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RESEARCH ARTICLE

Medium latency excitatory reflex of soleus re-examined

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Abstract
We aimed to study the receptor origin and postsynaptic potential profile of the medium latency reflex (MLR) response that develops in the soleus muscle when common peroneal nerve of antagonist tibialis anterior (TA) muscle is electrically stimulated. To achieve this aim, we electrically stimulated common peroneal nerve and recorded surface electromyography (SEMG) responses of soleus and TA muscles of informed volunteers. Additionally, we recorded intramuscular EMG from the soleus muscle. Stimulation of common peroneal nerve induced a direct motor response (M-response) in the TA and MLR in SEMG of the soleus. Using voluntarily-activated single motor units (SMUs) from the soleus muscle we noted that there were two distinct responses following the stimulus. The first response was a reciprocal inhibitory reflex probably originating from the antagonist muscle spindle primary (Ia) afferents. This was followed by an indirect reflex response activated by the contraction of the TA muscle during the M-response. This contraction generated a rapid acceleration in the direction of dorsiflexion hence inducing a stretch stimulus on soleus muscle. The response of soleus to this stimulus was a stretch reflex. We suggest that this stretch reflex is the main contributor to the so-called soleus MLR in the literature. This study illustrated the importance of using SMUs and also using discharge-rate based analysis for closely examining previously 'established' reflexes.

Keywords Human reflex · Muscle spindle · Tibialis anterior · Peroneal nerve · Soleus motor units · Electrical stimulation

Introduction

Stretch reflexes have been used as tools to study the pathways that connect muscle receptors to motoneurons that innervate homonymous muscles (Hultborn et al. 1996; Yavuz et al. 2014). This information is then used in diagnosis and treatment of neuromuscular disorders such as testing level of spasticity and tracking recovery following spinal cord injury (Mullick et al. 2013; Stampacchia et al. 2004). Most common method for inducing the stretch reflex is to apply a passive stretch to a limb (Beith and Harrison 2004; Burke and Schiller 1976; Frigon et al. 2011; Gandevia et al. 1986; Mrachacz-Kersting et al. 2006; O'Sullivan et al. 1998; Suresh et al. 2005; van Doornik et al. 2009) and the response recorded in the target muscle is dependent on the method used, e.g. level/presence of voluntary contraction and torque velocity (Gandevia et al. 1986; Sinkjaer et al. 1988). In the hand muscles, stretch stimulus usually induces two prominent peaks based on latencies, which have been named M1 and M2 responses (Thilmann et al. 1991). An additional peak has been often observed in the shoulder and lower limb muscles (M3) (Petersen et al. 1998; Sato et al. 2014).

While the M1 response has been claimed to be due to non-monosynaptic activation of spindle primary afferents (Grey et al. 2001), the origin of the M2 and M3 responses, however, is not so clear and has been subject of much controversy (Burke et al. 1983; Christensen et al. 2001; Lee and Tatton 1978; Sinkjaer et al. 1999). The origin of M2 in hand muscles has been claimed to be cortical (Matthews et al. 1990). This claim has been challenged since it occurs too fast to be cortical and its latency for foot and hand muscles is the same (Darton et al. 1985; Thilmann et al. 1991). Instead, it has been suggested that Ib or group II afferents may be responsible for the M2 response (Dietz 1998; Dietz et al. 1985; Grey et al. 2001; Schieppati and Nardone 1997). The origin the M3 response has been claimed to be group

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Ia afferents and its circuitry goes through the motor cortex (Petersen et al. 1998).

Another method for inducing stretch reflex in an agonist muscle is to induce contraction in its antagonist muscle. Stretch reflex of triceps surae muscle with stimulus induced contraction of the antagonist tibialis anterior muscle (Uysal et al. 2009), and stretch reflex of finger flexor muscles via contraction of wrist extensor muscles (Uysal et al. 2012) have been described.

When this method is applied via electrical stimulation of the common peroneal nerve, a medium latency reflex response (MLR) is obtained in the soleus muscle surface EMG (SEMG) (Uysal et al. 2009). Origin of soleus MLR in these studies has been claimed to be the group II afferents due to its latency and its responses to cold and ischemia manoeuvres (Uysal et al. 2009, 2011, 2012).

In the current study we planned to re-examine soleus MLR reflex using single motor units (SMU) from the soleus muscle. We also planned to use both the classical probability-based and relatively new discharge-rate-based analyses to pinpoint the exact sign and shape of the underlying postsynaptic potentials related to MLR. Once we establish the postsynaptic potentials that our stimulus induces, we can then hypothesise on the possible reflex origin and circuitry that is responsible for the MLR response. Our main hypothesis is that the so called MLR response is a net result of multiple neuronal pathways.

