Soleus Stretch Reflex Inhibition in the Early Swing Phase of Gait Using Deep Peroneal Nerve Stimulation in Spastic Stroke Participants

Marco M. Voormolen, MSc*,† ▪ Michel Ladouceur, PhD* ▪ Peter H. Veltink, PhD† ▪ Thomas Sinkjaer PhD*

**Center for Sensory-Motor Interaction, Department of Medical Informatics and Image Analysis, Aalborg University, Denmark and †Department of Biomedical Engineering, Faculty of Electrical Engineering, University of Twente, The Netherlands*

■ ABSTRACT

Objectives. To investigate the feasibility of inhibiting the stretch reflex of the soleus muscle by a conditioning stimulus applied to the deep peroneal nerve in spastic stroke participants during the early swing phase of gait.

Materials and Methods. This study investigated the effect of an electrical conditioning stimulus applied to the deep peroneal nerve on the magnitude at the peak of the soleus stretch reflex in the early swing phase of gait in six spastic stroke participants.

Results . Five of the six participants showed a reduced stretch reflex of more than 80%. On average ($n = 4$), it was shown that maximal inhibition occurred at a conditioning-test interval of 114 ms and had a magnitude of more than 90% ($p < 0.05$). For all five participants investigated, there was a significant reduction in the sensitivity of the soleus stretch reflex after conditioning ($p < 0.02$).

Conclusions. It is concluded that the inhibition of the soleus stretch reflex with an electrical conditioning stimulus applied to the deep peroneal nerve is feasible in the early swing phase of walking. This shows a potential for being used in the rehabilitation of walking by spastic stroke persons. ■

KEY WORDS: electrical stimulation, gait, inhibition, soleus muscle, spasticity, stretch reflex, stroke.

INTRODUCTION

Participants with injuries to the central nervous system (like multiple sclerosis, stroke, or incomplete spinal cord injury) show a significant reduction of the short latency stretch reflex (and/or H-reflex) modulation during gait(1–3). In stroke participants this impaired modulation results from the absence of an increased stretch reflex threshold in the swing phase in comparison to the stance phase of gait(4) as seen in able-bodied participants. This lowered threshold for the stretch reflex in spastic patients could lead to an increased soleus activity(5). Lack of foot clearance in the transition from stance to swing is often observed during walking by spastic participants, severely impairing their walking ability (4,6). It is often assumed to be related to the weakness of the dorsi-flexors, but could equally well be

Address correspondence and reprint requests to: M. Ladouceur, PhD, Aalborg University, Department of Medical Informatics and Image Analysis, Center for Sensory–Motor Interaction, Fredrik Bayers Vej 7D-3, 9220 Aalborg, Denmark. E-mail: mla@smi.auc.dk

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due to a reflex mediated tone in the plantar-flexors as they are stretched during this phase of the step cycle.

Inhibition of the soleus stretch reflex was found in both ablebodied and spastic participants lying supine with a conditioning stimulus applied to the deep peroneal nerve(7). Deep peroneal nerve stimulation was also found to inhibit the soleus stretch reflex in spastic stroke participants sitting with a relaxed or precontracted muscle(8). It is not known if results from the sitting task apply to walking since evidence has been presented that reciprocal inhibition is task-dependent(9). It is therefore necessary to extend to walking the results from the study of the inhibition of the soleus stretch reflex after stimulating the deep peroneal nerve in spastic stroke participants during sitting(8).

The objective of this study was to investigate the feasibility of inhibiting the stretch reflex of the soleus muscle by a conditioning stimulus applied to the deep peroneal nerve in spastic stroke participants during the early swing phase of gait.

METHODS

Six hemiplegic participants, from a stroke due to cerebral hemorrhage or infarction, volunteered for this study. Details of these six participants are summarized in Table 1. The study was approved by the local ethics committee and all participants signed an informed consent prior to the investigation.

Inclusion Criteria

The participants were a sample of convenience (not randomized). The criteria for inclusion were: (a) signs of spastic hypertonia in the left ankle plantar flexors (Ashworth score > 1); (b) no reports of peripheral neuromuscular diseases and/or injuries; (c) no reports of diseases of and/or injuries to the central nervous system other than the ones related to the stroke; (d) sufficient strength and coordination to walk on a treadmill for more than 5 min; and (e) an absence of inhibition of the soleus stretch reflex in the early swing phase.

Data Collection

Stretch reflexes where elicited during walking on a treadmill by an electromechanical device capable of stretching the human left ankle (Fig. 1). A detailed description on the design of the device is reported elsewhere(10). Briefly, the device consisted of a functional joint attached to the participant's ankle joint and an actuator system, involving a powerful AC-motor/gear system. The functional joint was mounted in alignment with the ankle joint and connected to the actuator system, which was placed next to the treadmill, by means of two flexible bowden wires. Position feedback was used to regulate the actuator and enable it to follow the movement of the ankle without influencing the pattern of gait. Whenever it was desired during the gait cycle, it was possible to evoke a stretch of the ankle extensor muscles by rotating the ankle joint with a displacement of up to 10 degrees.

