Exp Brain Res (1991) 87:679–687

Experimental Brain Research

Contribution of peripheral afferents to the activation of the soleus muscle during walking in humans

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Received December 24, 1990 / Accepted July 5, 1991

Summary. Small, rapid stretches were applied to the soleus muscle during the stance phase of walking by lifting the forefoot with a pneumatic device. Stretch responses were induced in the soleus muscle by the disturbance. The amplitude and time course of the responses from the soleus muscle were a function of both the kinematics of the disturbance and the time in the step cycle when the disturbance was applied. The step cycle was divided into 16 equal time parts, and data obtained within each of these parts were averaged together. The electromyographic (EMG) response of the soleus muscle showed a time course that was similar to the time course of the angular velocity induced by the disturbance at the ankle. Three linear equations were used to predict the EMG response from the soleus muscle as a function of the angular kinematics of the disturbance: 1) velocity, 2) velocity and displacement, 3) velocity, displacement and acceleration. Introduction of a pure delay between the EMG and the kinematics substantially improved the predictions. Most of the variance (70%) in the EMG response could be accounted for by the velocity of the disturbance alone with an optimal delay (average 38 ms). Inclusion of a displacement term significantly increased the variance accounted for (85%), but further addition of an acceleration term did not. Since the velocity of the disturbance accounted for most of the variance, the reflex gain was estimated from the velocity coefficient. This coefficient increased in a ramp-like fashion through the early part of the stance phase, qualitatively similar to the increase in the H-reflex. Based on these identified gains, this reflex pathway was estimated to contribute substantially (30% to 60%) to the activation of the soleus muscle particularly during the early part of the stance phase.

Key words: Stretch reflex – Locomotion – Human

Introduction

Two extreme views of how the nervous system controls walking, either exclusively by central circuitry or by reflexes alone, have given way to an intermediate position (reviewed in Delcomyn 1980; Grillner 1981). The wealth of evidence now suggests that both mechanisms contribute to the control of locomotion (Grillner, 1981; Rossignol et al. 1988). Information from the periphery can change the central rhythm (reviewed in Rossignol et al. 1988) as well as contribute to the activation of muscles in locomotion (Pearson and Duysens 1976; Wolf and Pearson 1988). Severin (1970) reported that in the early stages of a peripheral nerve block, one can selectively block the fusimotor axons. During this time period, the electromyogram (EMG) of the triceps surae group dropped by 50%, implying that muscle spindle activity contributed substantially to the activation of this muscle group. This is perhaps the only quantitative estimate available for the relative contribution of stretch reflexes, although some blockage of α-motoneurons can not be excluded.

The soleus (SOL) muscle appeared to us to be a good candidate with which to address this question of the relative role of central versus peripheral mechanisms in locomotion. The SOL undergoes an active lengthening contraction through most of the stance phase in both the cat and human. Since muscle spindle afferents fire during this lengthening contraction (Prochazka et al. 1976) and since the reflex pathway is open at this time (Akazawa et al. 1982; Capaday and Stein 1986; Crenna and Frigo 1987), muscle spindles could contribute to the activation of the SOL.

Two questions are addressed in this paper. First, are the results from H-reflex studies (Capaday and Stein 1986; Crenna and Frigo 1987) representative of the response to more realistic disturbances? Only electrical stimuli (e.g., the H-reflex) and very brief mechanical stimuli (e.g. tendon taps, see Llewellyn et al. 1986) have been used to test the myotatic reflex arc in human walking. Such stimuli elicit brief, synchronous afferent volleys, and therefore tend to be unphysiological. The afferents primarily responsible for the H-reflex are thought to be group Ia fibers from muscle spindles (Magladery et al. 1951). Since Ia afferents are velocity sensitive, a mechanical disturbance which stretches the muscle unexpectedly should activate these afferents, as might occur naturally when walking on uneven surfaces. Responses to mechanical stretches could then be compared with those to electrical stimulation.

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Second, if the reflex pathway is indeed open during the stance phase, how much does it contribute to the activation of the SOL muscle? This question might be addressed by using methods of system identification. The relationship between the input (muscle stretch) and output (EMG response) to a mechanical disturbance could be determined for the SOL muscle about an operating point, for example, a specific time point in the walking cycle. Repeating this procedure for different times in the walking cycle could then provide the time course of this relationship, a measure of reflex gain. This method is valid, so long as the system can be considered to behave linearly. The assumption that the system is linear is reasonable for: 1) a restricted operating point (i.e., a limited range of ankle joint position and bias torque, see Gottlieb and Agarwal 1978; Weiss et al. 1986), 2) a limited range of disturbance amplitudes (Kearney and Hunter 1983) and 3) disturbances spaced adequately in time (Aldridge and Stein 1982).

Reflex gains identified in this way should be representative of those in normal walking, so long as the velocity of the mechanical stretch is close to the rate of stretch normally experienced in walking. The proportion of SOL EMG in walking that results from muscle stretch can be obtained by multiplying the reflex gain and the velocity of stretch of the SOL muscle at a particular time in a walking cycle. A quantitative value can then be estimated for the relative contribution of peripheral versus central mechanisms in walking, at least for the activation of the SOL muscle.

Methods

Device for applying muscle stretch

It is technically difficult to apply mechanical disturbances to the triceps surae group during walking because of the extremely high torques this muscle group generates (Winter 1987). A pneumatic device was designed to apply a stretch to the SOL muscle by rapidly lifting the forefoot off the ground during the stance phase of walking. Two double-acting air cylinders (Clippard Instruments Laboratory Ltd., Cincinnati, Ohio) were mounted on a U-shaped aluminum plate (11.5 cm × 7 cm × 5 cm), which could be attached to the subject's shoe. The cylinders contain 2 air chambers that are separated by a piston. The position of the piston and its attached pushrod was determined by the difference in air pressure between the two chambers. Each chamber could be connected to either compressed air or vented to atmosphere. Compressed dry air was regulated from 20 MPa to 1700 kPa (the tolerance of the air cylinders), using an L-TEC variable regulator. The pathway of airflow was controlled by a 4-way solenoid air valve (Ascolectric Ltd., Brantford, Ontario), as shown in Figure 1. The air valve controlled which chamber the compressed air was directed to and which chamber was vented to atmosphere. When the compressed air was directed to the bottom chamber, the pushrod and piston would assume a high position (Fig. 1A), and the parallelograms would not disturb walking. When the compressed air was directed to the top chamber, the pushrod would assume a low position (Fig. 1B), and the parallelogram would lift the forefoot off the ground. This in turn dorsiflexes the ankle and stretches the SOL muscle. Note that parallelogram structures, one on each cylinder, were driven by the pistons and pushrods of the cylinders to distribute the forces evenly as the lifting occurred. The total apparatus added approximately 1.5 kg of mass to the subject's shoe.

