 1C). The tibialis anterior muscle was silent throughout the power phase, and at no time was co-contraction of soleus and tibialis anterior muscles evident for any subject during any phase of the movement.

Following the power stroke, the ankle angle remained steady for a brief time and was followed by a short dorsiflexion movement starting just prior to the recovery phase of the stroke and continuing through to the top of the crank cycle (Figure 1B). The soleus remained silent throughout recovery, whereas the tibialis anterior activity increased toward the final part of the stroke.

## *Stretch Reflex During Cycling*

Ankle perturbations were produced by applying torque pulses with the motor. The amplitude and duration of the torque pulse required fine-tuning throughout the experiment in order to generate ankle perturbations of equal amplitude and velocity for each of the positions tested over the crank cycle. Larger torques were required at the 30° and 150° crank angles because the direction of the force applied to the foot through the pedals was more parallel to the foot at these points, compared with the intermediate crank angles, resulting in a reduced moment arm about the ankle joint.

On average, the mechanical stretch produced a reflex response with an onset latency of 43.6 ± 2.9 ms. This latency is consistent with the short latency stretch reflex response reported for the human soleus muscle (Toft et al., 1991) and reflects the monosynaptic response from group Ia afferents originating from the muscle spindles.

A typical set of perturbations over the power phase of the movement is shown for one subject in Figure 2A. These data are an ensemble average of 15 trials. In this diagram, the perturbations are shown superimposed on an unperturbed ankle angle trace. The corresponding soleus reflex responses are highlighted with the 200-ms sections presented above the perturbations. For comparison, the background EMG throughout the crank cycle is shown in Figure 2B. The reflex responses increased during the middle of the power stroke and decreased at crank angles of 120° and 150°. In this case, the reflex response had dropped to zero by 150°.

In 3 subjects, stretch reflexes were recorded at regular intervals from 30° to 330°. Stretches could not be elicited at the top or bottom of the crank cycle (zero or 180°) because the direction of force at these points was almost parallel to the pedal, resulting in a small moment arm about the ankle. Ensemble averaged results from one subject are presented in Figure 3. The stretch reflex modulation throughout the crank cycle is shown in Figure 3A. Figures 3B, 3C, and 3D show the unperturbed background EMG, ankle angle, and ankle velocity modulation, respectively. The stretch amplitude and velocity is superimposed on the position and velocity traces in Figures 3C and 3D, respectively. The average perturbation amplitude and perturbation velocity is shown by dashed lines in Figures 3C and 3D. In this case, the reflex was greatest during the power stroke between the crank angles 60° and

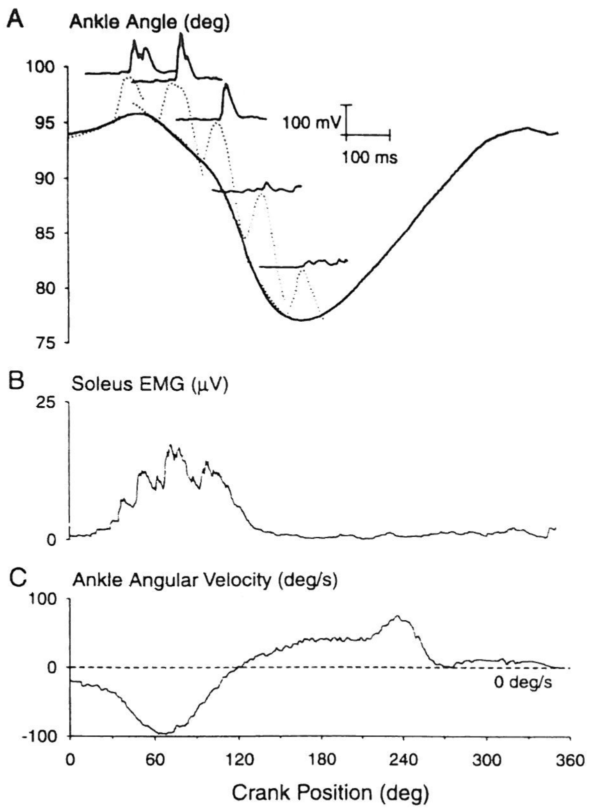


**Figure 2 — A. Ankle perturbations (dotted lines) are shown superimposed over an unperturbed ankle trajectory (solid line). The perturbation amplitude is approximately 4°. Soleus reflex responses to the perturbations are shown above the corresponding ankle trace. Unperturbed traces for rectified and filtered soleus EMG (B) and ankle velocity (C) are shown corresponding to the unperturbed ankle position in panel A. Traces are shown with respect to the crank angle.**

120°. The stretch reflex magnitude was greatest just prior to maximum plantar flexion velocity and decreased to zero during the recovery phase of the cycle, corresponding with the silent period of the soleus muscle. A similar modulation throughout the cycle was observed in both of the other subjects.