Four carbon fiber-reinforced epoxy castings of different legs were made to have a good mechanical interface from the functional joint to the participant's ankle. The two-part casting was strapped to the calf and the foot with Velcro and broad adhesive tape. A pair of shoes was worn with the device attached to the leg. The right shoe was provided with an insole to compensate for the extra height

Table 1. Relevant details of the participants, all with left sided hemiplegia. The walking speed is the preferred walking speed of the participants. The stride duration is the average over at least 50 strides with its estimated standard deviation (SD).

Participant	Sex	Age	Months since stroke	Anti-spastic medication	Walking speed (km/h)	Stride duration (s)
JE	M	62	35	no	1.3	1.57 (0.14)
HC	M	52	44	no	4.0	1.11 (0.04)
PB	M	50	68	yes	1.3	1.92 (0.10)
EJ	M	53	82	no	0.5	2.08 (0.09)
BE	M	62	82	yes	1.6	1.69 (0.14)
DN	F	30	39	no	0.6	2.41 (0.12)

Figure 1. Front and back view of a participant walking while using the ankle stretch device.

of the left foot caused by the casting. The weight of the functional joint attached to the participant's leg was 0.9 kg. In order to adapt to the experimental set-up the participants were asked to walk on the treadmill for about 10 min with the device attached to the leg. During these 10 min unrecorded stretches were applied. After this adaptation period the recordings where started. To provide the appropriate timing for the stretches, foot switches were placed under the heel and under the fore foot. The timing was done from one of these two switches, whichever gave the most reliable timing. The amplitude of the soleus stretches was set to 8 degrees and the velocity was approximately ranging from 100 to 900 degrees per second.

Besides the conditioned and test stretches, two types of control recordings where collected: strides with the conditioning stimulus only and unaffected strides. The stretches were randomly mixed with the control recordings and applied infrequently. A recording (with stretch or control) was taken every two to four strides. To avoid fatigue, rest periods were added according to the participant's needs.

An electromyogram (EMG) of the soleus and tibialis anterior muscle of the impaired left leg was recorded with bipolar surface electrodes (centres 2 cm apart). The electrodes for the soleus EMG where placed over the distal end of the soleus muscle. A ground electrode (4 × 6 cm, oval) was placed on the shinbone near the ankle. The EMG signals were amplified and bandpass filtered from 20 to 1000 Hz. Angular position of the ankle and EMG signals were collected with a PC at a sampling rate of 2 kHz.

Conditioning Stimulus

The conditioning stimulus was applied to the deep peroneal nerve through a monopolar surface electrode (32 mm in diameter) which was placed distally and somewhat laterally in relation to the patella, close to where the deep peroneal nerve branches off the common peroneal nerve. To make sure that the common peroneal nerve was not stimulated, the tendons of the peroneus longus and brevis muscles were palpated while applying test stimuli. An anode was placed halfway on the shin (4 × 6 cm oval). The conditioning stimulus consisted of a train of five 1-ms pulses applied at a frequency of 200 Hz for a total duration of 21 ms.

The stimulus intensity was set at four times the motor threshold of the tibialis anterior muscle (MT), except for participant 2. For this participant a stimulus intensity of 3X MT was used because 4X MT was uncomfortable. A stimulus intensity of 4X MT was chosen to elicit the highest inhibition possible as seen in a previous study effectuated in sitting participants(8).

Data Analysis

The ankle position recordings were numerically differentiated to obtain the ankle velocity. The exact conditioning test interval and the stretch reflex latency were, respectively, determined from the onset of the stimulus artifact and the peak of the stretch reflex in relation to the onset of the stretches. Furthermore, the stretch velocity was defined as the average velocity of the 4.5–5.5 degree interval (out

of 8 degrees) of the stretch's ramp with the highest velocity.

The EMG signals were digitally rectified and low-pass filtered at 20 Hz. All recordings were examined on timing as well as stretch velocity and stretch amplitude, when appropriate. From the selected recordings the peak amplitude of the soleus short latency stretch reflex was obtained. Background EMG values were extracted from both types of control recordings by averaging the EMG activity for 50 ms starting from the times of the onset of the stretch reflex. Peak stretch reflex amplitudes and background EMG values were averaged over each trial. Background EMG was subtracted from the peak stretch reflex amplitudes to calculate the stretch reflex amplitudes.

The stretch reflex/stretch velocity relation was established by calculating the least squares linear regression between stretch reflex amplitude and stretch velocity for both test and conditioned stretches. Threshold (minimum stretch velocity at which just a detectable stretch reflex is elicited) and sensitivity (stretch reflex amplitude variation to a unity stretch velocity increase) of the stretch velocity/stretch reflex relation were also extracted.

