Experimental Brain Research (2019) 237:777–791
https://doi.org/10.1007/s00221-018-5459-0

RESEARCH ARTICLE

H-reflex modulation preceding changes in soleus EMG activity during balance perturbation

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Received: 24 June 2018 / Accepted: 19 December 2018 / Published online: 2 January 2019

Abstract

When balance is compromised, postural strategies are induced to quickly recover from the perturbation. However, neuronal mechanisms underlying these strategies are not fully understood. Here, we assessed the amplitude of the soleus (SOL) H-reflex during forward and backward tilts of the support surface during standing ($n = 15$ healthy participants). Electrical stimulation of the tibial nerve was applied randomly before platform tilt (control) and 0, 25, 50, 75, 100 or 200 ms after tilt onset. During backward tilt, a significant decrease in H-reflex amplitude was observed at 75, 100 and 200 ms. The onset of the decreased H-reflex amplitude significantly preceded the onset of the SOL EMG decrease (latency: 144 ± 16 ms). During forward tilt, the amplitude of the H-reflex increased at 100 and 200 ms after tilt onset. The onset of H-reflex increase did not occur significantly earlier than the onset of the SOL EMG increase (127 ± 5 ms). An important inter-subject variability was observed for the onset of H-reflex modulation with respect to EMG response for each direction of tilt, but this variability could not be explained by the subject's height. Taken together, the results establish the time course of change in SOL H-reflex excitability and its relation to the increase and decrease in SOL EMG activity during forward and backward tilts. The data presented here also suggest that balance mechanisms may differ between forward and backward tilts.

Keywords H-reflex · Perturbation · Postural strategies · Soleus · Electromyography

Introduction

Given the unstable nature of human bipedal stance and locomotion, the central nervous system (CNS) has developed mechanisms to quickly compensate for perturbations of balance and maintain an upright posture. These include afferent feedback mechanisms that ensure postural stability by rapidly triggering muscle responses in different parts of the body following a perturbation (Massion 1992). Many studies have assessed postural responses to forward and backward tilts of the base of support during standing and have characterized various strategies used to compensate for perturbations (Nashner et al. 1989; Nardone et al. 1995; Carpenter et al. 1999; Creath et al. 2008). One of them is the ankle strategy, which involves muscles spanning the ankle joint. In response to forward tilts of the platform (toes down), the center of mass is displaced forward and the activity of soleus (SOL) muscle is increased to move the center of mass backward and regain stability. Conversely, responses to backward tilts (toes up) involve the increased activation of the tibialis anterior (TA) muscle and a pronounced decrease in SOL muscular activity to recover balance (Nashner 1976, 1977; Horak and Nashner 1986). The electromyographic (EMG) responses that underlie the postural reactions following a sudden perturbation of the base of support have been well-described (see Diener et al. 1984; Nardone et al. 1995). These EMG responses include a short latency response (SLR 40–65 ms), medium latency response (MLR 65–100 ms) and long latency response (LLR ≥ 100 ms).

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The neuronal mechanisms responsible for these EMG responses following a perturbation involve both spinal and supraspinal structures, notably at the cortical level (Horak et al. 1990; Schieppati et al. 1995; Allum and Honegger 1998; Bloem et al. 2000; Taube et al. 2006; Fujio et al. 2018; Petersen et al. 2009), but are not yet fully understood. Previous studies have shown that the SLR results from the activation of a monosynaptic spinal reflex pathway and is a response to either a rapid stretch or shortening of the muscle (Schieppati and Nardone 1995). Transcortical pathways involving the primary sensory and motor cortices contribute to the latter part of the MLR and the LLR (Zuur et al. 2009; Taube et al. 2006), together with oligosynaptic excitation of spinal motoneurons through group II and Ib afferents (Grey et al. 2001; Dietz 1998; Schieppati and Nardone 1997). Due to its cortical component, only the LLR is considered functionally relevant in enabling the postural compensatory responses (Taube et al. 2006) and preventing the fall.

Modulation of the SOL Hoffmann reflex (H-reflex) has been used to gain insight into the neuronal mechanisms that help to control gait (Capaday and Stein 1986, 1987) and upright standing or postural tasks (Tokuno et al. 2008). The SOL H-reflex is evoked by electrical stimulation of the tibial nerve (TN) in the popliteal fossa, which results in direct activation of Ia sensory afferents and monosynaptic excitation of SOL motoneurons (Pierrot-Deseilligny and Mazevet 2000; Knikou 2008). H-reflex amplitude depends on afferent inputs, and on descending inputs, notably via presynaptic inhibition. Here, we will assess the modulation of the SOL H-reflex to gain insight on neuronal excitability following perturbations of the base of support.

In the present study, we examined the time course of change in H-reflex amplitude in relation to the changes in SOL EMG activity during balance perturbations. We hypothesized that the amplitude of the H-reflex would be modulated in a direction-dependent manner. Given that SOL EMG is decreased in backward tilt, we postulated that the H-reflex would also be decreased during backward tilt. Conversely, the amplitude of the H-reflex would be increased in the forward condition. We further postulated that changes occurring in the H-reflex would precede changes occurring in the EMG activity, suggesting the involvement of central mechanisms in this modulation. To verify these hypotheses, we quantified SOL H-reflex amplitude as well as EMG background activity before, and at different delays after sudden forward or backward tilts of the base of support on which participants were standing. Preliminary results have been reported in abstract form (Miranda and Barthélemy 2014).

Methods

Participants

Fifteen individuals (nine women and six men, age: 26 ± 7 years; height: 1.72 ± 0.09 m—range 1.6–1.85 m) participated in this study. All participants were healthy, with no known neurological or orthopedic impairment. The experimental protocol was approved by the local ethics committee (Centre for Interdisciplinary Research in Rehabilitation; CRIR) and was conducted in accordance with the Declaration of Helsinki. Participants received oral and written information about the study and then gave their written consent. Each participant took part in one 3-h session, except for one subject whose data were collected over two sessions.

Instrumentation and evaluation

To evoke the SOL H-reflex, a 1-ms single-pulse monopolar electrical stimulation (constant-current stimulation *Digitimer DS7*, Digitimer Ltd., UK) was applied to the tibial nerve of the right popliteal fossa using a half-ball metal cathode of 22 mm in diameter. The optimal stimulation site was determined by adjusting the position of the stimulating electrode in the popliteal fossa until the site with the lowest threshold for the H-reflex was identified. The rectangular anode (8 × 15 cm) was placed on the anterior aspect of the thigh, just above the patella. The electrodes were fixed in place and held under constant pressure using a custom-made strap, made from a semi-rigid frame to maintain the active electrode in position and two straps extending around the knee, above and below the patella. The superior strap also stabilized the anode.

Following standard skin preparation procedures—'abrasive tape (3M Red Dot Trace Prep) was rubbed on the skin over the targeted muscle prior to placing the electrodes'—electromyographic (EMG) activity was recorded from the right SOL (15 participants) and right TA (11 participants), ipsilateral to tibial nerve stimulation using surface electrodes (Ag–AgCl; 1.5 to 2 cm centre-to-centre spacing). TA EMG was not recorded in the first four participants as our focus was on SOL H-reflex modulation. However, after a preliminary analysis, we extended our data collection and analysis to include TA EMG recording for all subsequent participants to have a more complete description of the EMG activity. EMG electrodes were positioned according to the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles project (Hermens et al. 2000). EMG signals were filtered (10–1000 Hz), amplified (× 1000), sampled at 2 KHz and

recorded on a computer using *Signal 4.07* software for online and offline analyses (CED micro 1401 interface, Cambridge Electronic Design Ltd., UK).

Randomized ramp-shaped forward and backward tilts (magnitude 8°, duration to reach the maximal tilt: 400 ms, speed 20°/s) of the base of support were induced by a *SMART EquiTest* (NeuroCom, version 8.1.0)® every 15–20 s. The rotation axis was parallel and aligned to the right and left lateral malleoli. Participants could not anticipate the exact timing or direction of the perturbation and as a result, could not anticipate the appropriate balance strategy. The relatively small amplitude of the perturbation was sufficient to evoke clear EMG responses while avoiding the need to use a safety harness.