Materials and methods

Subjects

Experiments were performed in Akdeniz University Hospital EMG/EEG Laboratory and Koç University School of Medicine Neurophysiology Laboratory. Total of 11 healthy males (age: 21.3 ± 1.2 years and body mass index: 22.5 ± 2.7 kg/m^2) with no chronic back pain, history of neuromuscular diseases and medication were recruited from Akdeniz University and Koç University. The reason for using male population only in the experiments was due more reliable detection of the stimulation site (common peroneal nerve at the head of fibula) in male subjects. The Human Ethics Committee of Koç University and Akdeniz University approved the experimental procedure conformed to the Declaration of Helsinki.

Setup

For recording and analysis, software Spike2 version 7.20 (Cambridge Electronic Design, Cambridge, UK) and Synergy (Synergy Healthcare Solutions, Maryville, USA) were used. The hardware of The Nicolet EDX (Natus Neurology, Middleton, USA), CED 3601 Power 1401, Micro1401-3 DAC (Cambridge Electronic Design, Cambridge, UK) and linear strain gauge (Model LC1205-K020, A & D Co. Ltd., Tokyo, Japan: linear to 196 N) force transducer were used for data recording. A constant current stimulator (model DS7A, Digitimer Ltd, Hertfordshire, UK) and The Nicolet EDX system were used for electrical stimulation.

Electromyography

SEMG were recorded from both soleus and tibialis anterior (TA) muscles of left leg in a bipolar configuration with 20,000 Hz sampling rate and 20–500 Hz bandpass filter. After routine preparation (rubbing the skin, cleaning with alcohol and conductance gel application), two Ag/AgCl SEMG electrodes were placed on both muscles**.** Subjects were asked to perform strong dorsiflexion to observe exact location of the TA muscle and two SEMG electrodes were placed one on the muscle belly and the other 4 cm apart on the distal part of the muscle. For the soleus muscle, subjects performed strong plantar flexion and one SEMG electrode was placed on the posterolateral part of the leg (slightly distal to lateral head of gastrocnemius) and other electrode was placed 4 cm distal to the first electrode allowing an intramuscular electrode to be inserted in between the two surface electrodes (Tucker et al. 2005).

For, SMU recording from soleus muscle, we used sterile and disposable silver fine-wire electrodes coated with Teflon (75 µm in core diameter; Medwire, New York, USA). The tip of the wires was stripped for 3–5 mm to increase the active recording area to detect gross SMU response referred to as multi-motor unit (MMU). The needle with wires was inserted between two SEMG electrodes and subjects was asked for brief plantar flexion for improved wire electrode stabilization inside the soleus muscle and then the needle was withdrawn leaving a pair of fish-hooked wires inside the soleus. The SMU recording was sampled with 20,000 Hz and filtered with 200–5000 Hz bandpass filter. The ground electrodes were placed on medial and lateral malleolus for soleus and TA, respectively.

Experimental procedure

Subjects rested in prone position and asked to perform low intensity plantar flexion against a fixed platform to recruit low-threshold SMUs from the left leg with the help of visual feedback. A force transducer was fixed to this platform to record the twitch force. During sustained SMU recruitment, electrical stimuli with 1-ms pulse width and 1–2 s of interstimulus interval (Burke et al. 1989) were delivered to common peroneal nerve around the head of the fibula with bipolar stimulating electrodes which were fixed with an elastic bandage to reduce the movement of the electrodes during twitch. For each subject, the stimulus intensity was adjusted

to have an observable direct motor response (M-response) with twitch in TA and MLR in soleus SEMG with no field potential in SMU channel at the MLR latency. In addition, different intensity stimulations of common peroneal nerve, described in "Results" section, were also used.

After MLR experiments, subjects were asked to perform 3 maximum voluntary plantar flexions for soleus and dorsiflexions for TA to normalize the levels of contractions. These maximum contractions were followed by obtaining the maximum soleus M-response and determining H-reflex latency of soleus. For maximum soleus M-response, we stimulated the tibial nerve until no further increment in soleus M-response was detected. The reason for taking soleus maximum M-response was to normalize the MLR response and soleus H-reflex according to maximum M-response to see how large the responses, but not for comparison purposes (an example is provided in Fig. 1a, c). Then, stimulating electrodes relocated to midpoint of the popliteal fossa to find H-reflex latency in soleus to compare it with the MLR latency to understand the MLR circuitry (Özyurt et al. 2018). Also, maximum M-response and H-reflex latency for TA muscle were also recorded. The