Experimental Protocol

The participants were asked to walk with their preferred walking speed that ranged from 0.5 to 4.0 km/h (see Table 1). With all participants the following protocol was performed, except for participant JE for whom only the first two parts of the protocol were completed:

1. The determination of early swing. The stride duration was calculated from one of the foot switches and averaged over at least 50 strides. Early swing was defined and timed to occur 5% of the stride duration after toe off. All stretches in this experiment were applied at this moment in the gait cycle.

2. The optimal conditioning test interval. Test (unconditioned) stretches were applied with 4–6 different stretch velocities ranging from moderate to high (approximately 300–900 degrees per second). For each stretch velocity at least 10 stretch reflexes were recorded. Through a preliminary analysis, the average amplitudes of the stretch reflexes (without subtraction of the background EMG) were obtained and the least squares linear regression with stretch velocity was calculated. Subsequently, the average amplitudes of 4–10 stretch reflexes from conditioned stretches with four different conditioning stretch intervals were obtained. Inhibition values of these average stretch reflex amplitudes were calculated in relation to the regression line of the test stretches. The delay with the maximum inhibition was extracted and used in the characterization of the conditioned stretch reflex/stretch velocity relation.

3. The stretch reflex/stretch velocity relation. To investigate the influence of the conditioning stimulus on the stretch reflex/stretch velocity relation, test and conditioned stretches were applied with 4–6 different stretch velocities. At least 10 stretch reflexes were recorded for each stretch velocity, both for the test and conditioned stretches.

Statistics

The individual results from the optimal conditioning test interval measurements were tested with a one-way ANOVA test or a Kruskal-Wallis anova on ranks test in case of non-normality or unequal variance. Multiple comparisons were done with Dunnett's test for one-way ANOVA and with Dunn's test for Kruskal-Wallis anova on ranks. The combined results of the optimal conditioning-stretch interval measurements were tested with the Friedman repeated measures ANOVA on ranks test and multiple comparison was performed with Dunnett's test. All other results were tested with *t*-tests after verifying their normality with the Kolmogorov-Smirnov test ($p < 0.05$).

RESULTS

Time Course of the Responses

Figure 2 depicts the average stretch responses obtained from participant DN in the early swing phase of gait. With the exception of the clonic activity, the responses shown in Fig. 2 are representative of the whole population of participants. Panels A to D of Fig. 2 show the responses to a test (unconditioned) stretch whereas panels E to H show the responses to a conditioned stretch. In panel A to D the thin line shows the strides without a stretch