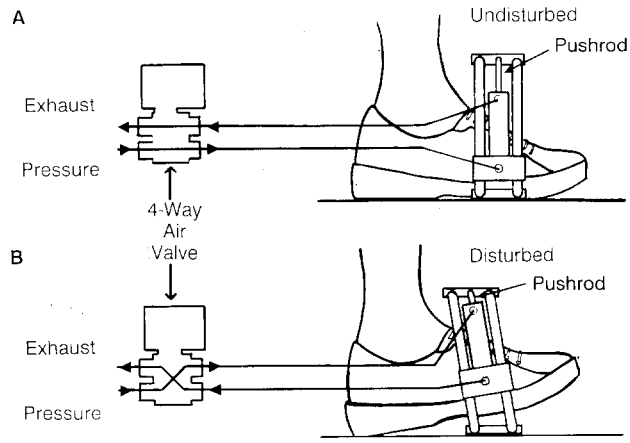

Fig. 1A, B. This schematic diagram illustrates how the airflow controlled the position of the cylinders. The cylinders contained 2 air chambers that were separated by a piston. Each chamber could be connected to the compressed air (pressure) or vented to atmosphere (exhaust). A 4-way solenoid air valve controlled this connection. When the compressed air was directed to the bottom chamber while the top chamber was vented to atmosphere (as shown in **A**), the piston and its attached pushrod assumed a high position, which did not disturb walking. Note the lengh of the pushrod (arrow) exposed above the cylinder. To apply a disturbance, the pathway of airflow was switched (by the electrically operated air valve) so that the compressed air was now directed to the top chamber and the bottom chamber was vented to atmosphere (**B**). This causes the piston and pushrod to assume a low position, as seen by the reduced length of the pushrod exposed above the cylinder. The forces are transmitted via a parallelogram structure that lifts the forefoot, resulting in a sudden dorsiflexion of the ankle

Experimental procedures

Surface EMGs were recorded from the SOL and tibialis anterior (TA) muscles with Beckman type disk electrodes (8 mm in diameter). The raw signal was bandpass filtered (10 Hz to 10 kHZ), full-wave rectified and low-pass filtered at 30 Hz. The ankle angle was monitored with a goniometer. The goniometer was attached to the leg with elasticized velcro straps to prevent slippage. A footswitch attached to the heel of the shoe indicated the time of heel-contact.

Subjects walked on the treadmill at 4 km/h, first without the device to determine their EMG and ankle angle excursion during normal walking, and then with the device on but with no application of disturbances, to determine whether wearing the device altered their walking. The ankle angle and EMG associated with walking were averaged on-line from the time of heel-contact (approximately 30 strides were averaged for each condition).

Disturbances were then applied during the stance phase, about one every one to four cycles, in a random fashion so that the subjects could not anticipate which steps and what part of the stance phase would be disturbed. A total of approximately 120 disturbances were applied. The raw EMG, ankle angle, footswitch and stimulus trigger were recorded on magnetic tape for off-line analysis.

Data analysis

Data were averaged as a function of the time the disturbance occurred in the step cycle. The step cycle was divided into 16 equal segments in time, starting from the time of heel-contact (each segment was approximately 60–70 ms duration). Ankle angle and EMG data associated with stimuli which occurred within one of the segments were averaged together (approximately 20 disturbances per

segment). Averaging was initiated by the electrical pulse that triggered the solenoid valve to switch states. These averages represent the time course of responses to the disturbances superimposed on the profile associated with undisturbed walking (see for example, Fig. 3). The appropriate profile of undisturbed walking was subtracted from the total average to obtain the portion of the average associated with the response to the disturbance alone. The profiles for undisturbed walking were obtained from steps during the experimental trials in which no perturbations were applied. Care was taken to exclude steps that followed immediately after a perturbed step in case the subjects were still making some corrections in their walking. These subtracted responses from the first six segments of the walking cycle, which corresponded to the first 50% to 60% of the stance phase, were used in the subsequent analyses.

The time course of ankle displacement generated by the disturbance, obtained from each of the first six parts of the stance phase, was differentiated after digital smoothing to obtain the velocity and acceleration profiles. The corresponding EMG responses were digitally filtered in exactly the same way to preserve the timing relationship between the two signals. Linear models of the form:

$$EMG(t) = a_0 + a_2\dot{x}(t) \quad (1)$$

$$EMG(t) = a_0 + a_1 x(t) + a_2\dot{x}(t) \quad (2)$$

$$EMG(t) = a_0 + a_1 x(t) + a_2\dot{x}(t) - a_3\ddot{x}(t) \quad (3)$$

were used to fit the data from each of the six segments of the stance phase, separately. EMG(t) is the average EMG response to the disturbance at time t, x(t), $\dot{x}(t)$, and $\ddot{x}(t)$ are the average displacement, velocity and acceleration of the ankle in response to the disturbance at time t, respectively. a_0, a_1, a_2 and a_3 are constants estimated by an algorithm which minimized the sum of squared differences between the model and the data, using a gradient search method that selects the slope of steepest descent (Stein et al. 1988). The coefficients a_1, a_2, and a_3 were constrained to be positive, whereas a_0 could assume any value. The goodness of fit was quantified by the proportion of variance accounted for (VAF) by the fitted curve:

$$VAF = \left(1 - \frac{\sum_{j=1}^{n} (P_j - M_j)^2}{\sum_{j=1}^{n} (M_j - \bar{M})^2}\right) \times 100 \quad (4)$$

where P_j is the predicted EMG response for time j, M_j is the observed EMG response at time j, and $\bar{M}$ is the overall average of the observed EMG over the 200 ms window:

$$\bar{M} = \frac{1}{n} \sum_{j=1}^{n} M_j$$

Better fits were obtained if a pure delay was introduced to account for the transmission time required between the disturbance and the response. The delay time which resulted in the maximum variance accounted for was defined as the optimal delay, and was determined iteratively for each of the models.