Experimental protocol

Participants stood on the platform with their weight equally distributed between both legs, their arms hanging freely at their sides and looking directly in front of them. Markers on the platform indicated the position of the participant's feet with a transversal line crossing the internal and external malleoli. The participant's position was verified throughout the experiment. The experimental protocol consisted of two parts. As a first step, the SOL H-M recruitment curves were obtained in standing position to determine the appropriate stimulation intensity to elicit the H-reflex. Second, perturbations were applied in three different sequences. Each recording sequence included a maximum of four stimulation delays, lasted 20–22 min and included 80 perturbations. The following paragraphs detail these two experimental components.

Part 1—determination of H-reflex intensity during quiet stance

H-reflex and M-wave recruitment curves were recorded in standing position for all participants at the beginning of each experimental session (Fig. 1a). Stimulus intensity was increased by 2 mA increments (1 reflex per intensity) until the maximum H-reflex amplitude was obtained, and in 15 mA increments until the maximum motor response (Mmax) was reached. Control H-reflexes were all on the upper ascending part of the H-reflex recruitment curve and the H-reflex amplitude obtained varied between 20 and 45% of M_{max} for the group. With this approach, the amplitude chosen was optimal for observing clear condition-dependent changes (facilitation or inhibition) in SOL motoneuron excitability (Pierrot-Deseilligny and Mazevet 2000; Crone et al. 1990; Knikou 2008), while maintaining a small M-response. To control for stimulation intensity applied to the tibial nerve, the M-response of the SOL muscle was monitored and kept constant at a targeted amplitude of 5% for all participants. The range of accepted M-responses varied for each subject, such that intrasubject variation in M-wave amplitude was between 1 and 3% (for example, values accepted for a given subject could be spanned over 3–5% or could be between 5 and 7% for another subject). As four delays were tested in each recording session, small adjustments of a few mA to the intensity were necessary before each stimulus for the M-response to remain within the preset window of amplitude at each stimulation delay (Capaday 1997). Indeed, although the perturbations and stimulation delays were randomized by computer, the experimenter was aware of the upcoming stimulation delay about 5–7 s before the application of the stimulation. The experimenter had time to make the necessary adjustments, based on the prior elicited M-wave.

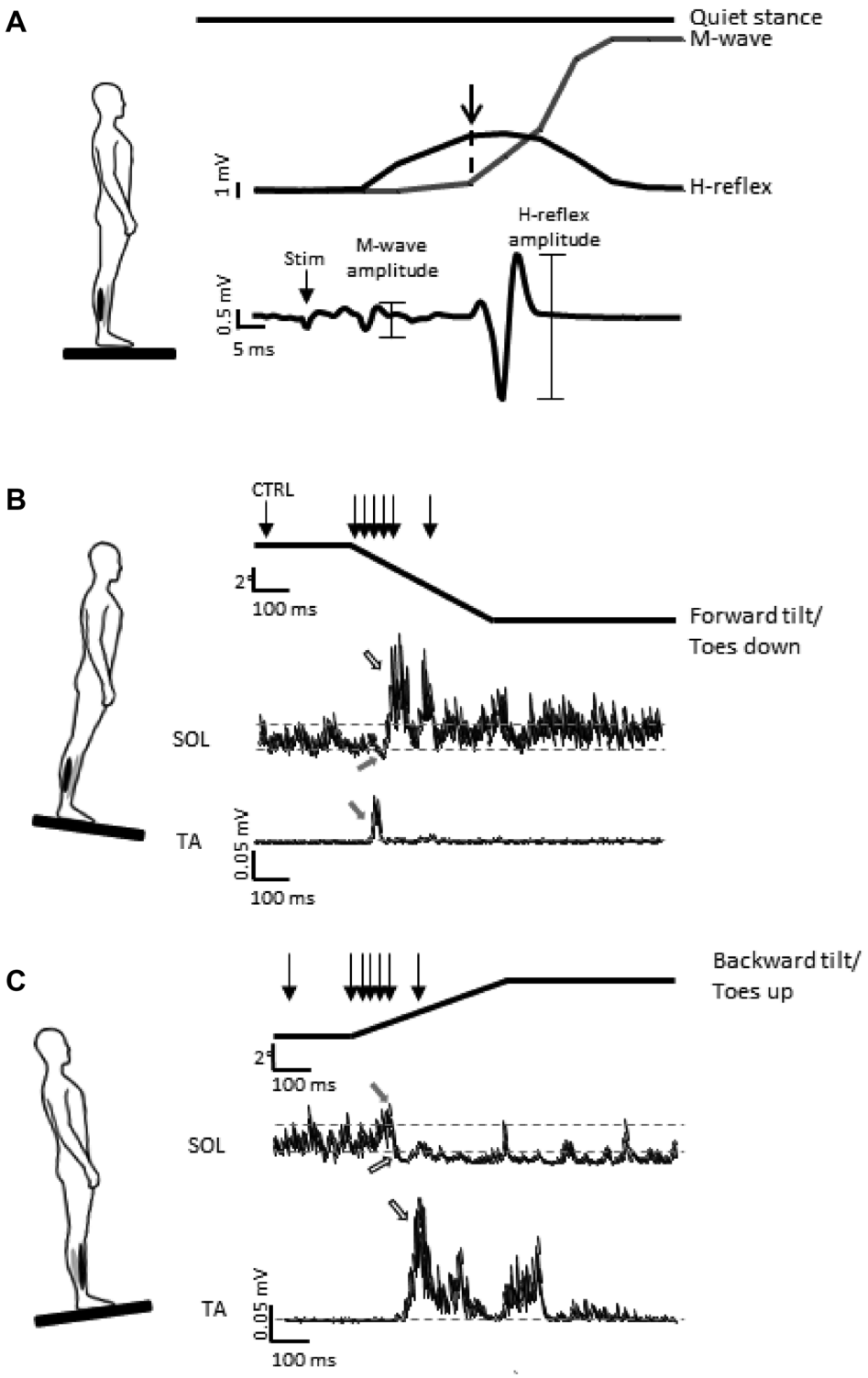
Part 2—H-reflex assessment during platform perturbation

To assess changes in reflex excitability, H-reflexes were evoked before the onset of the tilts (which is referred to as the control condition) and at the following delays after tilt onset: 0, 25, 50, 75, 100 and 200 ms (Fig. 1b, c). These delays were chosen based on our own pilot experiments, as well as existing literature supporting that appropriate EMG responses elicited by similar perturbations occur within the first 200 ms in healthy adults (Diener et al. 1984; Bove et al. 2003; Mummel et al. 1998; Thigpen et al. 2009; Nardone et al. 1995; Schieppati and Nardone 1995). Instructions given to the subjects were to 'maintain the position'. Ten stimuli were applied randomly for each delay and each direction (forward or backward tilt). To avoid fatigue due to prolonged standing, rest periods were taken every 10–20 tilts or earlier if requested by the participant. To assess ongoing EMG level at each of the delays tested, six to ten control trials in each tilt direction were randomly performed without stimulation during the perturbation.