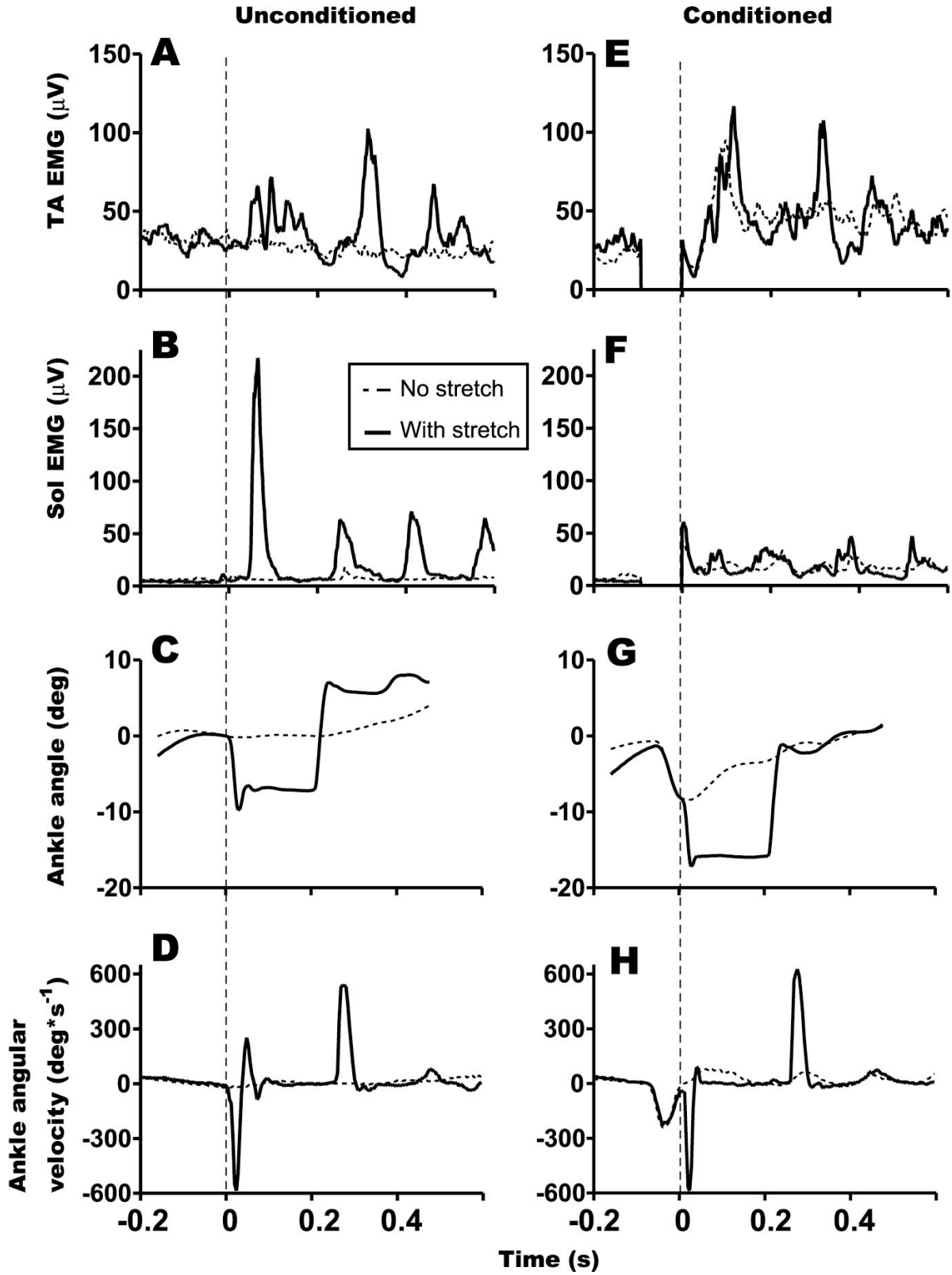

Figure 2. Example of the inhibition of the soleus stretch reflex in the early swing phase of gait in hemiplegic participants from a conditioning stimulus applied to the deep peroneal nerve. Average responses (n = 5) to an unconditioned (A to D) and a conditioned stretch (E to H) from participant DN. Thick lines show the recordings with a stretch and the thin dotted lines the control recordings. The control recordings of panels E to H show the conditioning stimulus with no ankle stretches.

whereas in panel E to H it shows the changes when only the conditioning stimulus was applied. The artifact of the conditioning stimulus was removed from the EMG recordings before rectifying and filtering, hence the blank period in the EMG recordings of Figs 2E and 2F.

The stretches had a velocity of 554 (SD, 16) degrees per second and an amplitude of approximately 8 degrees. In the test stretches a large reflex (Fig. 2B) was elicited with a peak occuring 50(SD:2.7) ms after the onset of the stretch. It can be seen in Fig. 2F that the stretch reflex following the conditioning stimulus was strongly suppressed. Furthermore, the clonus that was elicited after the test stretch (the three reflex bursts after the short latency reflex) was reduced, but still present, after the conditioned stretch. This effect was observed in the two participants that exhibited clonus activity (participants BE and DN). As seen in Fig. 2G, the twitch of the tibialis anterior muscle that was induced by the conditioning stimulus caused a dorsi-flexion of approximately 8 degrees. This dorsi-flexion stretched the soleus muscle and thus could have elicited a stretch reflex as indicated by the relative fast plantar-flexion that followed the dorsi-flexion caused by the stimulus in the control recording (Fig. 2G). However, a large stretch reflex is not present in the corresponding soleus EMG (Fig. 2F). Further considerations are discussed later.

The Optimal Conditioning Test Interval

An example of the results from the second part of the protocol obtained from participant DN is shown in Fig. 3A. The figure shows the average stretch reflex amplitude and stretch velocity for conditioned and test stretches in the early swing phase of gait. Conditioning stretch interval are also noted. Conditioned stretches were taken at conditioning test intervals of 54, 114, 167, and 266 ms (on average) for each participant.

The stimulation at different conditioning test intervals caused variations in ankle stiffness. These variations in ankle stiffness caused stretch velocity variations due to the limited stiffness of the stretching device. The conditioned stretch reflexes could therefore not be measured at matched stretch velocities with the test stretch reflexes. To compare the conditioned stretch reflexes with the test reflexes at matched stretch velocities the least squares linear regression of the test stretch reflexes was calculated. With this regression the inhibition of the conditioned reflexes was determined in relation to the regression line at their corresponding stretch velocity. Figure 3A shows the regression line with its 95% confidence intervals.