Since good fits were generally obtained by using model 1 (i.e. the velocity-sensitive model), the coefficient associated with the velocity term, a_2, was used to estimate the contribution of a velocity sensitive element to the activation of the SOL during early stance. The velocity of dorsiflexion during undisturbed walking was multiplied by this coefficient a_2, identified at the corresponding time in the stance phase, to estimate this contribution.

Results

Effect of wearing the device

Generally, wearing the device did not alter the walking pattern substantially. The duration of the step cycle was shortened slightly in some subjects, and the amplitude of the TA activity was slightly increased to control the additional weight (Fig. 2). In some subjects, the ankle was slightly (approximately 3°) more plantarflexed over the step cycle when wearing the device; no other changes were observed in the ankle trajectory.

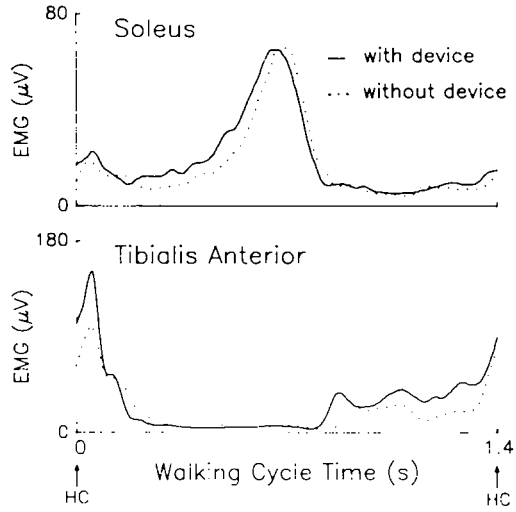

Fig. 2. The soleus and tibialis anterior EMG associated with treadmill walking in one subject, with normal footwear (dotted lines) and with the pneumatic device on but with no disturbances applied (solid lines). Each condition represents an average of 50 strides. The horizontal axis represents time in the walking cycle starting from heel-contact (HC) and ending with heel-contact. Wearing the pneumatic device caused a slight shortening of the stance duration (note the slight phase shift in the EMG's) and required slightly more tibialis anterior activity to control the added weight

Effect of the disturbance

The time course of ankle dorsiflexion is shown in Fig. 3 (top traces) for the first segment of the stance phase in one subject. The average trajectory of the ankle angle is shown for disturbed and undisturbed trials superimposed. The difference between the two trajectories representing the effect of the disturbance is shown on the right. Note that the electrical event that triggered the averaging (i.e., the beginning of the trace) occurred slightly before heel-contact. The mechanical disturbance was initiated approximately 130 ms later. This time delay between the electrical event and the mechanical event was the time required for the compressed air to travel from the air tank to the cylinders. Generally, the disturbance was ramp-shaped with a peak amplitude of between 3 to 9 degrees depending on the subject, and a time to peak of approximately 100 ms. The peak amplitude of the disturbance varied between subjects because the device generated a fixed vertical displacement of the forefoot. The angular displacement at the ankle was a function of the distance from the pneumatic device to the axis of the ankle joint, a distance that varied with the size of the subject's foot. The peak velocity of stretch was on average 40 to 100 deg/s, similar to the peak velocity of ankle dorsiflexion observed during

the stance phase of walking in these same subjects. The corresponding EMG averages are shown in the middle and bottom traces of Figure 3. The subtracted responses showed an early excitation in the SOL which was followed by a response in the TA and a second excitatory response in the SOL. The first excitation observed in the SOL will be referred to as the early response. The exact latencies of these responses are difficult to determine from data such as shown in Fig 3, because the time course of the change in ankle angle is not abrupt. The latencies are better exposed by fitting the whole time course of the EMG response to the angular velocity (described later).

All subjects showed an early excitatory response from the SOL muscle and a later excitatory response from the TA. Some differences were noted between subjects in the time course of the SOL response. Two extreme examples are shown in Fig. 4. The subject in A showed a single burst of SOL EMG, while the subject in B showed a double burst. The SOL response in the first subject clearly resembled the velocity profile of the disturbance. The double burst of the SOL EMG shown in Fig. 4B obviously deviates from the single peak observed in the angular velocity. Nevertheless, the overall duration of the excitatory response, the rate of rise and fall of the EMG, the