For most subjects, the time required to collect a full set of data far exceeded a point were fatigue effects could be ruled out. For this reason, measurement at positions over the entire crank cycle was limited to 3 subjects. For all other subjects, measurements were restricted to the power phase of the movement because it was here that the soleus muscle was most active and where the afferent mediated feedback could be expected to add most importantly to the EMG. In addition, the stretch reflexes recorded over the recovery phase were small and not modulated to the same extent as in the power phase.



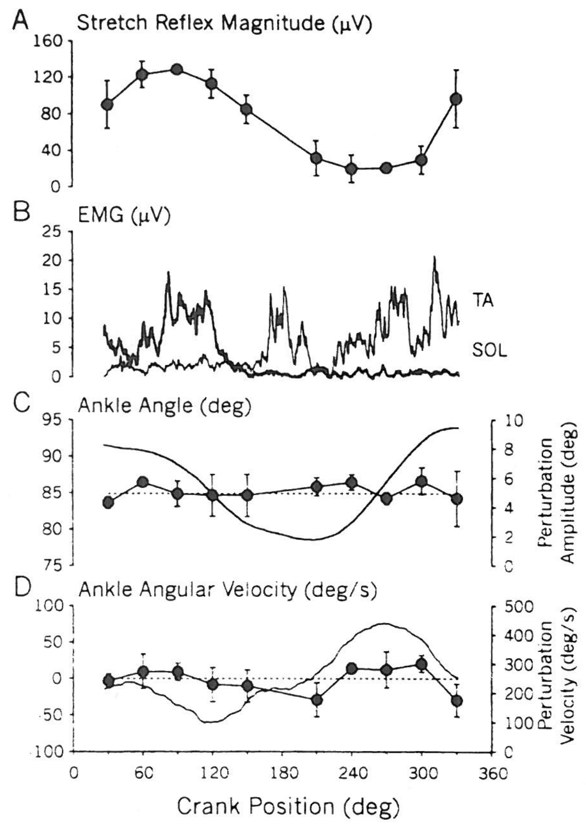


**Figure 3 — A. Stretch reflex modulation over the pedal cycle for one subject. B. Rectified and filtered soleus EMG (bold line) and tibialis anterior EMG (light line). C. Ankle angle and perturbation amplitude plotted on the left and right axes, respectively. The dashed line at 4.9° represents the mean perturbation amplitude. D. Ankle velocity and perturbation velocity plotted on the left and right axes, respectively. The mean perturbation velocity at 245°/s is denoted by the dashed line. Error bars represent standard deviations.**

## *Dynamic and Static Stretch Reflex Responses*

For comparison of the stretch reflex responses in the dynamic and static cases, the pre-stretch ankle angle, background soleus EMG level, perturbation amplitude, and perturbation velocity were matched between the two experimental conditions. The effect of the dynamic and static conditions on the stretch reflex for 1 subject is compared in Figure 4A together with matched background EMG (Figure 4B), ankle perturbation amplitude (Figure 4C), and ankle perturbation velocity (Figure 4D). Two-way repeated measures ANOVA tests were conducted for each of background EMG, perturbation amplitude, and perturbation velocity to confirm that there was no difference in these variables between the two movement conditions. A statistically significant difference between the dynamic and static conditions was not found for any of these variables.