Data analysis

Characterization of EMG postural responses to platform tilt in conditions without stimulation

For each condition (forward tilt or backward tilt), SOL and TA EMG activity recorded in control trials (without stimulation) were rectified and averaged (see Fig. 1b, c). Baseline background muscle activity was quantified by averaging the mean EMG level over a 200 ms period before tilt onset. The onset of increased EMG activity after tilt onset was identified when activity surpassed background EMG activity by two standard deviations (SD) for a minimum of 30 ms (Bove et al. 2003; Thigpen et al. 2009; Barlaam et al. 2016). The same procedure was applied to determine the onset of EMG decrease; however, in this condition EMG activity had to



decrease below background activity by 1SD for a duration of 30 ms. A value of 1SD was chosen because the level of 2SD below the mean was very often below 0. As the EMG data is rectified, it was not possible for the EMG to reach that level. Once the EMG increase or decrease was deemed to be significant, the onset of the EMG burst was followed back to the mean baseline value, and the latency of this point was recorded as the onset of the muscle burst (Thigpen et al. 2009). The end of the EMG burst was determined when the EMG burst crossed back the mean baseline value and stayed below the mean EMG level for 30 ms. The latency obtained for each participant was averaged to determine the mean

◂**Fig. 1** Experimental set-up. **a** H-reflex and M-wave recruitment curves were recorded in the right leg for each participant at the beginning of each session while standing on an immobilized platform. The upper trace corresponds to the movement of the platform. Here, the participant is tested during quiet stance so the platform is stable. The second trace corresponds to the recruitment curve taken in one participant. Along the *X* axis, the intensity of the stimulation is gradually increased (see methods for details) and the *Y* axis represents the peak-to-peak amplitude of either the M-response (gray trace) or the H-reflex (black trace). The arrow and dotted line represent the time point where the M-wave and H-reflex displayed in the third trace was chosen. In the single sweep shown in the third trace, the amplitude of the control H-reflex and targeted M-wave were determined. **b** The solid line on the upper trace illustrates the forward tilt (toes down) of the platform. H-reflexes (black arrows) were evoked before the tilts (control) and at 0, 25, 50, 75, 100 and 200 ms after onset of forward tilt. The second and third traces indicate, respectively, right SOL and right TA EMG activity during perturbations, while no stimulation was applied. EMG of SOL and TA represent an average of ten trials. The lower dash line represents 1SD of the baseline EMG, and the upper dash line, 2SD. These lines were used to determine whether the EMG bursts were significantly different than the baseline EMG. Forward tilt induced a decrease in SOL EMG amplitude (gray arrow) followed by an increase in SOL EMG (white arrow) which corresponds to the appropriate postural response to prevent a fall in this context. TA EMG activity shows a short latency, short duration EMG burst. **c** During backward tilts (upper trace, toes up), H-reflexes were also evoked before and at 0, 25, 50, 75, 100 and 200 ms after backward tilts. The tilts induced a small increase in the SOL EMG (gray arrow) followed by an important decrease in EMG amplitude. This decrease in SOL EMG was accompanied by an increase in TA EMG amplitude (white arrows). Those two latter responses correspond to the appropriate postural responses in this context

latency of EMG responses in SOL and TA for the group. The area of the EMG response was compared to the area of the baseline background EMG obtained over the same duration before tilt onset and expressed as a percentage of the baseline background EMG. For responses exceeding 200 ms, the area obtained during the first 200 ms was compared to the area of background EMG calculated over 200 ms. The ratio obtained was multiplied by 100.

Characterization of SOL H-reflex modulation during platform tilt

Peak-to-peak amplitude of the H-reflex was measured within a 30-ms window (between 25 and 55 ms) after the onset of tibial nerve stimulation for each trial. For each H-reflex, we confirmed that the amplitude of the corresponding M-wave was in the appropriate bracket, defined at the beginning of the experiment (see description in Part 1). In eight subjects, one to three trials had to be discarded at one ($n = 4$ subjects), two ($n = 3$ subjects) or three delays ($n = 1$ subject) as the M-wave was either too high or too low. A maximum of three trials were discarded for a given delay and we ensured that at least 7 H-reflexes were kept and averaged for each delay. Then, the amplitude of the H-reflexes was averaged for each delay. Corresponding ongoing EMG was measured in trials without stimulation as the mean amplitude of EMG level during the same time window as the H-reflexes measurements were taken (25–55 ms after tibial nerve stimulation). All measurements were normalized to the maximum M-wave (M_{max}) obtained during standing. To analyze the pattern of H-reflex modulation for the entire group (pooled data), H-reflexes evoked at each delay were divided by the H-reflex obtained before tilt in the 'control' position and multiplied by 100.

Statistics

Descriptive statistics of H-reflex and M-wave amplitude were calculated for all participants for each condition: (1) standing before platform tilt (control); (2) backward tilt at different delays; and (3) forward tilt at the same delays. Averages are expressed as mean ± SEM.

Repeated-measures ANOVAs were conducted across the seven delays (control, 0, 25, 50, 75, 100 and 200 ms), for forward and backward tilts separately, for H-reflex, M-wave and mean ongoing EMG amplitudes. Due to technical problems, there were two missing data (in one subject, data for backward tilt at 50 ms could not be analyzed and in another subject, data for forward tilt at 25 ms could not be analyzed either). They were imputed using the expectation–maximization procedure to retain those two participants in the analyses. We then ran Bonferroni-corrected post hoc analyses to determine if there were significant differences between the control condition and the multiple delays.

To gain more insights into changes in H-reflex excitability leading to onset of EMG responses in both forward and backward tilts, comparison of the onset of the modulation of H-reflex amplitude were performed between forward and backward tilt. Then, comparison between the onset of H-reflex modulation and onset of SOL EMG responses were performed. As the data did not follow a normal distribution (Shapiro–Wilk test), a related samples Wilcoxon signed rank test was performed for all comparisons.

We also assessed the correlations between the amplitude of H-reflex and the level of background EMG in both forward and backward tilts, using Pearson's correlation coefficient.

To determine whether height could explain the variability of responses in H-reflexes modulation and in the onset of EMG responses, correlations were performed (Pearson's if normally distributed, Spearman's if not).

All effects were considered significant for $p < 0.05$. All statistical analyses were performed using IBM SPSS Statistics V20.

Results

EMG responses during platform tilt

Perturbations induced short-, medium- and long-latency EMG responses in the subjects tested as described in the methods. Figure 1b, c illustrate the EMG activity in right SOL and TA during forward and backward tilts for a single representative participant (average of 10 trials). Horizontal dotted lines show the level of significance for the EMG activity. During forward tilts (platform: toes down; Fig. 1b), a decrease in SOL EMG activity was observed at 97 ms after the tilt onset (MLR; 47% decrease with respect to background EMG, gray arrow) and was followed by an increase in SOL EMG amplitude at 114 ms (LLR; 317% increase, white arrow). An increase in the amplitude of TA EMG was also observed at 82 ms after tilt onset (MLR; 746% increase; gray arrow) and lasted 42 ms.

Group data analysis corroborates what was seen in this participant. During forward tilt, 11/15 subjects showed a brief decrease in SOL EMG (51 ± 4% of background EMG) at 72 ± 6 ms and lasting 46 ± 6 ms. This was followed by an EMG increase (264% ± 27%), observed in all 15 subjects starting at 127 ± 5 ms and lasting 179 ± 11 ms. This increase corresponded to the LLR and constituted the appropriate muscular activation of SOL in this condition to prevent a fall. EMG activity in TA was assessed on 11 subjects and in all subjects tested, an increased EMG (MLR; 755 ± 138%) was observed in the TA starting at 91 ± 5 ms and lasting 101 ± 22 ms. In six participants, the responses seen in the TA were of a very short duration ranging from 32 to 55 ms (42 ± 3 ms) with a mean onset at 89 ± 3 ms. However, in five participants, the responses observed in the TA were of longer duration, being above 110 ms. In these five subjects, co-contraction between TA and SOL was observed.

Data from the same representative subject during backward tilts (platform: toes up, Fig. 1c) show a small increase in SOL EMG amplitude at 42 ms after tilt onset (SLR; 184% increase with respect to background EMG; gray arrow) and lasted 49 ms. This increase in SOL EMG amplitude was followed by a prolonged decrease in SOL EMG activity (43% of background EMG), at 91 ms that lasted longer than the 200 ms analyzed (white arrow). In this subject, this EMG response corresponded to the appropriate muscular decrease in this condition to prevent a fall, even though it occurred before 100 ms. In parallel with the decrease in SOL EMG, an increase in TA EMG activity occurred at 112 ms (LLR; 1400% of background EMG).

This pattern was also seen for the group. A small increase in SOL EMG amplitude was observed in 14/15 subjects at 47 ± 4 ms (SLR; 257% ± 29%) and lasted 82 ± 15 ms. In 12/15 subjects, a decrease in EMG amplitude followed (48% ± 3%) starting at 144 ± 16 ms and lasted 138 ± 22 ms. This latter response corresponded to the LLR. Three participants did not show a significant decrease in SOL EMG. In TA EMG, an increase (LLR; 2870%) is observed in the 11 participants tested starting at 113 ± 5 ms and lasted 180 ± 14 ms. Despite inter-subject variability, the EMG activity underlying the LLR and appropriate postural responses in the SOL (increased EMG in forward tilt and decreased EMG in backward tit) is consistent across the group. Hence, future sections will mainly describe the changes in H-reflex amplitude relative to the LLR.

