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RESEARCH NOTE

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On the soleus H-reflex modulation pattern during walking

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Abstract In a recent paper it was claimed that in the majority (9/15) of subjects studied the soleus H-reflex increases progressively during the swing phase of walking. This pattern was at odds with our numerous observations made since 1986, as was the very large proportion of subjects reported to exhibit this pattern. We therefore reinvestigated the issue in an extensive series of experiments and detailed subsequent analysis on 21 subjects. In most subjects (13/21) the soleus H-reflex was completely inhibited during most or all of the swing phase (group A). In 8/21 subjects (group B) there was a small H-reflex mean 16% (SD=10.6%) of the value in quiet standing present during most or all of swing, but there was no systematic modulation pattern; the reflex amplitude fluctuated in a seemingly random manner. The difference between the two somewhat arbitrary groups could not be explained on the basis of greater electromyographic activity in the tibialis anterior (TA) during the swing phase or at the time of heel contact. However, by normalizing the mean level of TA activity to the peak level, the ratio was significantly greater for the group A subjects. This highlights the importance of reciprocal inhibition in accounting for the suppression of the soleus H-reflex in swing. In the discussion we emphasize that the presence of a small H-reflex during swing in the group B subjects is unlikely to have any functional role. What is of functional importance is the strong inhibition of the H-reflex during swing which reflects the ensemble of neural mechanisms at play to prevent the unwanted activation of the powerful ankle extensor muscles.

Keywords H-reflex · Monosynaptic reflex · Human walking · Spinal cord circuits

Introduction

During human walking the soleus H-reflex increases progressively during the stance phase nearly in parallel with the soleus electromyographic (EMG) activity (Capaday and Stein 1986) and is suppressed or markedly reduced during the swing phase while the tibialis anterior (TA) is active (Capaday and Stein 1986; Yang and Whelan 1993; Lavoie et al. 1997; Andersen and Sinkjaer 1999; Schneider et al. 2000). The modulation pattern of the H-reflex during normal walking thus follows the classic pattern of reciprocal inhibition between antagonistic muscles (Lavoie et al. 1997). Furthermore, as demonstrated in the latter study inhibition of the soleus H-reflex during the swing phase of walking is much more potent than during voluntary and postural activation of soleus's antagonist, the TA. We had also reported that in subjects with an H_{max} to M_{max} ratio of less than 60% during quiet standing (QS) the soleus H-reflex was more likely to be completely inhibited during swing. Contrarily, those with an H_{max} to M_{max} ratio greater than 60% were more likely to have a small H-reflex present during swing. However, there was no systematic pattern of H-reflex modulation during the swing phase; the reflex amplitude fluctuated in a seemingly random manner. In a recent paper, Simonsen et al. (2002) reported that subjects could be classified into two distinct groups on the basis of the H-reflex modulation pattern during swing. In one group the H-reflex was, as they expressed it, suppressed during swing. In the other group, termed less suppressed, the H-reflex was claimed to increase progressively during swing starting from its minimum value at the stance to swing transition (see Fig. 3 in Simonsen et al. 2002). We were surprised by this report as we had not observed the latter

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pattern in our extensive research on this subject (see, for example, Capaday and Stein 1986, 1987a; Lavoie et al. 1997; Schneider et al. 2000). We were also surprised that they reported this to be the case in the majority (9/15) of subjects they studied. We therefore reinvestigated the issue in a new series of extensive experiments and detailed subsequent analysis. We found no subjects with an H-reflex that systematically increases from the beginning of the swing phase to the subsequent heel contact (HC) and confirm that in most subjects (13/21) the H-reflex is completely inhibited during most or all of the swing phase.

Materials and methods

The data reported herein were obtained from experiments on 21 neurologically normal human subjects ranging in age between 22 and 42 years (SD=5.9 years). The study was approved by Laval University's ethics committee. The experimental procedures used in the present study and their neurophysiological basis are described in detail in several publications from this laboratory (Capaday et al. 1995; Capaday 1997; Lavoie et al. 1997; Schneider et al. 2000; Schneider and Capaday 2003). Methodological issues that are of particular relevance to the present study are provided in what follows. Subjects were asked to walk at their own preferred speed on a treadmill (range 3.1–5.1 km/h, mean=4.0 km/h, SD=0.55 km/h). They were instructed to walk without drifting position on the treadmill. This insured highly repeatable cycle times and stance and swing duty cycles. The mean EMG activity of the soleus and TA during the walking cycle were determined several times during the course of the experiment to be sure that the cycle time and duty cycles were repeatable. The data are plotted in this paper as the time at which the H-reflex was actually elicited in the step cycle, not the time of stimulation. All automated measurements of H-reflex amplitude were corroborated by visual inspection of each record. This insures that all reported small amplitude values correspond to a genuine small H-reflex, not random EMG activity or noise. The average of four H-reflexes was obtained at each tested point in the step cycle.