Figure 3B presents the individual time course of the inhibition seen in the conditioned stretch reflexes. It can be seen in that panel that five of the six participants showed a marked reduction (> 80%) of their stretch reflex. Figure 3C shows the grouped inhibition of the soleus stretch reflex and three of the four delays showed a significant reduction in the stretch reflex ($p < 0.05$). For both individual and combined results the stretch reflex amplitude was found to depend significantly on the conditioning stretch interval ($p < 0.001$). The highest inhibition was found to be at a conditioningtest interval of 114 ms (SD, 7.2) and had a magnitude of 91 ms (SD, 5.2). It should be noted that the inhibition results of participant JE and HC were not included in the combined results for the following reasons: (a) conditioning stretch intervals used with participant JE were not comparable to the other participants and (b) participant HC had a conditioning stimulus intensity that was not comparable to the other participants (3X MT instead of 4X MT). On average, the stretch reflex returned to its unconditioned value at a conditioning test interval of 266 ms (SD, 7.9).

The Stretch Reflex/Stretch Velocity Relation

To gather evidence on the neural mechanisms involved in the inhibition of the soleus stretch reflex during the early swing phase, stretch reflex/stretch velocity relations were established for the test and conditioned stretches by recording from stretches applied at 4–6 different stretch velocities. The conditioning stretch interval used for the conditioned stretches was the individually determined optimal conditioning test intervals from the second part of the protocol. As explained in the methods section the least squares linear regression with stretch velocity was calculated to establish the relation. Figure 4A shows an example of the regression lines and their 95% confidence intervals obtained from participant DN. The figure also shows the average stretch reflex amplitudes. Note the positive extrapolated *y*-axis intercept for the regression of the uncondi-