H-reflex modulation during platform tilt

Figure 2 shows the H-reflex and M-wave evoked by tibial nerve stimulation during tilts in a representative participant. The trace shown in Fig. 2a corresponds to the responses observed when tibial nerve stimulation is applied prior to tilt onset (control condition; CTRL). An M-response was evoked with an amplitude of 6 ± 2% of M_{max} (gray arrow), followed by an H-reflex with an amplitude of 21 ± 7% of M_{max} (white arrow). H-reflex amplitudes at all post-tilt delays are expressed as a percentage of the H-reflex amplitude observed prior to tilt onset (control H-reflex; defined as 100%). When stimulation was applied at or after the onset of forward tilt (Fig. 2b–g), the M-response could still be evoked consistently and remained at an amplitude of 5–6% Mmax at all time points tested. In this participant, the amplitude of the H-reflex varied slightly between the control condition (100% of control; Fig. 2a), 0 (100%; Fig. 2b), 25 (90%; Fig. 2c), 50 (115%; Fig. 2d) and 75 ms after tilt onset (100%; Fig. 2e). At 100 ms (Fig. 2f) and 200 ms (Fig. 2g), the amplitude of the H-reflex greatly increased to 176% and to 310% of the control H-reflex respectively. When stimulation was applied after onset of backward tilt (lower panel), H-reflex amplitude was first increased at 25 ms (Fig. 2j) and 50 ms (Fig. 2k) (amplitude of 136% of control H-reflex for both delays), but then decreased from 75 ms (Fig. 2l) to reach an amplitude of 8% and 4% of control H-reflex at 100 ms (Fig. 2m) and 200 ms (Fig. 2n), respectively, where a clear H-reflex could not be observed. In all these conditions, the M-wave remained between 5 and 7% of M_{max}.

This pattern was representative of all participants tested and Fig. 3 represents the modulation of amplitude in H-reflex, M-wave and mean background EMG for the group in both forward and backward tilts.

During forward tilts (Fig. 3a), the amplitude of the H-reflex for the group showed no modulation of amplitude at earlier delays but showed an increased amplitude at 100 ms which is maintained at 200 ms. A repeated-measures

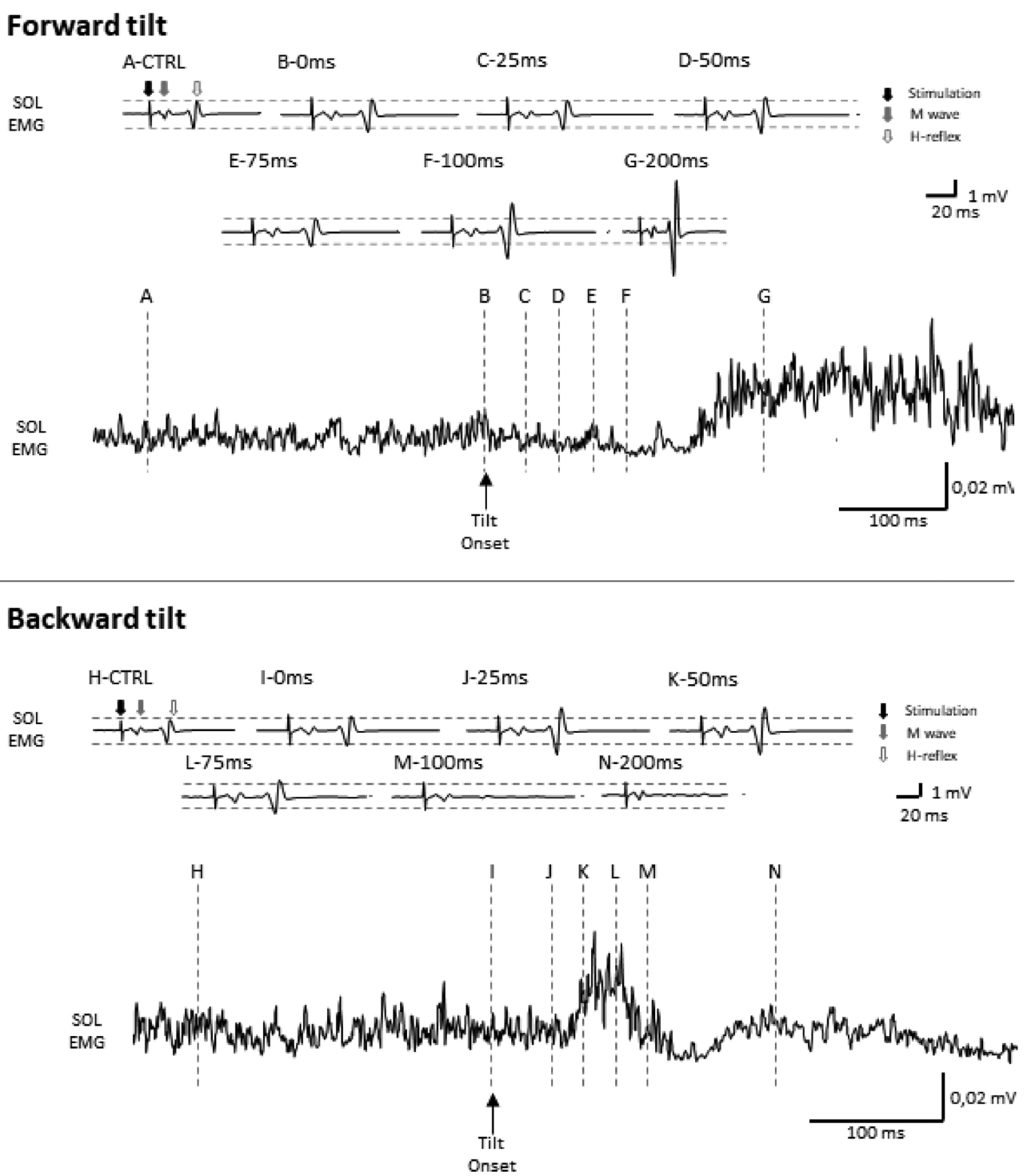


Fig. 2 Modulation of H-reflex and M-wave amplitude in a single participant. Upper panel. Forward tilt. **a**–**g** These seven graphics illustrate the response to the tibial nerve stimulation applied at the different delays tested (a single trial is represented). At all those delays, a small M-wave and an H-reflex were elicited. For each graphic, the corresponding identifying letter is plotted on the EMG below to indicate the timing of the stimulation with respect to the onset of the perturbation. EMG of SOL represents an average of ten trials. While amplitude of the M-wave remains stable, the amplitude of the H-reflex varies. The dotted lines indicate the peak-to-peak amplitude of the H-reflex in the control condition. Although small variations of the H-reflex amplitude are observed at delays 0–75 ms, an important increase in amplitude is seen at 100 and 200 ms. Lower panel. Backward tilt. **h**–**n** Display is similar to what is described for the upper panel (**a**–**g**). In the backward tilt condition, an increase in H-reflex amplitude is observed at 25 and 50 ms followed by an important decrease in H-reflex amplitude, which reaches 8 and 4% M_{max} at 100 and 200 ms, respectively