Results

The H-reflex modulation pattern of all 21 subjects studied is shown in Fig. 1 on a normalized time scale, along with the amplitude of the H-reflex during QS. Note how during early and midstance the H-reflex is lower than during QS despite higher EMG activity, as first reported by Capaday and Stein (1986). The subjects were divided according to whether or not there occurred a complete inhibition during some part of the swing phase. In 13/21 subjects (62% of the total) the H-reflex was completely inhibited

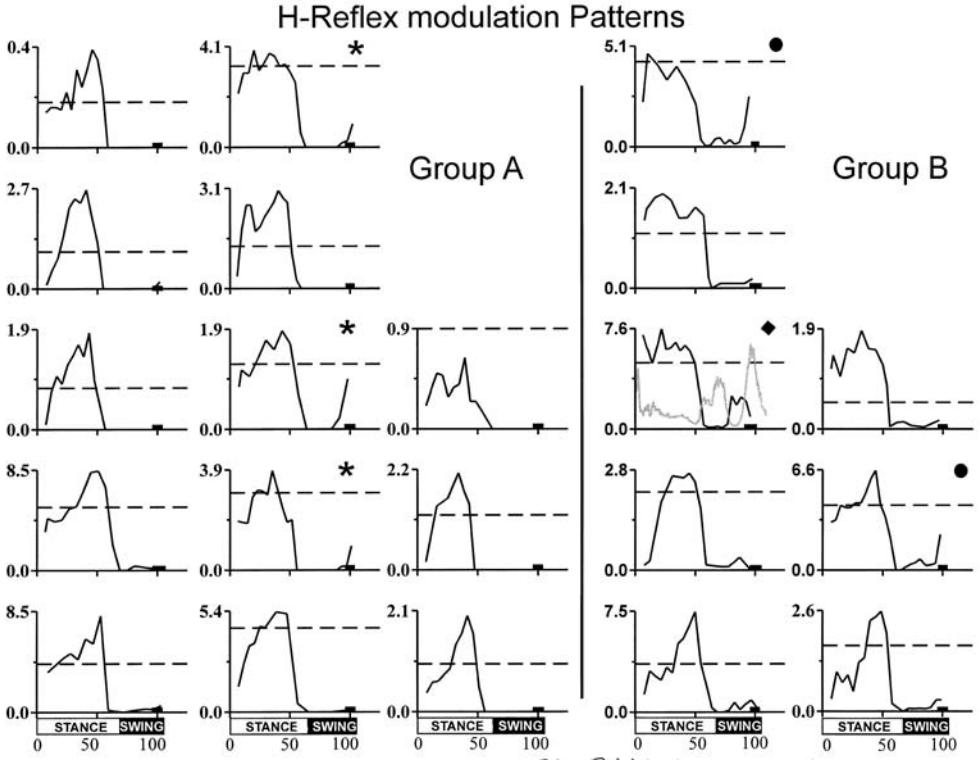

Fig. 1 The soleus H-reflex modulation pattern of the 21 studied subjects during the walking cycle. The walking cycle time was normalized. The variability of heel contact (HC) time is represented in each case by the length of the *black bar* above the time axis. Estimates of the stance and swing phase durations are also shown. The *ordinate* of each graph represents the peak-to-peak amplitude of the H-reflex in millivolts. The *horizontal dashed line* in each graph represents the value of the soleus H-reflex during quiet standing. For the subject in group B identified by a *black diamond symbol* the tibialis anterior (TA) electromyographic (EMG) pattern is also shown along with the soleus H-reflex modulation pattern. *Star symbol* Subjects where the H-reflex was completely inhibited during most of swing and began to increase just before or at HC. *Black circle* Subjects where the H-reflex began to increase in late swing just prior to or at HC