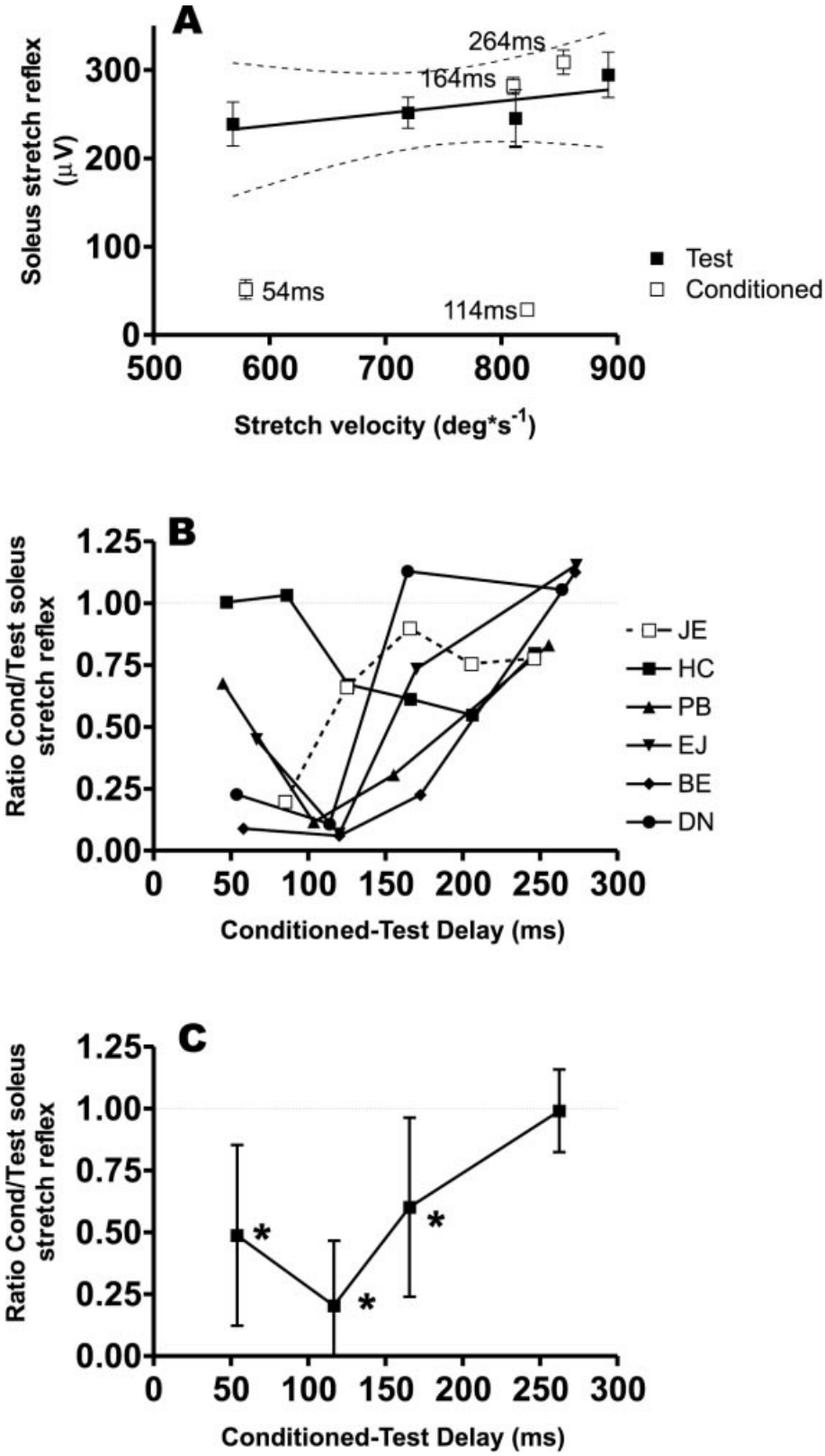

Figure 3. The time course of the inhibition from a conditioning stimulus applied to the deep peroneal nerve in the early swing phase of gait in hemiplegic participants. Panel A presents the raw results on the changes in the inhibition as a function of the conditioning test interval for one participant (DN). The stretch reflex inhibition, as calculated from the ratio of the amplitude of the conditioned over the test stretch reflex, are presented for each participant (Panel B) and as a group for four different conditioning test intervals (panel C). In both panels the dotted horizontal line represents no changes between the conditioned and test stretch reflex. In panel C the error bars represent the standard deviation. Significant changes are marked with an asterisk.

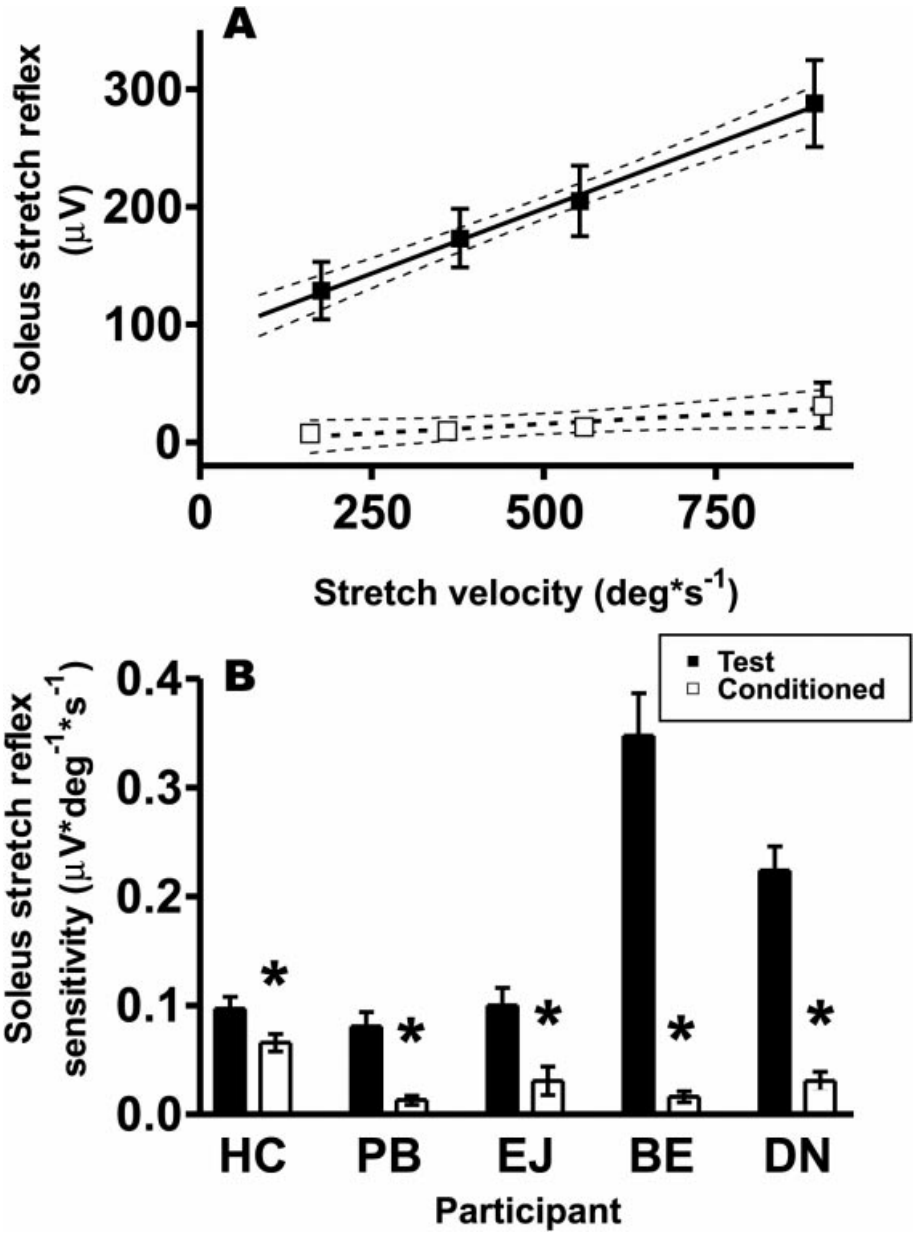

Figure 4. Effect of a conditioning stimulus on the stretch reflex amplitude–stretch velocity relationship. Panel A presents an example (participant DN) of the stretch reflex amplitude–stretch velocity relationship for test (solid squares) and conditioned (open squares) stretches. For both test and conditioned stretch reflex amplitudes the error bar represent the standard deviation and the linear regression is shown (line) with its 95% confidence interval (dotted line). The individual stretch reflex sensitivities for test and conditioned stretches are shown in panel B. The error bars represent the estimated standard error of the sensitivities and significant changes in the sensitivity are represented by an asterisk.

tioned stretch reflex amplitudes that could be translated into a negative threshold.