It was not possible to maintain the same perturbation amplitude and velocity across every crank position for all subjects. Although infrequent, when this did occur it was always at crank angles of 30° or 150°, where the perturbation was the most difficult to generate. In all cases, care was taken to ensure the perturbation characteristics were as close as possible to the perturbations generated at each of

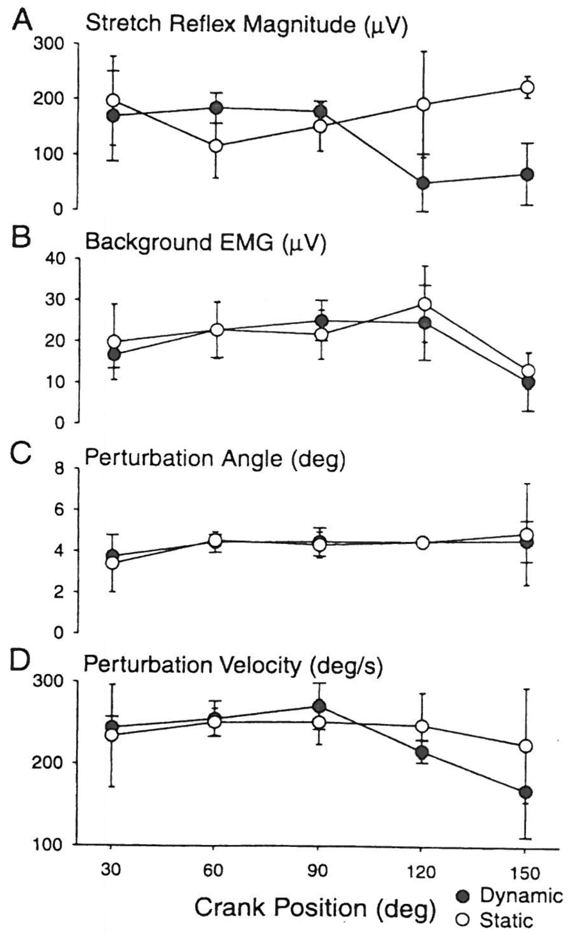


**Figure 4 — Stretch reflex magnitude during dynamic (closed circles) and static (open circles) conditions for 1 subject at each crank position tested in the power phase. A. Stretch reflex magnitude determined from the rectified and filtered soleus EMG. B. Background EMG activity determined from soleus muscle immediately prior to the perturbation. C. Perturbation amplitude. D. Perturbation velocity of the ankle in response to a torque pulse. Background EMG, perturbation amplitude, and perturbation velocity are matched at each crank position. Error bars indicate standard deviation.**



the other crank positions. At most, the perturbation amplitudes differed by 1.5°. The perturbation velocity and background EMG were more variable than the perturbation amplitude. This was due primarily to the low resolution afforded by the oscilloscope screen. Traces that looked quite similar on the oscilloscope as the data was being acquired were sometimes found to be different when displayed at higher resolution during the offline analysis. In addition, some subjects experienced difficulty in maintaining the same level of background EMG during the static condition as during the dynamic phase. In general, they perceived that more effort was required to maintain the static contraction and the ankle angle for the crank positions where the soleus was most active during pedaling (60° and 90°). There was comparatively little variation in the perturbation amplitude between the static and dynamic conditions and between the crank positions.

The stretch reflex magnitudes for the dynamic and static conditions, pooled across all subjects, are compared in Figure 5. There was a trend for the stretch reflex to be greater for the dynamic condition than for the static condition from 30° to 90°. It was then depressed with respect to the static condition at 120° and 150°. Despite this trend, the repeated measures ANOVA results indicate that the stretch reflex in the dynamic and static conditions was not statistically different across all crank angles ($p = .85$). There was, however, a strong interaction effect between the crank angle and movement condition ($p < .001$), supporting the observation that the two movement conditions differ among the various crank angles. Given this strong interaction effect, the dynamic and static conditions were compared at 60°, 90°, and 150° with independent students' $t$ tests. In all three cases, a significant