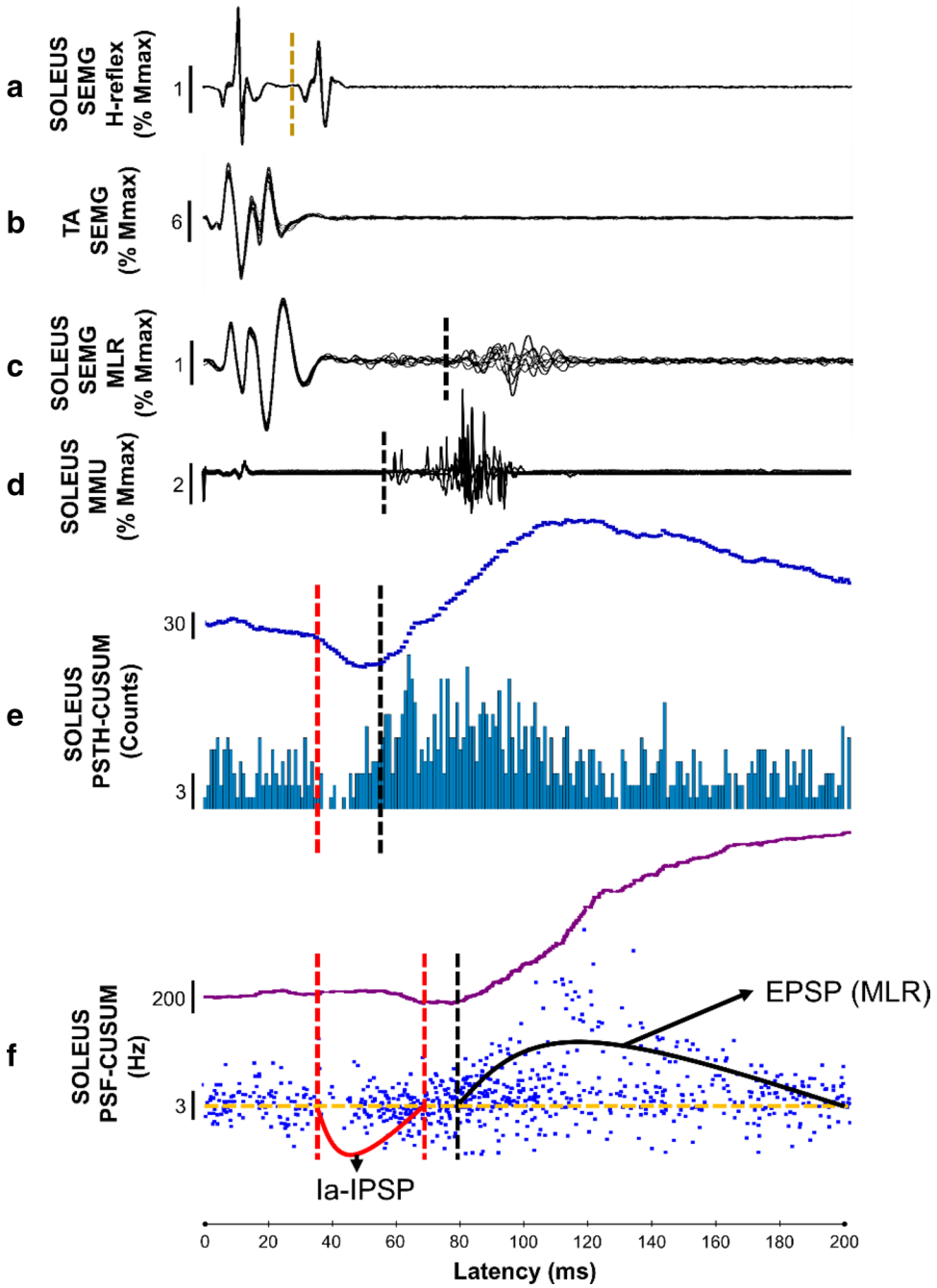

Fig. 1 Responses with different stimulation and analysis methods. **a** Soleus SEMG recording demonstrates the H-reflex when tibial nerve was stimulated. **b**–**f** Common peroneal nerve was stimulated to induce M-response without H-reflex in TA. **b** TA averaged M-only response (SEMG). **c** Soleus SEMG with superimposed MLRs. **d** Soleus MMU with superimposition of SMUs fired at MLR latency. **e** Firing probability of soleus SMUs in PSTH-CUSUM presents IPSP and MLR. **f** The discharge rate of soleus SMUs in PSF-CUSUM also shows IPSP and MLR. Red dashed lines are the onset of IPSP and black dashed lines are the onset of MLR, gold dashed line is the onset of soleus H-reflex. The yellow horizontal dashed line in PSF is the average discharge rate. Red and black parabolic curves are imaginary profile of Ia-IPSP and MLR-EPSP, respectively

maximum TA M-response was obtained similarly and the rationale behind taking this recording was to determine the intensity of common peroneal nerve stimulation. The reason for finding TA H-reflex was to compare the central delay of the responses with the monosynaptic H-reflex.