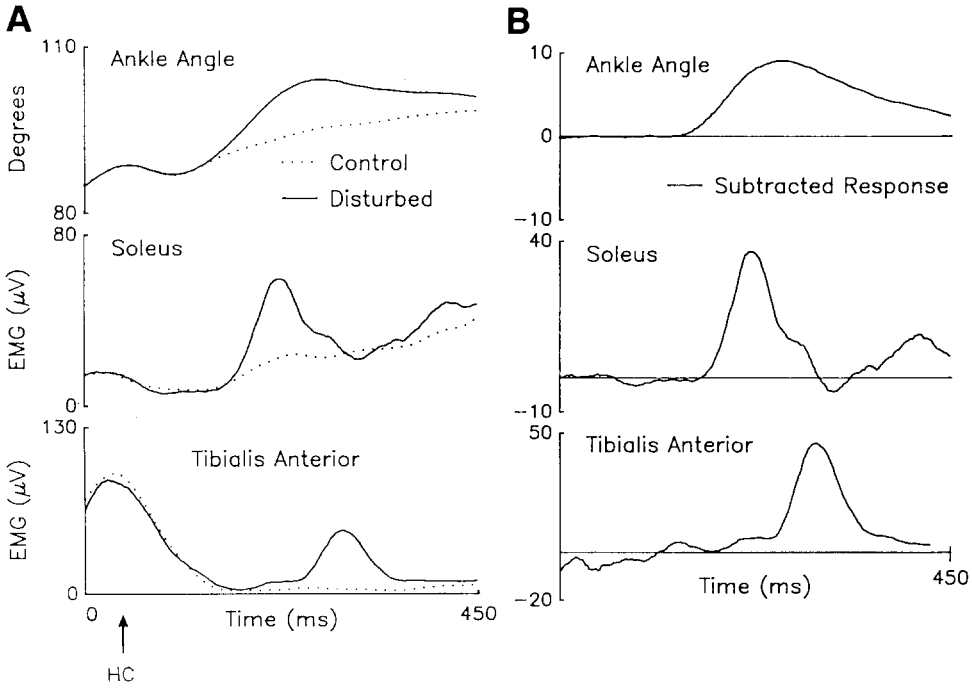

Fig. 3. A Responses to the disturbance (solid lines) are superimposed on the profiles associated with undisturbed walking (dotted lines) for one subject. These represent an average of 20 trials which were elicited during the early part of the stance phase. Note that the electrical event that triggered the averaging occurred slightly before heel-contact (HC). **B** Responses to the disturbance after subtraction of the profile associated with undisturbed walking (i.e., subtracting the dotted line from the solid line in **A**). The disturbance produced a rapid ramp-like stretch of the ankle, with a peak displacement of 9 degrees in this case, and a time-to-peak of approximately 100 ms. The soleus muscle was activated at a short latency and the tibialis anterior at a longer latency

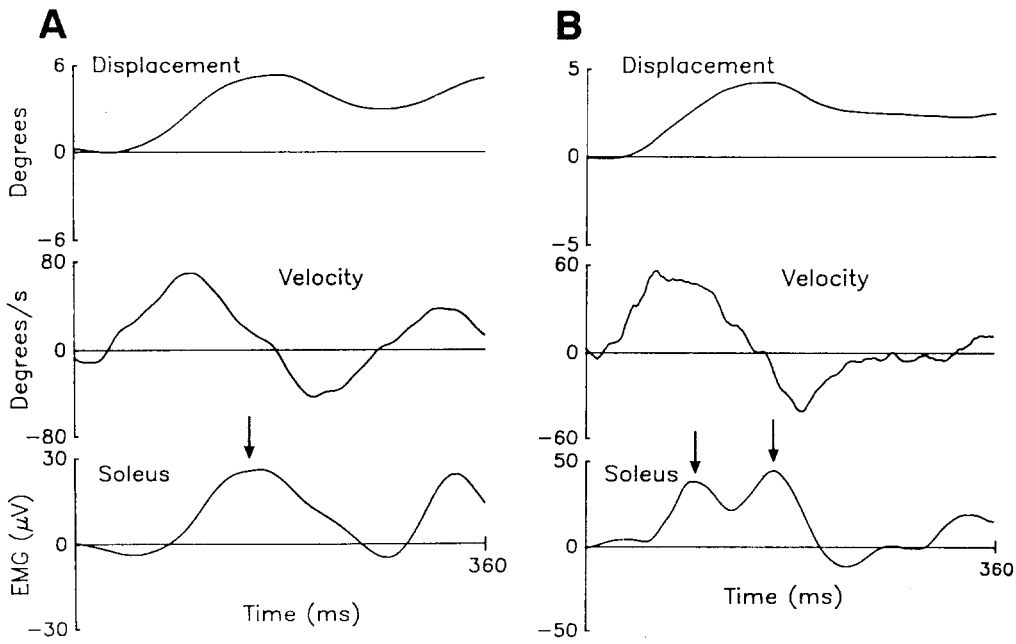

Fig. 4A, B. Two subjects (shown in **A** and **B** respectively) demonstrate differences in the response observed between subjects. All traces represent subtracted responses (as in **3B**). The velocity traces were obtained by differentiating the displacement (top traces) after digital filtering. The soleus muscle responded at a short latency in both cases, but the subject in **A** showed a single soleus burst while the subject in **B** showed a double burst (EMG bursts are indicated with arrows). Interestingly, the overall profile of both responses resembled the velocity of the disturbance qualitatively

subsequent inhibitory and excitatory responses all qualitatively resembled the velocity profile.

The peak displacement generated was a function of the time in the step cycle (Fig. 5). Larger displacements could be generated in the early part of stance when the plantarflexor torque was low. As the ankle rotated over the foot, the moment arm length of the disturbance force about the ankle joint is reduced and the device could no longer generate enough torque to counteract the increasingly high plantarflexor torque of push-off, even though the force generated was sufficient to lift the body.

Fitting the models to the data

Three examples of individual fits are shown in Fig. 6, for a duration of 200 ms starting from the beginning of the EMG response. These represent a range of extremely good (>90% variance accounted for) to moderately good (between 70–80% variance accounted for) fits using the velocity sensitive model alone, and an optimal pure delay.