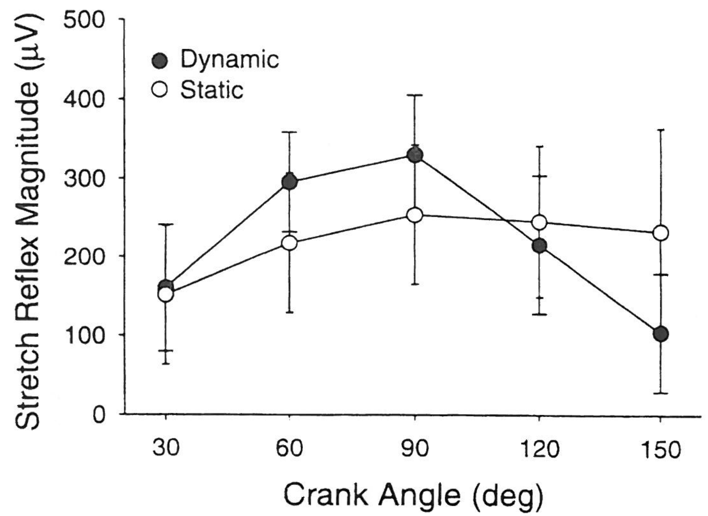


**Figure 5 — Stretch reflex magnitude averaged across all subjects comparing the dynamic (filled circles) and static (open circles) conditions for each crank position tested in the power phase of the crank cycle. Background soleus EMG, perturbation amplitude, and perturbation velocity were matched in all experiments. Error bars indicate standard deviation.**

difference was found between the two movement conditions ($p < .001$ for all three comparisons).

## Discussion

We have shown that the magnitude of the soleus stretch reflex is modulated in a cyclic manner over the crank cycle. The reflex is greatest during the power phase of the movement and decreases to near zero during the recovery phase. The pattern of stretch reflex modulation observed in this study is comparable to that reported during walking (Sinkjaer et al., 1996). In both cases, the stretch reflex rises and falls in a manner similar to the background EMG, although it does not strictly follow the background EMG. In both Figures 3A and 4A, the stretch reflex in the dynamic condition slightly precedes the background activity.

### *Methodological Considerations*

Although the length and background activity of the soleus muscle was the same in both the static and dynamic cases, the external constraints for the two conditions are not alike. Part of the EMG activity seen during the dynamic condition may be related to maintaining a given ankle angle in the face of perturbing forces due to reaction, centripetal, and Coriolis forces from hip and knee motion. Under the static condition, the soleus EMG is used only to counteract gravity and to exert force on the pedal. Therefore, it is possible that the stiffness of the soleus muscle is different in the two conditions. In the dynamic case, the soleus may be more or less stiff than in the static condition, depending on whether or not the muscle is being lengthened or shortened. This could mean that the muscle fibers do not receive the same stretch during the two conditions despite the fact that the overall muscle length was matched.

It could also be argued that homonymous connections onto the soleus by the medial and/or lateral heads of the gastrocnemius (Brooke & McIlroy, 1985) may account for the differences observed between the static and dynamic conditions. Because we did not record the activity from either of these muscles, we cannot be certain that this trend is not accounted for by changes in gastrocnemius activity between the two tasks.

Another factor that may potentially influence these results is the use of a torque pulse to generate the perturbation. It could be argued that the torque pulse would perturb the knee and hip, resulting in a stretch of other muscles that could have inhibitory or excitatory effects on the soleus stretch reflex through presynaptic mechanisms. During the pilot experiments for this study, a second goniometer was fixed to the knee of the subjects. Analysis of these data revealed that the stretch was almost entirely taken up by the ankle, while the knee moved 2° at most. Although we cannot rule out the possibility of a stretch of the quadriceps muscles influencing the soleus stretch reflex, it could be argued that the effect of such a small motion at the knee would be minimal. This experiment has been repeated with a portable stretcher device capable of applying a perturbation directly to the ankle (for device details, see Andersen & Sinkjaer, 1995). Using this device, we have seen the same pattern of stretch reflex modulation in the dynamic and static movement conditions as reported in the present study (unpublished results: Larsen, B., Grey, M.J., Voigt, M., & Sinkjaer, T., 2000).



### *Comparison of Soleus H-reflex and Stretch Reflex*

Although the methodological considerations indicate that caution must be used in the interpretation of these results, they are still comparable to the H-reflex study of Brooke et al. (1992) because the two studies use the same paradigm.

The depression of the H-reflex during active pedaling has been explained by centrally and/or peripherally mediated presynaptic inhibition of the Ia afferents that elicit the reflex during a cyclic movement (e.g., Brooke et al., 1992; Capaday & Stein, 1986). Capaday and Stein (1986) reported that the H-reflex during standing was up to 3.5 times as large as that during walking. This is a much greater difference between active movement and a static control than has been observed during cycling (Brooke et al., 1992).