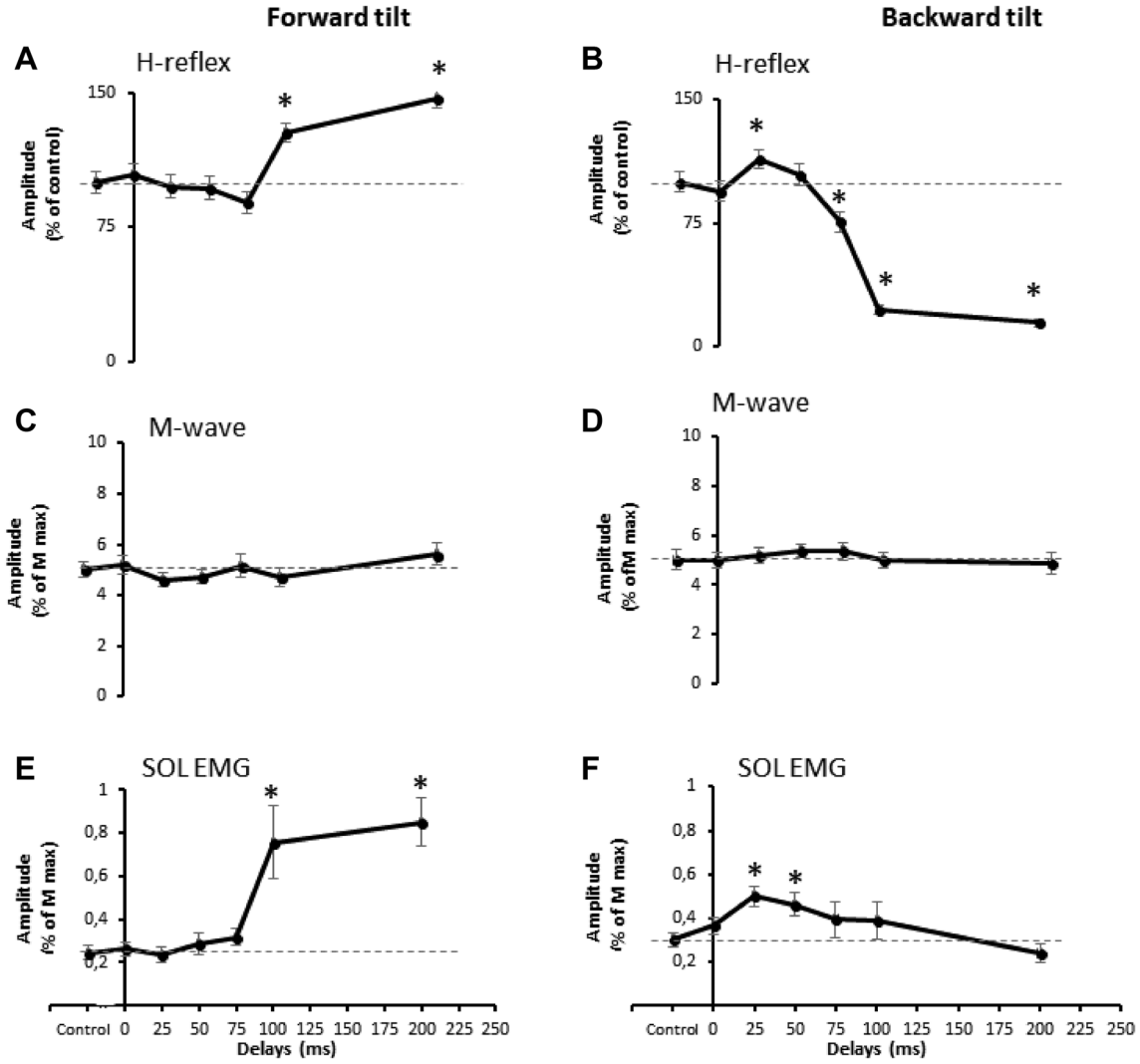


Fig. 3 Modulation of H-reflex and M-wave amplitude. Group data. Group data during forward (left column) and backward tilt (right column). Amplitudes are given as mean ± standard error of the mean. Asterisk statistically significant; dotted line: value of the control. **a, b** H-reflex amplitudes are expressed as a % of the control H-reflex recorded before the tilt of the supporting surface. **a** During forward tilt, the H-reflex is significantly increased at 100 and 200 ms. **b** During backward tilt, the H-reflex amplitude is significantly increased at 25 ms and significantly decreased at 75, 100 and 200 ms after tilt onset. **c, d** M-response amplitude are expressed as a % of the maximal M-wave. In both forward and backward tilts, no statistically significant changes are detected in the M-response. **e, f** Mean amplitude of ongoing EMG in SOL are expressed as a % of the maximal M-wave. **e** During forward tilt, the mean amplitude of ongoing EMG is significantly increased at 100 and 200 ms. **f** During backward tilt, the mean level of ongoing EMG is significantly increased at 25 and 50 ms, but no change is observed at delays 75, 100 and 200 ms

ANOVA confirmed this finding, as there was a significant difference between the responses across delays ($p < 0.001$, partial $\eta^2 = 0.560$). Post hoc analyses indicated that, before multiple testing correction, there was a significant difference between control ($44 \pm 23\%$) and 100 ms ($56 \pm 21\%$; mean diff. = 12.206, $p = 0.002$, Cohen's $d = 0.973$), and control and 200 ms ($65 \pm 20\%$; mean diff. = 20.649, $p < 0.001$, Cohen's $d = 1.764$). Both remained significant after correction (corrected $p = 0.012$ and < 0.001, respectively).

During backward tilt (Fig. 3b), an increase in the H-reflex amplitude for the group is observed at delay 25 ms, followed by a marked decrease at 75, 100 and 200 ms. A repeated-measures ANOVA confirmed this, as there was a significant difference between the responses across delays ($p < 0.001$, partial $\eta^2 = 0.788$). Post hoc analyses indicated that, before multiple testing correction, there was a significant difference between control ($48 \pm 24\%$) and 25 ms ($55 \pm 22\%$; mean diff. = 6.586, $p < 0.001$, Cohen's $d = 1.275$), control and 75 ms ($37 \pm 25\%$; mean diff. = 11.741, $p < 0.001$, Cohen's $d = 1.243$), control and 100 ms ($11 \pm 11\%$; mean diff. = 37.656, $p < 0.001$, Cohen's $d = 1.872$), and control and 200 ms ($7 \pm 8\%$; mean diff. = 41.404, $p < 0.001$, Cohen's

$d = 2.181$). All differences remained significant after correction (corrected $p = 0.001$, 0.002, < 0.001 and < 0.001, respectively).

To confirm that H-reflex modulation was not due to changes in the stimulation intensity, the mean M-wave amplitude for each stimulation delay and perturbation direction was also assessed using repeated-measures ANOVA (Fig. 3c, d). There was no significant difference between the amplitude of M-responses across delays during backward tilt (values range from 4.9 ± 1.7 to $5.4 \pm 1\%$ M_{max}; $p = 0.474$, partial $\eta^2 = 0.063$) or forward tilt (values range from $4.7 \pm 1\%$ to $5.6 \pm 1.7\%$; $p = 0.079$, partial $\eta^2 = 0.144$).

To determine if modulations in SOL H-reflex amplitude could be explained by changes in ongoing EMG activity, correlation between H-reflex amplitude and background EMG levels was assessed in all participants. A weak but positive association between EMG and H-reflex across all delays was detected ($r = 0.358$, $p < 0.001$) in forward tilt, such that an increase in EMG was associated with increased H-reflex levels. A weak association was also observed during backward tilt ($r = 0.251$, $p = 0.01$).

Due to the weak strength of the correlations across all delays, and to further understand the relationship between background EMG and the SOL H-reflex during forward and backward perturbation, the mean level of SOL EMG amplitude observed in unstimulated trials at each of the tested delays was assessed using RM ANOVA (Fig. 3e, f). In the forward tilt condition, there was a significant difference between mean SOL EMG amplitude across delays tested ($p < 0.001$, partial $\eta^2 = 0.526$). Before multiple testing correction, there was a significant difference between control ($0.25 \pm 0.12\%$) and 100 ms ($0.76 \pm 0.65\%$; mean diff. = 0.512, $p = 0.003$, Cohen's $d = 0.920$), and control and 200 ms ($0.85 \pm 0.42\%$; mean diff. = 0.607, $p < 0.001$, Cohen's $d = 1.751$). Both remained significant after correction (corrected $p = 0.019$ and < 0.001, respectively).