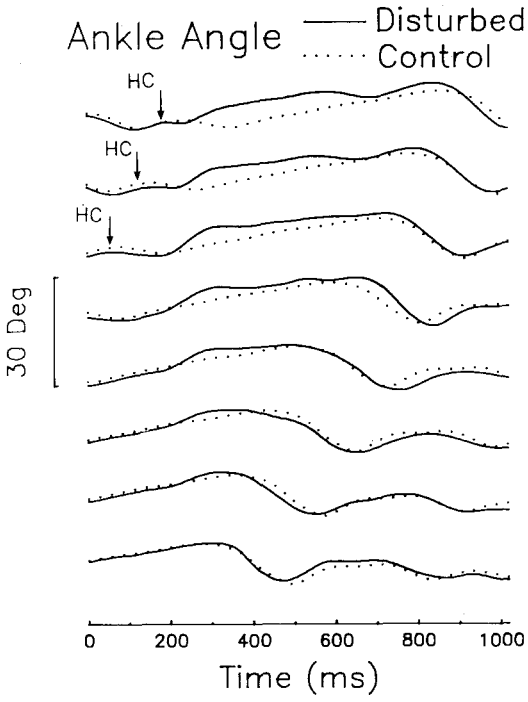

Fig. 5. The pneumatic device was effective at dorsiflexing the ankle only during the first 50% to 60% of the stance phase. Each of the traces represent the trajectory of the ankle angle obtained from a particular part of the stance phase in one subject, starting with disturbances that occurred in the beginning of stance (top trace) to those that occurred at the end of stance (bottom trace). The solid lines represent averages of disturbed trials (approximately 20) while the dotted lines represent the corresponding trajectories of undisturbed walking (average of more than 100 steps). Each trace begins at the time the electrical trigger is sent to the solenoid valve. Note that there is a pure delay of approximately 200 ms between the beginning of the electrical trigger and the application of a perturbation (a deviation between the solid and dotted lines). The data are shown for a time interval of 1 s, much longer than the interval used in subsequent analyses, to illustrate the total time course of the response. Note also that the total cycle duration was somewhat longer than 1 s in this particular subject (approximately 1.3 s)

A pure delay between the kinematic and EMG responses substantially improved the fit for all subjects. The optimal delay, averaged across 7 subjects is shown in Fig. 7A for each of the models. These delays are in the range expected for segmental reflexes of the fastest conducting fibres. The optimal delay did not vary systematically with time in the walking cycle. An average of 70% of the variance could be accounted for by the velocity sensitive model alone (Fig. 7B). The addition of a length sensitive term improved the fits significantly ($p<0.05$,

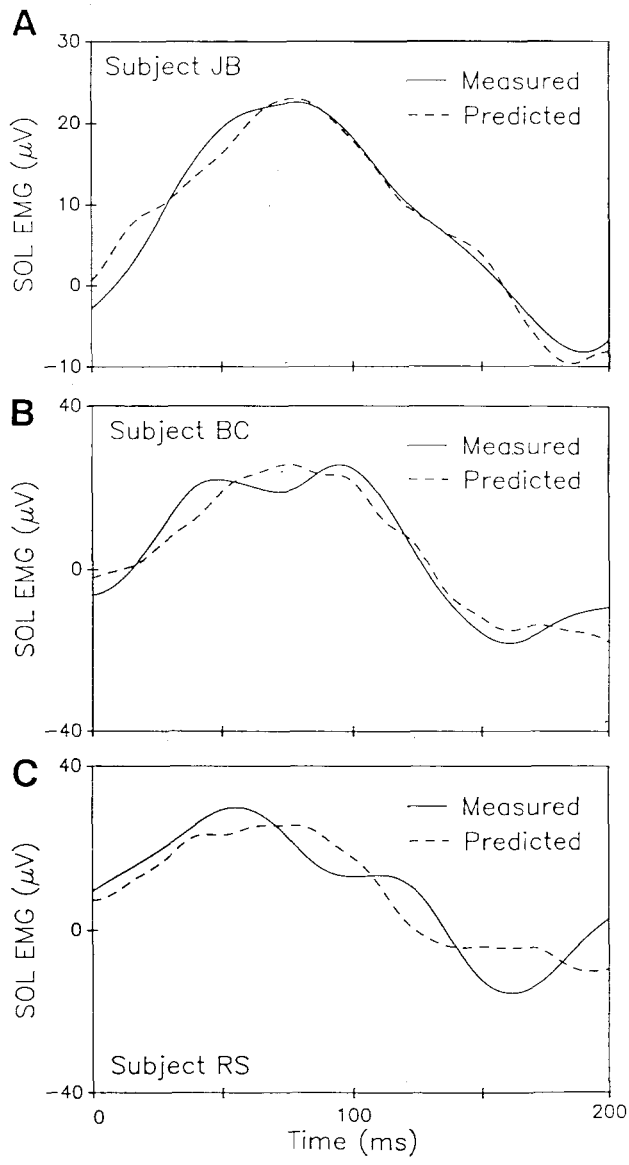

Fig. 6A–C. The response of the SOL muscle was well predicted by the velocity sensitive model (i.e., model 1) in most cases. Examples from three different subjects are shown for a duration of 200 ms starting from the beginning of the EMG response. In **A**, 97% of the variance was accounted for by this model. In **B**, the SOL responded with a double burst which was not reflected in the velocity trajectory. This only resulted in a slightly worst fit than that in **A** (93% variance accounted for). In **C**, the response deviated from the velocity somewhat more, resulting in 79% of the variance accounted for by this model

repeated measures ANOVA). Further addition of an acceleration term did not improve the fits significantly. The response of isolated muscle spindles from cats is well approximated by a first order lead system with a length and velocity dependence and a break frequency (i.e., the frequency at which the contribution of length and velocity is equal) of approximately 2 Hz (Matthews and Stein 1969). Human muscle spindles behave in a similar way (Poppele and Kennedy 1974). The coefficients a_1 and a_2, identified based on model 2, were used to determine the break frequency from the current data using the following equation:

$$f_b = a_1/2\pi a_2 \quad (5)$$

where f_b is the break frequency. Seventy percent of all trials provided break frequencies between 0.5 and 4 Hz, close to that reported for isolated muscle spindles.

The duration over which the fits were obtained was varied systematically from 100 to 250 ms measured from the beginning of the response. No significant degradation in the fits were observed until more than 200 ms of data were included (Fig. 7C). This suggests that triggered responses of longer latency and more distant pathways (e.g. Nashner 1980; Marsden et al. 1983; Dietz e al. 1987) start to emerge after 200 ms.