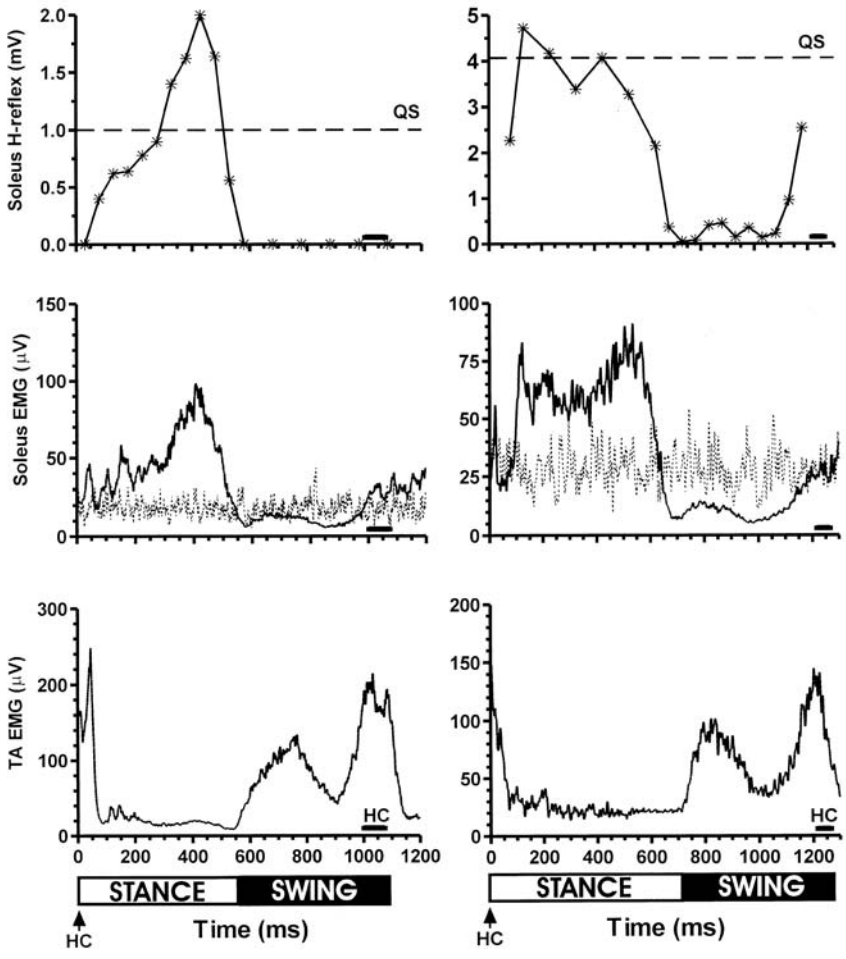

Fig. 2 Representative examples of the H-reflex modulation pattern of a group A (*left*) and a group B (*right*) subject during a walking cycle, along with the mean rectified and filtered EMG activity of the soleus and TA. The variability of HC time is indicated by the length of the *horizontal black bars* just above the time axis. Estimates of the stance and swing phase durations are also shown. The level of EMG activity in the soleus during quiet standing (*QS*) is displayed with *lighter dotted traces*. Note the gradual ramp like increase of soleus activity during stance in the group A subject and the more abrupt increase of activity in the group B subject

during some portion of swing; these will be arbitrarily designated group A. In 8 of the group A subjects the H-reflex was completely inhibited throughout the swing phase. This stands in marked contrast to the data reported by Simonsen et al. (2002) in which none of their subjects had a completely suppressed H-reflex in swing. In 3 subjects of group A, the H-reflex was completely inhibited during most of swing and began to increase just before or at HC. These subjects are identified with a star symbol in Fig. 1. The remaining 2 subjects in group A had a very small H-reflex (mean 4% of the H-reflex in QS) during most of swing. In 8/21 (38% of the total) a small H-reflex was present throughout the swing phase; these will be designated arbitrarily as group B. On average the H-reflex was about 16% of its value during QS (SD=10.6%; range 6–37%). It should be noted however, that this seemingly high mean value was due to the unrepresentative influence of 3 subjects. In 2 of these subjects, identified by a small black circle in Fig. 1, the H-reflex began to increase in late swing just prior to or at HC. In the other subject, identified by a black diamond symbol, the H-reflex suddenly increased at about the time of midswing. This unusual occurrence will be dealt with in greater detail below. What is important is that there is no systematic pattern to the H-reflex during the swing phase in the group B subjects. Furthermore, averaging the H-reflex modulation pattern across subjects, as was done by Simonsen et al. (2002), would lead to an artificial modulation profile, which is neither representative of individuals nor the population. Linear regression analysis showed that in 4 of the 8 subjects in group B there was no significant correlation between time in the swing phase and H-reflex amplitude. In the remaining 4 there was a significant, but weak correlation ($0.4<r^2<0.6$) between the variables. However, this is a spurious result because in 3 subjects a sudden rise of the H-reflex (i.e., a single point) at the end of swing or early stance contributes to making the correlation significant. As stated above, a sudden rise of the H-reflex at the end of swing or early stance is not particular to group B subjects and is therefore not a discrimination criterion.