In contrast to the study reported by Brooke et al. (1992), the results of the present study suggest that during cycling the stretch reflex is not different from static controls and may even be facilitated during the power phase. One explanation for this result is that the dynamic increase in activity during the EMG burst, where new motoneurons are being recruited, causes the motoneuron pool to be more excitable to stretch induced afferent input than in the static situation, where a fixed number of motoneurons are active (Capaday & Stein, 1987b). The same argument applies to the later part of the power stroke, where both the background soleus EMG and the stretch reflex decline. Here the stretch reflex is reduced in amplitude to a level below that observed during the static condition. As the motor units are de-recruited during this part of the cycle, their excitability is decreased. This will be expressed as a decreased stretch reflex despite the fact that the background EMG is matched to the static condition (Romanò & Schieppati, 1987).

The observation that the stretch reflex may be facilitated in part of the active power phase is in contrast to the H-reflex observations described above for cycling (Boorman et al., 1992; Brooke et al., 1992; Cheng et al., 1995; Collins et al., 1993; Misiaszek et al., 1995) and walking (Capaday & Stein, 1986, 1987a; Morin et al., 1982; Stein & Capaday, 1988). This lack of depression during active movement may be explained by the results of Morita et al. (1998), who showed that the stretch reflex is less sensitive to presynaptic inhibition than is the H-reflex. They suggested that a sudden perturbation of the limb would lead to a high discharge rate in the Ia afferents, which would overcome the effects of presynaptic inhibition. Presynaptic inhibition would, therefore, modulate the central effect of the normally low rate of Ia afferent discharge and allow compensatory reflexes to exert their full action. Another possible explanation for the overall lack of inhibition in the stretch reflex during cycling may be that changes in gamma motor activity during dynamic movements (Prochazka et al., 1985) may counteract the increase in presynaptic inhibition that is inferred by H-reflex studies.

Brooke et al. (1992) concluded that the H-reflex was depressed during active cycling based on an ANOVA comparing the movement condition and crank position. However, they made this comparison over the whole crank cycle. A close look at the H-reflex modulation, comparing the two movement conditions in their study (Figure 2; Brooke et al., 1992) shows that during the power phase of the movement, the two conditions are not very different and that the most striking difference occurs only during the recovery phase. Notwithstanding this observation, their data still show a trend towards depression of the H-reflex during the dynamic condition.

Both walking and cycling are automatic cyclic processes. However, the mechanics of the two movements are quite different. During walking the soleus muscle undergoes a distinct phase of eccentric contraction during early to mid stance, and it has recently been shown that up to 50% of the background soleus EMG is caused by afferent feedback from the stretched ankle extensor ( Sinkjaer et al., 1996; Yang et al., 1991). Although there is a very brief period of eccentric soleus activity during cycling, the magnitude and duration of this phase is a great deal smaller than that during walking. Therefore, the amount of afferent feedback from the soleus muscle that contributes to the background EMG is very likely lower during the power phase of cycling compared with the stance phase of walking. This might explain why the phase advance of the soleus stretch reflex with respect to the background EMG is somewhat less during cycling than has been reported during walking (Sinkjaer et al., 1996).

The increased stretch reflex observed during the power phase of the movement might have functional significance by enhancing the stiffness of the ankle. An increased joint stiffness would also increase the power transmission efficiency at the ankle by reducing the energy dissipated in damping from viscous elements.

## References


Andersen, J.B., & Sinkjaer, T. (1995). An actuator system for investigating electrophysiological and biomechanical features around the human ankle joint during gait. *IEEE Transactions on Rehabilitative Engineering*, **3**, 299-306.

Boorman, G., Becker, W.J., Morrice, B.L., & Lee, R.G. (1992). Modulation of the soleus H-reflex during pedalling in normal humans and in patients with spinal spasticity. *Journal of Neurology, Neurosurgery & Psychiatry*, **55**, 1150-1156.

Brooke, J.D., & McIlroy, W.E. (1985). Locomotor limb synergism through short latency afferent links. *Electroencephalography and Clinical Neurophysiology*, **60**, 39-45.