Since the velocity-sensitive model accounted for most of the variance, this model was used for the following calculations. The velocity coefficient (i.e., a_2 in Eq. 1) can be regarded as a measure of reflex gain. Reflex gain is operationally defined here as the relationship between the

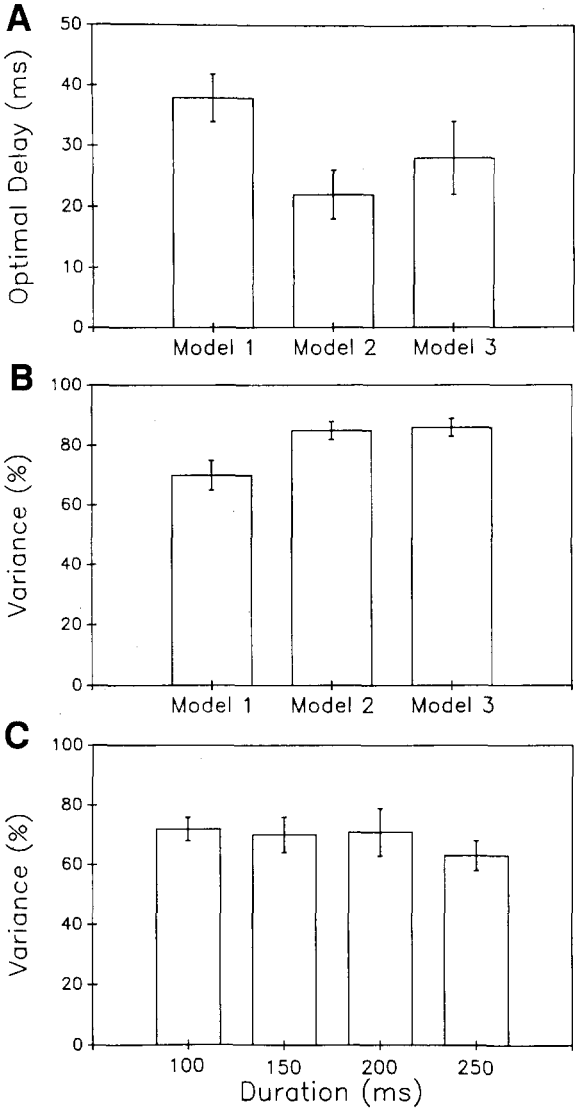

Fig. 7. A The optimal delay, averaged across all subjects ranged between 22 to 38 ms for the three models, well within that expected for a segmental reflex. One standard error is shown about the mean. **B** Most of the variance (70%) in the SOL EMG was accounted for by model 1, which was sensitive to velocity alone. Adding a length dependence (model 2) reduced the unexplained variance by a further 15%, while adding an acceleration dependence did not significantly improve the fits further. **C** The SOL EMG response was well predicted by the velocity sensitive model (model 1) over a wide range of response durations. Deteriorations in the fits were noted only when more than 200 ms of data were included

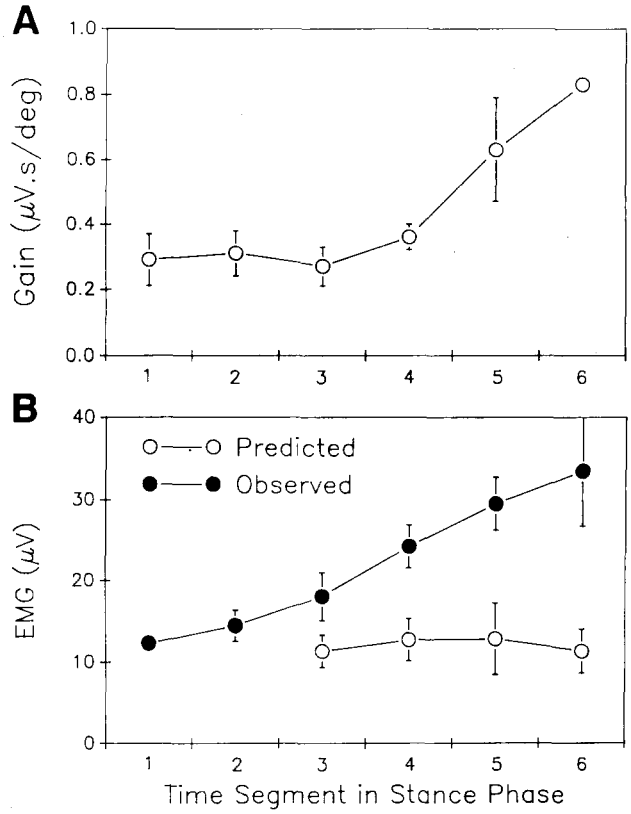

Fig. 8. A Coefficient a_2 in model 1 can be regarded as a measure of reflex gain. This gain increased as a function of time in the stance phase. Time segment one corresponds to the beginning of the stance phase. Open circles represent the mean across 7 subjects ± 1 standard error. **B** Multiplying each of the gains from part **A** with the corresponding velocity of ankle dorsiflexion in undisturbed walking provides an estimate for the contribution of a velocity-sensitive element to the activation of the SOL muscle (open circles). The solid circles represent the SOL EMG amplitude associated with normal walking, averaged across the subjects (± 1 standard error). Between 30% and 60% of the SOL EMG in early stance is estimated to result from muscle stretch. No estimate is shown for the first and second segments of the stance phase because the SOL was shortening during this time as the foot is lowered to the ground

input (the velocity of stretch) and the output (the response of the SOL as reflected in the EMG). This gain, shown averaged across the seven subjects in Fig. 8A, clearly increases over the early part of the stance phase. When this gain is multiplied by the corresponding velocity of ankle dorsiflexion during the stance phase in undisturbed walking, the contribution of a velocity sensitive element is obtained (Fig. 8B, open circles). We estimate that afferent activity associated with stretch velocity contributes to between 30% and 60% of the amplitude and 46% of the area of SOL EMG (filled circles in Fig. 8B) during early stance. Note that the SOL shortens during the first 100 ms of the stance phase as the foot is lowered to the ground. During this period of muscle shortening, no contribution from a velocity-sensitive element is expected.