The H-reflex modulation pattern of one group A subject and one group B subject during a walking cycle are shown in greater detail in Fig. 2, along with the average soleus and TA EMG patterns. In both cases the H-reflex modulation pattern during stance is closely related to the average soleus EMG activity and is strongly inhibited during activity of the TA in swing. Several other observations can be made from these figures. The H-reflex of the group A subjects is on average lower in the

first 10% of the walking cycle, i.e., near the time of HC, than is that of the group B subject (peak to peak amplitudes, 1.3 mV, SD=1.27 mV versus 2.4 mV, SD=2.06 mV), but so too is the level of soleus EMG activity (mean=27.6 μV, SD=15.6 μV versus mean=50.8 μV, SD=22.9 μV). In fact, there was a tendency for a low H-reflex near the time of HC to be associated with a gradual, ramp-like, increase of soleus EMG activity (Fig. 2 *left*); whereas, a higher H-reflex near the time of HC tended to be associated with a more abrupt onset of soleus EMG activity (Fig. 2 *right*).

We attempted to understand what physiological variables may predict whether a subject will display a group A or B modulation pattern during swing. The TA motor output during the swing phase of walking is characterized by two bursts of activity (Fig. 2), with the second larger burst beginning just before HC. When considering the absolute EMG values, the mean size of neither burst was predictive of the H-reflex modulation pattern during swing. In the group A subjects the mean value of the first TA burst was 57.1 μV (SD=17.5 μV) versus a mean value of 50.8 μV (SD=12.3 μV) for the group B subjects. The mean value of the higher amplitude second TA burst was 138.7 μV (SD=54.8 μV) for the group A subjects and 157.8 μV (SD=49.6 μV) for the group B subjects. However, when the mean values of each burst were normalized respectively to the peak value of the second TA EMG burst, the results were consistent with the idea that the extent of the H-reflex suppression in swing is the result of reciprocal inhibition. For the group A subjects this ratio was 0.41 versus 0.28 for the group B subjects, meaning that the first TA burst was larger relative to the peak value of the second TA burst in the group A subjects. Similarly, the mean value of the second TA EMG burst relative to its peak value was greater in the group A subjects (ratio=0.63) than in the group B subjects (ratio=0.45). This indicates that in the group A subjects the second TA burst may be more sustained than in group B subjects. In any case, the importance of reciprocal inhibition in suppressing the soleus H-reflex during swing and its temporal association with TA α-motoneuron activity, as determined from the electromyograph, is strikingly illustrated in the records of the subject identified by a black diamond in Fig. 1. The unusual aspect of this subject's TA EMG pattern was that there occurred a clear pause of about 80 ms between the first and second burst of activity. Coincident with this, the soleus H-reflex suddenly increased and remained elevated until the onset of the second TA burst when it was once again reduced.

The H_{max} to M_{max} ratio measured during QS was on average lower (38%) for the group A subjects compared to that for the group B subjects (47%). The difference in the present sample, however, did not reach accepted statistical significance levels (Mann-Wittney *U*-test, *P*=0.25), but nonetheless the difference was in the same direction as that reported by Lavoie et al. (1997).

Discussion

The purpose of this study was to examine in detail the report of Simonsen et al. (2002) that there exist two patterns of H-reflex modulation during human walking, especially the claim that in most of their subjects (9/15) the H-reflex increases progressively during swing. In the event, we have not observed in any subject a progressively increasing H-reflex during the swing phase. Moreover, it is clear that in the majority of subjects (group A) the H-reflex is completely inhibited during most or all of the swing phase. This contrasts sharply with the findings of Simonsen et al. (2002) who did not report a complete inhibition of the H-reflex in any of their subjects. In subjects that have a residual H-reflex in the swing phase (group B), the reflex amplitude varies in a random manner and there is no systematic pattern that characterizes this group. There is thus no facilitation of the H-reflex during swing as reported by Simonsen et al. (2002). On the contrary the swing phase is characterized by strong inhibition in all subjects.