In the backward tilt condition, there was a significant difference between the mean ongoing EMG amplitude across delays ($p = 0.035$, partial $\eta^2 = 0.194$). Before multiple testing correction, there was a significant difference between control ($0.3 \pm 0.1\%$) and 0 ms ($0.44 \pm 0.2\%$; mean diff. = 0.063, $p = 0.017$, Cohen's $d = 0.700$), control and 25 ms ($0.5 \pm 0.2\%$; mean diff. = 0.200, $p < 0.001$, Cohen's $d = 1.98$), and control and 50 ms ($0.5 \pm 0.2\%$; mean diff. = 0.160, $p = 0.001$, Cohen's $p = 1.033$). The difference between control and 0 ms was no longer significant after correction (corrected $p = 0.101$), but control/25 ms and control/50 ms differences remained significant after correction (corrected $p < 0.001$ and $= 0.008$, respectively). No significant difference was found in the mean level of ongoing EMG at 75, 100 or 200 ms.

Hence, an increase in mean amplitude of ongoing EMG is observed when the amplitude of the H-reflex is increased at 100 and 200 ms during forward tilt. However, although the increase in EMG activity observed at 25 and 50 ms is paralleled by an increase in H-reflex amplitude at 25 ms, no decrease in the mean amplitude of ongoing EMG of SOL was observed at 75, 100 and 200 ms after backward tilt when the H-reflex was significantly decreased.

H-reflex modulation precedes the onset of long latency EMG responses (LLR) in backward but not forward tilt

During backward tilt, group data shows a significant decrease in H-reflex amplitude at 75 ms after tilt onset; during forward tilt, a significant increase in H-reflex amplitude was observed at 100 ms. These results outline the timing of H-reflex modulation associated with LLRs following a perturbation for the group; however, important inter-subject variability is observed. To characterize this variability, the data of each subject was analyzed to identify the earliest delay at which a large variation in H-reflex amplitude (compared to control) was observed. Figure 4a shows that the group data showed an important decrease in H-reflex amplitude during backward tilts at 77 ± 5 ms (bold black diamonds): the decrease in H-reflex amplitude was observed at 50 ms for three subjects, 75 ms for eight subjects and at 100 ms for four subjects (individual data are shown as gray squares). For the forward tilt, whereas the increase in H-reflex amplitude was observed at 123 ± 13 ms for the group, single-subject analysis reveals that this increase occurred at 75 ms for two subjects, 100 ms for nine subjects and 200 ms for four subjects. Importantly, the delay of 200 ms does not mean that the change in H-reflex occurs at 200 ms. Rather, since no delays were assessed between 100 and 200 ms, the delay of 200 ms only reflects that changes in H-reflex amplitude occurred after 100 ms but before 200 ms. Thus, the onset of the H-reflex amplitude decrease during backward tilt precedes the onset of H-reflex amplitude increase during forward tilt (mean onset during backward tilt = 77 ± 5 ms vs forward tilt = 123 ± 13 ms, Wilcoxon signed rank test, $p = 0.0035$). An increase in H-reflex amplitude was also observed at 25 ms during backward tilt, but it did not correspond to the LLR observed in the EMG and was not studied in this section.

The variation seen in the onset of H-reflex modulation between subjects and between tasks may be reflected in the onset of EMG responses. To examine changes in H-reflex excitability preceding the onset of EMG postural activity, the interval of time between the onset of H-reflex modulation and the onset of the EMG responses that are appropriate to prevent a fall was analyzed (LLR; indicated by the white arrows in Figs. 1b, c, 4b).

During forward tilt, the increase in H-reflex amplitude preceded the increase in SOL EMG by 4 ± 11 ms

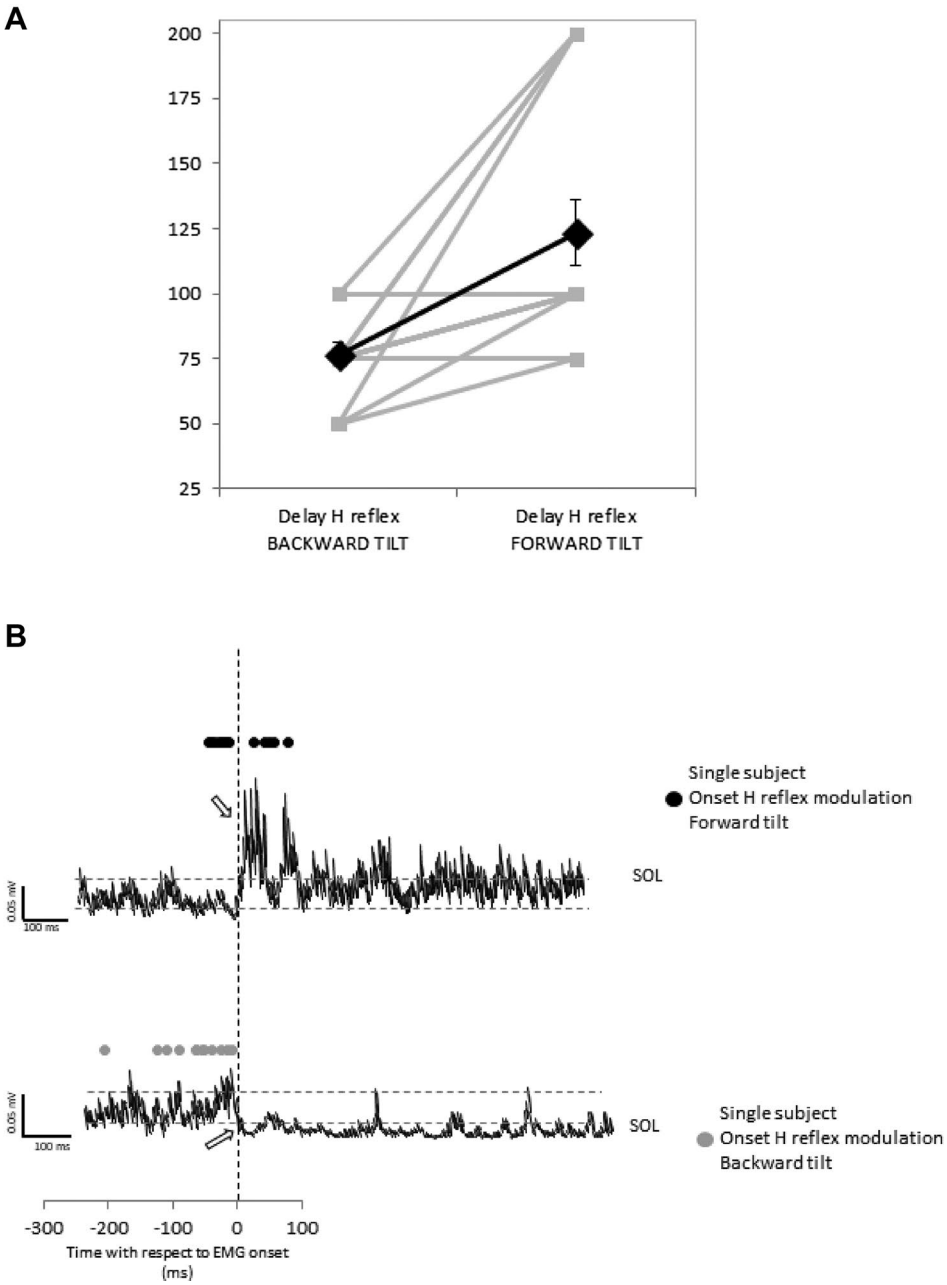


Fig. 4 **a** Comparison of delays when the H-reflex amplitude significantly decreases during backward tilt (Delay H-reflex BACKWARD) and the delay when the H-reflex amplitude significantly increases during forward tilt (Delay H-reflex FORWARD). The gray squares represent single-subject data and the black diamonds represent the mean for the group. **b** Comparison of the time interval between the onset of the increase (forward tilt) or decrease (backward tilt) in H-reflex amplitude (data plotted in **a**) and the onset of the late EMG activity, corresponding to the appropriate postural reaction to recover balance (white arrows and vertical dotted line). Time 0 corresponds to the onset of the EMG response during either forward (upper trace) or backward tilt (lower trace). Each black dot reflects onset of significant increase in H-reflex amplitude for one subject during forward tilt. Each gray dot reflects onset of significant decrease in H-reflex amplitude for one subject with respect to decreased EMG response. The EMG of SOL represents an average of ten frames

on average for the group, which was not significant (increase H-reflex = 123 ± 12 ms vs increase SOL EMG = 127 ± 5 ms; Wilcoxon signed rank test, $p = 0{,}742$). Figure 4b shows that the delay between onset of H-reflex modulation and onset of SOL EMG increase was quite variable between participants (black circles over the upper EMG trace in Fig. 4b), ranging from 46 ms before the onset of SOL EMG to 54 ms after the onset of SOL EMG. The H-reflex modulation occurred after the increase in SOL EMG in five participants. During backward tilt, the decrease in H-reflex amplitude preceded the decrease in SOL EMG by 73 ± 16 ms, which was significant (decrease in H-reflex = 75 ± 5 ms vs decrease in SOL EMG = 144 ± 16 ms, Wilcoxon signed rank test, $p = 0.00087$). Figure 4b also shows that during backward tilt, the decrease in H-reflex modulation preceded the decrease in SOL EMG in all participants (range of − 9 to − 208 ms; gray circles over the lower EMG trace). This difference between forward and backward tilt may show a difference in the neuronal preparation and modulation between both directions at the central nervous system level.