Discussion

Technical considerations

The use of transient inputs such as a ramp have been criticized for: 1) being limited in content to lower frequencies, and 2) generating voluntary responses (Hunter and Kearney 1982). Our primary aim here was to estimate the contribution of peripheral afferents during normal walking. The transient inputs need only to contain frequency components in the range normally encountered during walking, which are generally very low (Winter 1987). The disturbances applied generated peak velocities ranging from 40 to 100 deg/s of dorsiflexion, similar to velocities normally experienced by the SOL in the stance phase. The response of most subjects were well fitted with the simplest model sensitive to velocity alone (Eq. 1 in the Methods). Voluntary responses did not appear until after 200 ms from the beginning of the response, after which the fits started to deteriorate. Two subjects, however, did show earlier responses (after 100 ms) that deviated from the velocity profile, suggesting that voluntary or triggered responses were of substantial size in these two subjects. Disturbance patterns of a more random nature would be desirable to minimize voluntary responses (Hunter and Kearney 1982). This remains technically difficult to elicit reliably during walking, but would be useful in the future to verify the current findings.

The identification method used here assumes that the system is linear about each operating point (i.e. each time segment in the step cycle). The time segments were 60 to 70 ms in duration, on average. Over this time span in walking, the background EMG in the SOL varied by 7 μV and the ankle angle by 2 degrees, on average. Based on the findings obtained under isometric conditions, such small variations in position and activation levels were not associated with large changes in the reflex amplitude (Weiss et al. 1986). Moreover, if the responses were highly non-linear, linear models such as those used here would not be expected to provide good fits to the data.

The responses from the TA were not analyzed further because they were not central to the questions addressed in this paper. From a functional point of view, however, the TA burst is quite interesting. The mechanical disturbance created by lifting the forefoot during stance has a tendency to cause a posterior sway of the body. To maintain forward progression in walking, the functionally appropriate response would be to activate the TA. This functionally appropriate response appeared after the initial stretch response in the SOL, in a fashion analogous to those reported for a similar disturbance applied during standing (Nashner 1982).

What is the origin of the response?

A mechanical disturbance of this nature probably activates a variety of sensory afferents. Muscle stretch could activate both primary and secondary muscle spindle afferents as well as Golgi tendon organs. Pressure applied to the forefoot by the disturbance likely activates cutaneous afferents, and the resulting joint motion may also activate joint receptors. The earliest responses are most likely of segmental origin, although the population of afferents responsible is more difficult to determine.

The early responses (recorded within 200 ms from the beginning of the response) resembled the velocity profile very closely, often reflecting small irregularities of the disturbance, not unlike those reported under static and slow dynamic conditions (Gottlieb and Agarwal 1979; Kearney and Hunter 1983). The early response was delayed with respect to the velocity profile by an average of 38 ms, well within the conduction time expected for large afferent fibres. Moreover, the coefficients identified based on model 2 agreed well with that expected of muscle spindles (i.e., a break frequency between 0.5 to 4 Hz). While it is impossible to determine definitively which afferents are responsible for these responses, the velocity and length sensitive nature of the responses suggest that group Ia and II afferents might be primarily responsible (Matthews and Stein 1969). The monosynaptic excitation from group II afferents to motoneurons is considerably weaker than that of the group Ia (Lundberg et al. 1977) and probably contributed little to the early responses seen here. Whether the more complex polysynaptic pathways from group II afferents (Lundberg et al. 1987) contributed significantly to these responses is unclear, and beyond the scope of this paper. It is likely, however, that the responses observed here were dominated by velocity sensitive group Ia fibres, because the majority of the variance in the EMG response could be accounted for by the velocity of the disturbance alone. Indeed, it is interesting that the reflex gain of the SOL increased over the early part of the stance phase in a ramp-like manner, qualitatively similar to the behavior of the H-reflex (Capapday and Stein 1986; Crenna and Frigo 1987).

Not all subjects showed responses that faithfully reflected the velocity profile. The most common deviation was a double-burst of EMG that was not preceded by any such variation in the velocity of the disturbance (e.g., Fig. 4B). The second burst of EMG could be associated with afferent populations that have a slightly slower conduction velocity than the Ia fibres such as the group II (Matthews 1986) or large cutaneous afferents. Alternatively, small mechanical vibrations that might have

accompanied the disturbance but were not measurable with the goniometer could trigger later responses, such as those reported for mechanical disturbances of the wrist (Hagbarth et al. 1981). Finally, long-loop reflexes or triggered responses could contribute to the second burst of EMG (Marsden et al. 1983). Interestingly, the general profile of the response remained qualitatively similar to the velocity of the disturbance (Fig. 6B).

Functional significance

The early stretch induced response is of short latency (average latency of 38 ms) and the gain of the reflex arc is modulated in a similar way to that of the H-reflex, at least through the early part of the stance phase tested here. These findings are in agreement with those obtained using tendon tap (Llewellyn et al. 1986), and other mechanical devices to induce stretch (Yang et al. 1990). The results are somewhat different from those obtained by Dietz et al. 1987. Dietz and colleagues applied mechanical perturbations by suddenly accelerating or decelerating a treadmill. The velocity of stretch applied to the ankle with their method was similar to those used here, yet they failed to elicit the short latency responses during walking that we saw regularly. Reflexes associated with muscle spindle behavior (i.e., the H-reflex, tendon jerks and stretch reflexes) in walking have all shown that the reflex is inhibited at the beginning of the stance phase and again in the swing phase (Capaday and Stein 1986; Crenna and Frigo 1987; Llewellyn et al. 1986). Dietz and colleagues applied disturbances at the very beginning of the stance phase, a time when the reflex gain is extremely low. Perhaps the low reflex gain at this time resulted in the absence of an early response. However, even during the very early part of the stance phase, short latency responses were clearly elicited in most of our subjects. The reason for the discrepancy between the two studies remains unclear.

We suggest that a velocity sensitive element contributes substantially to the activation of the SOL muscle through the early stance phase when the muscle is actively stretched. The current estimates suggest that between 30% to 60% of the early SOL activity could come from velocity-sensitive peripheral afferents. These estimates must be qualified by the limitations of this study. First, it is assumed here that the responses are linear. We cannot rule out the possibility that these responses reflect more complex, non-linear phenomenon. Secondly, it is possible that the afferent information associated with normal walking is processed differently from that associated with unexpected disturbances. If this is the case, the direct application of gains identified from unexpected perturbations to normal undisturbed walking may be inaccurate. Finally, the contribution from other afferents cannot be estimated at this time, but could further contribute to the activation of the SOL. In spite of these difficulties, it is interesting to note that Severin (1970) predicted from a completely different approach of blocking conduction in the fusimotor axons, that muscle spindle acitivity contributed approximately 50% to the activation of the triceps surae group during walking in the mesencephalic cat. His estimate falls within the range predicted here.