In 3 out of 13 subjects in group A the H-reflex began to increase just prior to HC and similarly in 2 of the 9 group B subjects (Fig. 1). This reinforces the point that there is no essential difference between the two groups; the H-reflex is strongly inhibited during swing. What may underlie this increase of the H-reflex is an early starting or rapidly rising subthreshold locomotor drive potential (Jordan 1983). Lastly, Simonsen et al. (2002) stated that their reported modulation profiles did not depend on their method of normalization, described in Simonsen and Dyhre-Poulsen (1999). Indeed, our results are independent of any amplitude normalization procedure one cares to adopt, as the modulation profile would not be changed by amplitude scaling. Thus, the differences between the results of Simonsen et al. (2002) and the present ones are not due to differences in data treatment.

Functional considerations

The possible neural mechanisms involved in inhibition of the soleus H-reflex and its functional significance have been discussed by Lavoie et al. (1997). The strong inhibition during the swing phase as determined by measurements of the H-reflex reflects the need to suppress the powerful stretch reflexes of the ankle extensors during the rapid ankle dorsiflexion. In fact, as shown in the study of Lavoie et al. (1997) reciprocal inhibition of the ankle extensors is stronger in tasks involving stretch of these muscles and it is most powerful during the swing phase of walking (see details in Lavoie et al. 1997). This is a very different functional interpretation from that offered by Simonsen et al. (2002) who suggested that increasing excitability of the H-reflex during the swing phase reflects that the ankle extensor stretch reflex contributes to absorbing the impact at heel strike. The impact at HC is in fact absorbed by an eccentric contraction of the TA and the ankle extensors,

particularly the soleus, are in fact shortening at this time. Thus, there cannot be a stretch reflex contribution at this time in the step cycle. More importantly, a stretch reflex in the ankle extensors at the time of HC if strong enough would slap the foot toward the ground, or may destabilize equilibrium by moving the body backward. A low value of the H-reflex at HC is thus expected and indeed observed. Additionally, biomechanical considerations suggest that the ankle extensor stretch reflex sensitivity needs to be reduced in stance (Morin et al. 1982; Capaday and Stein 1986) in order to allow for controlled rotation of the body about the ankle joint. The reduction of the H-reflex in the early part of the stance phase of walking compared to standing (Capaday and Stein 1986) strongly supports this hypothesis. The finding that subjects with suppressed H-reflexes, termed group S by Simonsen et al. (2002), show a greater knee extensor moment some 150 ms after HC is incidental and thus unrelated to neural events occurring during swing.

Simonsen et al. (2002) suggested that in the studies of Capaday and Stein (1987a) and Edamura et al. (1991) the H-reflex was shown to be facilitated during swing. This is incorrect and stems from a misreading of the graphs presented in the figures. For example, in Fig. 3 of Capaday and Stein (1987a) slightly more than one walking and running cycle are plotted, with the H-reflex beginning to rise near the time of HC in each case. Simonsen et al. (2002) also erroneously stated that Capaday and Stein (1989) had shown that postsynaptic inhibition could not completely suppress the H-reflex. What we had in fact shown was that postsynaptic inhibition could not change the size of the monosynaptic reflex independently of the activity level of the α-motoneuron pool, a conclusion whose theoretical basis (Capaday and Stein 1987b) has been corroborated in several subsequent studies (Holt and Koch 1997; Capaday 2002; Chance et al. 2002).

Concluding remarks

The modulation pattern of the H-reflex during normal walking follows the classic pattern of reciprocal inhibition between antagonistic muscles (Lavoie et al. 1997). The present observation showing that the ratio of mean TA activity in the first burst relative to the peak value of the second burst is greater in group A than in group B subjects further reinforces this notion. A correct and detailed understanding of the H-reflex modulation pattern during walking is important for properly interpreting neurophysiological results. For example, the H-reflex can provide information on subthreshold events during walking (see, for example, Schneider and Capaday 2003) and can also be used to infer changes of presynaptic inhibition of the Ia-afferent terminal projecting to the α-motoneurons (Capaday 1997; Pierrot-Deseilligny 1997). Sound interpretation of such results is predicated on a proper understanding of the normal H-reflex modulation pattern. Likewise, sound interpretation of results obtained in neurological patients (Yang et al. 1991; Faist et al. 1994) is clearly dependent upon comparison with the normal pattern. The latter is simply characterized by an increasing H-reflex during stance, a sudden strong inhibition at the transition from stance to swing, and persisting strong inhibition during swing.

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