Inter-subject variability is not due to height difference between the participants

To assess the impact of participant height on the variability reported above, correlations were drawn between the height of the participants, the latency of onset of the H-reflex amplitude modulation in the forward and backward tilt and the latency of EMG responses in SOL and TA. No significant correlation could be drawn between onset of H-reflex modulation and height ($p < 0.05$ for both the forward and backward conditions). No significant correlation could also be drawn between the onset of short latency and medium latency EMG responses in SOL or TA and height. For the long latency responses, only the decrease of SOL EMG in the backward tilt condition was significantly associated with height ($r = -0.587$, $p = 0.045$).

Discussion

Results showed that the SOL H-reflex amplitude is modulated during postural reactions in response to forward and backward tilts of the base of support. During backward tilt, early increases in the H-reflex amplitude at 25 and 50 ms preceded the short latency response in SOL EMG (47 ± 4 ms). Thereafter, the amplitude of the H-reflex decreased at 75, 100 and 200 ms after tilt onset. This late change in H-reflex amplitude occurred on average 73 ± 6 ms before the decrease in SOL EMG (LLR), which is the appropriate muscular response to prevent a fall. Hence, the H-reflex modulation during backward tilt could not be mainly explained by changes in ongoing EMG activity.

During forward tilts, amplitude of H-reflex was significantly increased at 100 and 200 ms. This change paralleled the EMG increase in the SOL muscle (LLR; 127 ± 5 ms for the group). Hence, the H-reflex modulation observed during forward tilt could be mainly explained by variations in the background EMG as was shown during other tasks (Pierrot-Desseilligny and Mazevet 2000; Knikou 2008). Taken together, there results suggest direction-dependant difference in central control strategies of postural response to platform tilts.

Time course of H-reflex changes relative to EMG postural activity in SOL

Latencies obtained for the SLR, MLR and LLR in this study for SOL EMG were comparable to those reported in other studies assessing postural reactions in healthy subjects using similar perturbation parameters in terms of speed and amplitude (Diener et al. 1984; Thigpen et al. 2009; Nardone et al. 1995; Schieppati and Nardone 1995; Marigold and Eng 2006). The onset of the SOL H-reflex modulation observed here preceded modulation of EMG responses. After backward tilt, a clear tendency showing changes in H-reflex was observed at 25 and 50 ms. These changes were of short duration and in the opposite direction to the longer latency responses that corresponded to the appropriate postural reactions. Namely, a backward tilt first induced a short latency, short duration increase in H-reflex amplitude followed by a longer latency, long-lasting decrease in H-reflex amplitude and subsequently of the EMG. This short latency increase in H-reflex amplitude could in part reflect the activation of the monosynaptic stretch reflex, where discharge in Ia afferents from the SOL could increase and induce activation of the SOL motoneurons. An increase in EMG amplitude was also seen a few milliseconds later in the SOL muscle (47 ± 4 ms; see also Diener et al. 1984).

In forward tilt, EMG analysis showed that a small decrease in SOL EMG occurred at a short latency delay in 11 out of 15 participants. The decreased EMG observed in these 11 participants may be explained by the rapid shortening of the SOL muscle, which would induce a transient silence in muscle spindle discharge and lead to a decreased discharge in the Ia afferent. However, this was not reflected in the H-reflex amplitude or in the pooled EMG data of all participants taken at specific delays (Fig. 3a, e). It will therefore not be discussed further. For the late responses, H-reflex amplitude increased significantly at 100 ms, while SOL EMG increased significantly at an average latency of 127 ± 5 ms.

The SLR, MLR and LLR observed in the SOL have been widely described. The SLR is considered to be mediated by Ia-afferent fibers (Matthews 1991), the MLR is attributed to oligosynaptic excitation of spinal motoneurons via group II afferents (Schieppati and Nardone 1997; Grey et al. 2001) and possibly by group Ib afferents(Dietz 1998). The LLR is generally regarded as a transcortical response (Jacobs and Horak 2007) that mediates the changes in SOL EMG activity and contributes to generating appropriate postural responses to prevent falls.

Despite occurring at slightly later latencies that the SOL LLR described in literature (also referred to as M3, see Sinkjaer et al. 1999), the decrease in SOL EMG activity observed at 144 ± 16 ms during backward tilts, likely corresponds to a LLR due to differing perturbation parameters in this study. Indeed, the speed of the perturbation was higher in Sinkjaer et al. (8°stretch with a velocity of 280°/s) compared to the speed used in the present study (8°stretch with a velocity of 20°/s). The influence of perturbation speed on the latency of muscle responses has been shown before (Diener et al. 1984).

Although EMG onset occured relatively late, the decrease in H-reflex amplitude was already observed at 75–100 ms. In that respect, the data in Figs. 3 and 4 show that whereas the decrease in SOL H-reflex amplitude during backward tilt

is not paralleled by a decrease in SOL EMG, the increase in H-reflex amplitude observed in forward tilt is accompanied by an increase in SOL EMG. Thus, although H-reflex modulation was observed during both forward and backward tilts, the decrease in H-reflex amplitude during backward tilt occurred earlier than the increase in H-reflex amplitude during forward tilts.

Of note, the relation between H-reflex and EMG was not to be readily expected. Indeed, it is possible to have an increase or a decrease in H-reflex amplitude without changes in EMG level notably seen when presynaptic mechanisms are involved (Hultborn et al. 1987a, b). However, changes in the H-reflex amplitude are often observed in synchrony with changes in the level of EMG during gait, during movement or at the onset of movement.

Notably during gait, the modulation of H-reflex amplitude closely resembles the modulation of SOL EMG (Capaday and Stein 1986). These changes are task-specific and reflect a change in the neuronal excitability as the movement is being performed. Moreover, in the postural tasks studied, timing of EMG and H-reflex changes can be specifically assessed because the onset of perturbation is known and constitutes a clear event triggering both responses. As such, we had hypothesized that the perturbation would trigger a response in both H-reflex and EMG, but that since balance reactions are centrally mediated at the level of the nervous system, we expected to observe a change in the H-reflex amplitude before seeing a change in the EMG level.

EMG postural activity in TA

Concerning the TA EMG, a LLR was observed during backward tilt in all participants and, during forward tilt, a MLR was observed in the TA. However, in five participants, the MLR observed in forward tilt lasted longer leading to co-contraction between the TA and SOL. It remains unclear why the difference exists between these participants and the rest of the cohort. Further experiments could be done to try and determine the impact of these differences on the pattern of the postural reaction, notably by assessing the displacement of the center of pressure.