As seen in Fig. 4B, the stretch reflex sensitivities (ie, the slopes of the regressions) of the conditioned stretches were significantly smaller than that of the unconditioned stretches in all participants investigated ($p < 0.02$). Furthermore, the stretch reflex sensitivities of the conditioned stretches themselves were all significantly larger than 0 ($p < 0.02$). Changes in the stretch reflex threshold were not consistent with two participants showing an increased reflex threshold, two showing a decreased reflex threshold and two with no changes. Furthermore the results also were inconclusive because of the high variability of the calculated stretch reflex threshold.

An exploratory study with participant DN on the effect of the conditioning stimulus intensity is shown in Fig. 5. The conditioning stimulus in that figure consisted in a 1-ms pulse, applied at the optimal conditioning test delay, and at two different intensities (2X and 7X MT). Unlike in Fig. 4A it can be seen that in both conditioned relations there is a marked increase in the stretch reflex amplitude

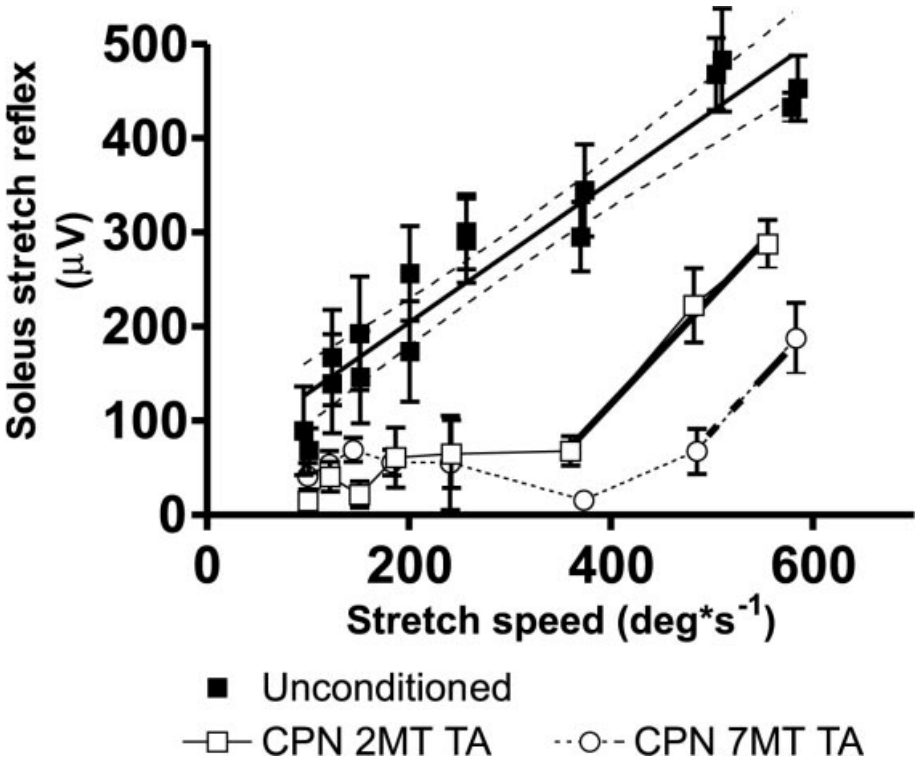

Figure 5. Effect of varying the conditioning stimulus intensity on the stretch reflex amplitude-stretch velocity relationship. This figure presents an example (participant DN) of the modification of the stretch reflex amplitude–stretch velocity relationship with a shorter conditioning stimulus pulse (1 pulse of 1 ms in comparison to a train of 5 pulse of 1 ms) and two different intensity (open squares, 2MT; open circles, 7MT). The error bars represent the standard error of the mean. The linear regression of the test stretch reflex amplitude–stretch velocity relationship is presented (line) with its 95% confidence interval (dotted line). Two lines are also drawn representing the slope in the rising part of the relationship for the conditioned reflexes (thick line, 2X MT; dotted line, 7X MT).

at either around 400 degrees per second for the 2X MT conditioned relation and 500 degrees per second for the 7X MT conditioned relation. Furthermore, it can be seen that the slope of the conditioned relations following those thresholds is somewhat similar to the one from the test stretch reflexes.