Brooke, J.D., McIlroy, W.E., & Collins, D.F. (1992). Movement features and H-reflex modulation. I. Pedalling versus matched controls. *Brain Research*, **582**, 78-84.

Burke D. (1985). Mechanisms underlying the tendon jerk and H-reflex. In P.J. Delwaide & R.R. Young (Eds.), *Clinical neurophysiology in spasticity* (pp. 55-62). Amsterdam: Elsevier.

Capaday, C., & Stein, R.B. (1986). Amplitude modulation of the soleus H-reflex in the human during walking and standing. *Journal of Neuroscience*, **6**, 1308-1313.

Capaday, C., & Stein, R.B. (1987a). Difference in the amplitude of the human soleus H reflex during walking and running. *Journal of Physiology*, **392**, 513-522.

Capaday, C., & Stein, R.B. (1987b). A method for simulating the reflex output of a motoneuron pool. *Journal of Neuroscience Methods*, **21**, 91-104.

Cheng, J., Brooke, J.D., Staines, W.R., Misiaszek, J.E., & Hoare, J. (1995). Long-lasting conditioning of the human soleus H reflex following quadriceps tendon tap. *Brain Research*, **681**, 197-200.

Collins, D.F., McIlroy, W.E., & Brooke, J.D. (1993). Contralateral inhibition of soleus H reflexes with different velocities of passive movement of the opposite leg. *Brain Research*, **603**, 96-101.

Crenna, P., & Frigo, C. (1987). Excitability of the soleus H-reflex arc during walking and stepping in man. *Experimental Brain Research*, **66**, 49-60.

Dietz, V. (1997). Neurophysiology of gait disorders: present and future applications. *Electroencephalography and Clinical Neurophysiology*, **103**, 333-355.





Jorge, M., & Hull, M.L. (1986). Analysis of EMG measurements during bicycle pedalling. *Journal of Biomechanics*, **19**, 683-694.

Misiaszek, J.E., Brooke, J.D., Lafferty, K.B., Cheng, J., & Staines, W.R. (1995). Long-lasting inhibition of the human soleus H reflex pathway after passive movement. *Brain Research*, **677**, 69-81.

Morin, C., Katz, R., Mazieres, L., & Pierrot-Deseilligny, E. (1982). Comparison of soleus H reflex facilitation at the onset of soleus contractions produced voluntarily and during the stance phase of human gait. *Neuroscience Letters*, **33**, 47-53.

Morita, H., Petersen, N., Christensen, L.O., Sinkjaer, T., & Nielsen, J. (1998). Sensitivity of H-reflexes and stretch reflexes to presynaptic inhibition in humans. *Journal of Neurophysiology*, **80**, 610-620.

Nielsen, J., & Kagamihara, Y. (1993). The regulation of presynaptic inhibition during co-contraction of antagonistic muscles in man. *Journal of Physiology*, **464**, 575-593.

Prochazka A., Hulliger, M., Zangger, P., & Appenteng, K. (1985). "Fusimotor set": new evidence for alpha-independent control of gamma-motoneurones during movement in the awake cat. *Brain Research*, **339**, 136-140.

Romanò, C., & Schieppati, M. (1987). Reflex excitability of human soleus motoneurones during voluntary shortening or lengthening contractions. *Journal of Physiology*, **390**, 271-284.

Sinkjaer, T., Andersen, J.B., & Larsen, B. (1996). Soleus stretch reflex modulation during gait in humans. *Journal of Neurophysiology*, **76**, 1112-1120.

Stein, R.B., & Capaday, C. (1988). The modulation of human reflexes during functional motor tasks. *Trends in Neurosciences*, **11**, 328-332.

Stein, R.B., & Kearney, R.E. (1995). Nonlinear behavior of muscle reflexes at the human ankle joint. *Journal of Neurophysiology*, **73**, 65-72.

Toft, E., Sinkjaer, T., Andreassen, S., & Larsen, K. (1991). Mechanical and electromyographic responses to stretch of the human ankle extensors. *Journal of Neurophysiology*, **65**, 1402-1410.

Yang, J.F., Stein, R.B., & James K.B. (1991). Contribution of peripheral afferents to the activation of the soleus muscle during walking in humans. *Experimental Brain Research*, **87**, 679-687.


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