Differences observed during forward and backward tilts

The difference observed is inherent to the tasks performed as backward tilt induced a decrease in EMG, whereas forward tilt induced an increase EMG. One possible explanation for the above-mentioned difference might be the possibility for the participant to see where they might fall during forward tilt, whereas it is not possible to do so during backward tilt. This might contribute to a perceived 'increased threat' during backward tilt compared to forward tilt despite identical perturbation parameters in terms of speed and amplitude. However, the importance of vision in the modulation of H-reflex amplitude described here during balance reactions is not likely as the visual scene is changing in both directions. Furthermore, Nakata and Yabe (2001) compared automatic postural responses to perturbations in blind and sighted subjects. They observed that automatic postural responses could occur in both groups with similar EMG amplitudes, but that blind individuals, who are highly dependent on somatosensory input, had faster reaction times than sighted subjects to platform perturbation during toe-up rotation. Similarly, sighted subjects showed similar reaction times whether their eyes were closed or open. Hence, these results suggest that simply the presence or absence of vision might not explain the discrepancy observed between the two conditions in this study.

An alternative explanation for this discrepancy might reside in the increased perception of danger during backward tilt, linked to the notion of limits of stability (Holbein-Jenny et al. 2007; Pickerill and Harter 2011). The limits of stability during standing would not be equally distributed around the center of pressure due to the shape of the base of support, which extends further in front of the body (forefoot) than it does behind the body (heel). As a result, the center of pressure reaches the limits of stability more quickly in backward tilts, which may lead to perturbations being perceived as more threatening. The perceived perturbation threat in backward tilt compared to forward tilt may therefore account for differences in the onset of H-reflex modulation. Previous studies have shown that perception of a postural threat has an influence on postural responses (Horslen et al. 2018; Cleworth et al. 2018; Tokuno et al. 2018). However, this hypothesis is speculative and would need to be tested specifically.

Inter-subject variability could not be explained by subject's height

Although the pattern of modulation of the SOL H-reflex amplitude and of the increase/decrease in SOL EMG amplitude was similar in all subjects tested, the exact latency at which an increase or decrease occurred was variable between subjects. The sample studied consisted of healthy young men and women and thus inter-individual differences are unlikely to be caused by neuronal or orthopedic deficits. We thus investigated whether height could explain the difference in latencies as the distance the neural influx has to travel to reach the spinal cord and back in a taller subject might be relevant to understand this discrepancy. Furthermore, a previous study had shown that the subject's height might have an influence in postural EMG reactions (Berger et al. 1992; the range of height of participants was from 113 to 193 cm). This conclusion was partially supported in the

current study as a significant correlation was found between the height of the subject and the onset of decrease in SOL EMG amplitude during backward tilt. However, we did not find any correlations between the height of the subject and onset of H-reflex modulation. One explanation for this difference might be the range or the subject's height in the current study (160–185 cm) which was smaller than in the study by Berger et al. (1992). Another explanation is that H-reflex amplitude was assessed at delays 0, 25, 50, 75. 100 and 200 ms. The relatively large gaps between the delays result in insufficient precision of the timing of H-reflex modulation. This could mask possible correlations between H-reflex amplitude and height.

Proposed mechanisms of task-dependent modulation of the H-reflex

SOL H-reflex modulation has previously been demonstrated during functional tasks such as locomotion and running (Capaday and Stein 1986, 1987; Knikou et al. 2011). SOL H-reflex amplitude was reported to be facilitated during the stance phase when SOL EMG activity increased and decreased during the swing phase when EMG activity decreased. This phase-dependent modulation in H-reflex amplitude during gait is thought to be induced by spinal interneuronal networks, namely the central pattern generator (CPG), which receives input from sensory afferents and supraspinal pathways. Rhythmic spinal neuronal networks might not be in play here as postural reactions are not a cyclic behavior and are activated following specific destabilizing cues.

H-reflex modulation has also been studied in relation to postural stability. When postural conditions became more challenging (e.g., during standing on an unstable surface), a decrease in SOL H-reflex amplitude was observed (Earles et al. 2000). Also, amplitude of SOL H-reflex (as well as H-reflex in gastrocnemius medialis) was larger when a standing subject swayed forward compared to a backward sway (Tokuno et al. 2008). It was suggested that subcortical mechanisms might be in play, including presynaptic inhibitory mechanisms, which could be responsible for H-reflex modulation observed during postural tasks. These mechanisms may also be involved in the modulation observed in the present study: during backward tilt, no change in EMG was observed during variations in H-reflex amplitude, which suggests that changes take place presynaptically rather than at the postsynaptic level. However, the involvement of Ia presynaptic inhibition as an explanation for H-reflex modulation between forward and backward centre of pressure oscillations has been questioned recently (Tokuno et al. 2009; Johannsson et al. 2017).

Another possible mechanism stems from the finding that changes in H-reflex amplitude initially resembled stretch reflex EMG responses but were then inverted (at 75 and 100 ms in backward and forward tilt respectively) to enable appropriate balance responses. This suggests the involvement of central mechanisms at that point in time, which may be responsible for changes in reflex gains and thresholds in either tilt direction. As is the case with gait, supraspinal centers, through their descending projections, including the corticospinal (Taube et al. 2006; Tokuno et al. 2009; Fujio et al. 2018), vestibulospinal (Hlavacka et al. 1999; Horak and Hlavacka 2002; Horak et al. 2002) and reticulospinal (Stapley and Drew 2009; Allum et al. 2011; Campbell et al. 2013) tracts, also contribute to postural reactions, both reactive (external perturbations) and proactive (e.g., anticipation of a perturbation; Petersen et al. 2009). These convey the wide source of afferent information that contribute to the control of postural responses, such as somatosensory afferents (Creath et al. 2008), vestibular afferents as well as vision (Bronstein 2016).

The influence of the corticospinal and cortical mechanisms might be preponderant in the results shown here: Taube et al. showed that the cortex was directly involved in the generation of postural reaction through transcortical loop (Taube et al. 2006). Notably they showed that, postural compensatory responses could be cortically mediated approximately at 86 ms after perturbation. Our results show that between 75 and 100 ms, H-reflex excitability changes to enable appropriate postural reaction. This change could be directly induced by the corticospinal system. Furthermore, intracortical mechanisms were also shown to be involved in balance control during upright standing, as decreased intracortical inhibition was observed when body sway was necessary in healthy individuals to maintain balance (Papegaaij et al. 2016).

Clinical implications of these findings

After a lesion to the central nervous system, many patients show decreased balance control and are at higher risk of falling. This decreased balance control is due to changes in neuronal mechanisms of balance. Although the present study does not identify the specific mechanisms, it describes the time course of changes occurring in the SOL H-reflex amplitude that lead to appropriate postural reactions in healthy adults. During backward tilt, H-reflex excitability seems to reflect appropriate balance control within the first 75 ms after the onset of the perturbation and during forward tilt, the change occurs within the first 100 ms after perturbation onset. Hence, the timeframe determined as being 75–100 ms seems to be critical for the involvement of neuronal mechanisms to induce appropriate postural reactions. In patients with CNS lesion, EMG activation (or inhibition) seems to be delayed (Thigpen et al. 2009; Marigold and Eng 2006) This might be a consequence of abnormal or delayed activation

of neuronal mechanisms beyond the 75–100 ms time frame, which could be a critical period beyond which balance control is perturbed. This timeframe could thus serve as a criterion to further understand mechanisms that might be altered following a CNS lesion.

Conclusion

H-reflex amplitude is modulated at the spinal cord level during forward and backward tilts. This modulation is induced before changes in EMG level during backward tilt, suggesting the involvement of central mechanisms in postural responses to perturbations. These mechanisms may include pre- and postsynaptic mechanisms contributing to change the motoneuronal excitability by supraspinal and spinal mechanisms, as well as modulation of afferent feedback (reflexes) to motoneurons. In future studies, knowledge of the relative contributions of the mechanisms underlying postural control would lead to a better understanding of balance control and a more precise assessment of balance deficits in individuals with injuries to the central nervous system.

Acknowledgements This work was supported by the Natural Sciences and Engineering Research Council of Canada (NSERC), Réseau provincial en adaptation-réadaptation du Québec (REPAR) and scholarships from Fonds de recherche du Québec en Santé (FRSQ) and Canadian Institutes of Health research (CIHR) to Annie Pham. The authors also wish to thank Daniel Marineau, Loyda Jean-Charles, El-Mehdi Meftah and Valérie Bernier for technical assistance.

Compliance with ethical standards

Conflict of interest The authors have no conflicts of interest to disclose.

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