DISCUSSION

In summary, the results of this study show that the short latency stretch reflex was markedly reduced (> 90%) by applying a conditioning stimulus to the deep peroneal nerve in spastic stroke participants during the early swing phase of gait. The optimal conditioning test interval was found to be 114 ms (SD, 7.2). Furthermore, it was also shown that the sensitivity of the stretch reflex/stretch velocity relation was reduced in the conditioned relation. However, changes in the threshold of this relation were not consistent and due to their poor reliability no conclusions could be made.

The observed inhibition of the short latency stretch reflex could have resulted from two different afferent volleys: the direct afferent volley caused by the conditioning stimulus and the indirect afferent volley caused by the contraction of the tibialis anterior muscle, or from a combined effect of those two.

This indirect afferent volley could be originating from the contraction of the tibialis anterior muscle or the dorsi-flexion caused by the conditioning stimulus. Cooling the tibialis anterior has been shown to increase the delay of the optimal conditioning test interval(7). Furthermore, a stretch that precedes the onset of the test stretch has been shown to cause an inhibition over a conditioning test interval of more than 400 ms during upright stance in able bodied participants(11). This inhibition of the stretch reflex is similar to the postactivation depression seen for the soleus H-reflex(12). However, recent evidence from experiments with able bodied participants done after an anesthetic block of the common peroneal nerve, to abolish any indirect afferent volley, show that the observed inhibition was still present. This suggests that the inhibition is mediated, to a large extent, from the stimulation of afferents in the common peroneal nerve and not from the indirect afferent volley(13). Furthermore, the observed inhibition is also highly similar to the one reported in spastic stroke participants after stim-

ulating the deep peroneal nerve during sitting with a relaxed soleus muscle(8) in which the ankle was prevented from flexing with the conditioning stimulus.

Disynaptic reciprocal inhibition from muscle spindle Ia afferents of the tibialis anterior could be suggested as a possible pathway for mediating the observed inhibition. However, the time course of the inhibition as presented in Fig. 3A precludes such a conclusion since the latency of disynaptic reciprocal inhibition is smaller than 10 ms(14). It should be noted that this experimental protocol could not reveal if such a pathway was used since our shortest conditioning test delay was about 50 ms.

Based on the results that the sensitivity of the stretch reflex/stretch velocity was decreased it can be speculated that reciprocal presynaptic inhibition(15) from Ia afferents is more likely to have caused the observed inhibition. Such an explanation would be in accordance with the presumed effect of presynaptic inhibition on the stretch reflex sensitivity(4). However, it has been shown that sensitivity change can be achieved with a combination of weighted postsynaptic excitation and inhibition using a relatively simple motoneuron pool model(16). Furthermore, results presented in Fig. 5 could be used to speculate that the conditioning stimulus used in this study was effective to the point of keeping the stretch reflex below threshold for the whole spectrum of stretch velocities studied. This means that the threshold was shifted beyond the highest stretch velocity used. In that case, the best linear fit would be misleading and mechanisms that increase the stretch reflex threshold, like postsynaptic inhibition, changes in gamma motor neuron activity or changes in descending motor pathway activity from supraspinal centers(4) would also need to be considered. It should be noted that all stretch reflex sensitivities were significantly different from 0.

The precise mechanism(s) responsible for the observed inhibition cannot be derived from our experimental set-up and should be investigated in more details in the future.

Functional and Rehabilitation Significance

Some stroke participants lack the modulation of the short latency stretch reflex modulation between the stance and swing phase of gait(4). This lack of modulation increases the risk of eliciting a stretch reflex in the swing phase, which could cause problems with lifting the foot off the ground. The presented results indicate that the stretch reflex sensitivity can be reduced with a conditioning stimulus applied to the deep peroneal nerve. It would be interesting to see what impact the observed inhibition would have on the effectiveness of foot-drop stimulators. Furthermore, future studies should also investigate the relationship between the increased stretch reflex of spastic stroke patients during the early swing phase of gait and their impaired walking ability or, more specifically, their ability to clear their foot off the ground. The inhibitory effect of deep peroneal nerve stimulation on the stretch reflex in early swing could be valuable for such an investigation.

CONCLUSIONS

On average, deep peroneal nerve stimulation reduces the stretch reflex in the early swing phase of gait for more than 90% in spastic stroke participants at an optimal conditioning-test interval of 114 ms (SD, 7.2). The results also show a significantly reduced stretch reflex sensitivity in all participants investigated while no conclusion can be drawn from the results on the stretch reflex threshold. Both optimal conditioning stretch interval and reduced stretch reflex sensitivity suggest a presynaptic reciprocal inhibition, but further investigations are needed to investigate the neural mechanisms involved.

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