A quantitative value has been placed on the relative contribution of peripheral versus central mechanisms in locomotion, albeit limited to the activation of a single muscle. We suggest that peripheral afferents not only signal unexpected disturbances (Dietz et al. 1987) and allow transition between phases in walking (Grillner and Rossignol 1978; Pearson and Duysens 1976), but they also contribute to the activation of a muscle on an on-going basis during walking.

Acknowledgements. This work was supported by the Medical Research Council of Canada. JF Yang was a postdoctoral fellow of the Alberta Heritage Foundation for Medical Research.

References

Akazawa K, Aldridge JW, Steeves JD, Stein RB (1982) Modulation of stretch reflexes during locomotion in the mesencephalic cat. J Physiol (Lond) 329: 553–567

Aldridge JW, Stein RB (1982) Nonlinear properties of stretch reflex studied in the decerebrate cat. J Neurophysiol 47: 179–192

Capaday C, Stein RB (1986) Amplitude modulation of the soleus H-reflex in the human during walking and standing. J Neurosci 6: 1308–1313

Crenna P, Frigo C (1987) Excitability of the soleus H-reflex arc during walking and stepping in man. Exp Brain Res 66: 49–60

Delcomyn F (1980) Neural basis of rhythmic behavior in animals. Science 210: 492–498

Dietz V, Quintern J, Sillem M (1987) Stumbling reactions in man: significance of proprioceptive and pre-programmed mechanisms. J Physiol (Lond) 386: 149–163

Gottlieb GL, Agarwal GC (1978) Dependence of human ankle compliance on joint angle. J Biomech 11: 177–181

Gottlieb GL, Agarwal GC (1979) Response to sudden torques about ankle in man: Myotatic reflex. J Neurophysiol 42: 91–106

Grillner S (1981) Control of locomotion in bipeds, tetrapods and fish. In: Brooks VB (ed) Handbook of physiology, Sect I, Vol II, Part 2. American Physiological Society, Bethesda, pp 1179–1236

Grillner S, Rossignol S (1978) On the initiation of the swing phase of locomotion in chronic spinal cats. Brain Res 146: 269–277

Hagbarth K-E, Hägglund JV, Wallin EU, Young RR (1981) Grouped spindle and electromyographic responses to abrupt wrist extension movements in man. J Physiol (Lond) 312: 81–96

Hunter IW, Kearney RE (1982) Dynamics of human ankle stiffness: variation with mean ankle torque. J Biomech 15: 747–752

Kearney RE, Hunter IW (1983) System identification of human triceps surae stretch reflex dynamics. Exp Brain Res 51: 117–127

Llewellyn M, Prochazka A, Vincent S (1986) Transmission of human tendon jerk reflexes during stance and gait. J Physiol (Lond) 382: 82P

Lundberg A, Malmgren K, Schomburg ED (1977) Comments on reflex actions evoked by electrical stimulation of group II muscle afferents. Brain Res 122: 551–555

Lundberg A, Malmgren K, Schomburg ED (1987) Reflex pathways from group II muscle afferents. 1. Distribution and linkage of reflex actions to α-motoneurones. Exp Brain Res 65: 271–281

Magladery JW, Porter WE, Park AM, Teasdall RD (1951) Electrophysiological studies of nerve and reflex activity in normal man. IV. The two-neurone reflex and identification of certain action potentials from spinal roots and cord. Bull John Hopkins Hosp 88: 499–519

Marsden CD, Rothwell JC, Day BL (1983) Long-latency automatic responses to muscle stretch in man: origin and function. In: Desmedt JE (ed) Motor control mechanisms in health and disease. Raven, New York, pp 509–539

Matthews PBC (1986) What are the afferents of origin of the human stretch reflex, and is it a purely spinal reaction? Prog Brain Res 64: 55–66

Matthews PBC, Stein RB (1969) The sensitivity of muscle spindle afferents to small sinusoidal changes of length. J Physiol (Lond) 200: 723–743

Nashner LM (1980) Balance adjustments of humans perturbed while walking. J Neurophysiol 44: 650–664

Nashner LM (1982) Adaptation of human movement to altered environments. Trends Neurosci 5: 358–361

Pearson KG, Duysens J (1976) Function of segmental reflexes in the control of stepping in cockroaches and cats. In: Herman RM, Grillner S, Stein PSG, Stuart DG (eds) Neural control of locomotion. Plenum Press, New York, pp 519–537

Poppele RE, Kennedy WR (1974) Comparison between behavior of human and cat muscle spindles recorded in vitro. Brain Res 75: 316–319

Prochazka A, Westerman RA, Ziccone SP (1976) Discharges of single hindlimb afferents in the freely moving cat. J Neurophysiol 39: 1090–1104

Rossignol S, Lund JP, Drew T (1988) The role of sensory inputs in regulating patterns of rhythmical movements in higher vertebrates. In: Cohen A, Rossignol S, Grillner S (eds) Neural control of rhythmic movements in vertebrates. John Wiley and Sons, New York, pp 201–283

Severin FV (1970) The role of the gamma motor system in the activation of the extensor alpha motor neurones during controlled locomotion. Biophysics 15: 1138–1144

Stein RB, Bobet J, Oguztoreli MN, Fryer MW (1988) Kinetics relating calcium and force in skeletal muscle. Biophys J 54: 705–717

Weiss PL, Kearney RE, Hunter IW (1986) Position dependence of stretch reflex dynamics at the human ankle. Exp Brain Res 63: 49–59

Winter DA (1987) The biomechanics and motor control of human gait. University of Waterloo Press, Waterloo

Wolf H, Pearson KG (1988) Proprioceptive input patterns elevator activity in the locust flight system. J Neurophysiol 59: 1831–1853

Yang JF, Winter DA. Wells RP (1990) Postural dynamics of walking in humans. Biol Cybern 62: 321–330