Analysis

Twenty-three distinct SMUs analysed for supramaximal stimulation experiments from 9 participants. (2 SMUs from 5 subjects, 3 SMUs from 3 subjects, 4 SMUs from one subject). To determine the effect of the stimulus, we also performed submaximal stimulus experiments in four SMUs recorded in another 2 subjects (total of 11 subjects) and compared it with supramaximal response of the same 2 subjects. During SMU recruitment, 308 ± 141 stimuli were delivered to the common peroneal nerve. The average background firing rate of the units was 6.33 ± 1.13 Hz. We investigated the duration of the inhibitory postsynaptic potentials (IPSP) and excitatory postsynaptic potentials (EPSPs), central delay of the IPSP and latency of EPSP.

For SMU analysis, we used template matching algorithm in Spike2 and converted recognizable action potential shapes into acceptance pulses. Using the acceptance pulses, we built peristimulus time histogram (PSTH), peristimulus frequencygram (PSF; Türker and Powers 2005) and their cumulative sum (CUSUM) for each unit (Ellaway 1978). Besides using the acceptance pulses of selected units, we also used the rectified averaged analysis of the MMU response to obtain a more general response of the muscle.

Latencies of IPSP and EPSP were calculated using the onset of the reduced spike (PSTH) or EMG (MMU and SEMG) activity compared to prestimulus average activity. EPSP latency was calculated as the onset of increased activity compared to prestimulus activity. On the other hand, the latency of IPSP and EPSP in PSF was calculated using reduced or increased firing rate of SMUs compared to background discharge rate, respectively. For all analysis methods, CUSUM error box approach was used to detect the significant changes in the records (Brinkworth and Türker 2003).

The latency and duration of the soleus MLR were noted and H-reflex latency values were compared with MLR to calculate central delay. Therefore, we calculated the central delay as the additional latency difference between the H-reflex and IPSP latency in the experiments. This delay occurs due to Ia inhibitory interneuron at spinal cord, so referred as "central".

Statistics

The duration of the IPSP was compared between PSTH, PSF, and MMU using repeated measures one-way ANOVA with Tukey correction. The duration of soleus MLR (EPSP) between PSTH, PSF, MMU, and SEMG, on the other hand, was compared using ordinary one-way ANOVA where multiple comparisons were corrected with Tukey test, and, a similar test was applied to check the latency variations of MLR between the various analysis methods (in their CUSUMs). The level of significance was set at $p < 0.05$.

Results

Stimulation of common peroneal nerve induced an IPSP which was followed by an EPSP in soleus muscle

The SEMG and SMU responses to electrical stimulation of common peroneal nerve were recorded and analysed using averaged SEMG, PSTH and PSF (Fig. 1). The soleus H-reflex at SEMG was obtained by stimulating the tibial nerve (Fig. 1a). Common peroneal nerve stimulation resulted in M-response at SEMG of TA muscle (Fig. 1b) and MLR in soleus SEMG (Fig. 1c) as well as accumulation of SMUs between 50 and 120 ms after the stimulus (Fig. 1d). A reduction in firing probability of SMUs was also observed immediately before this spike accumulation as shown in PSTH-CUSUM (Fig. 1e). The PSF analysis, however, indicated that the reduction in discharge rate continued much longer than the spike reduction probability in PSTH and its CUSUM. This reduction in discharge rate was then followed by an increased rate of SMU firing for an extended period (Fig. 1f).

The duration of IPSP was shorter than EPSP

The duration of IPSP [$F(20, 40) = 1.416$] and EPSP [$F(3, 71) = 28.40$] were calculated for all experiments (Fig. 2). Duration of the IPSP obtained using PSF, 24.5 ± 6.3 ms, was significantly longer than that calculated using PSTH ($p < 0.0001$) and MMU ($p < 0.0001$) which were 15.4 ± 3.1 ms and 15.8 ± 3.2 ms, respectively. However, no significant difference of duration was found between PSTH and MMU ($p = 0.9304$) (Fig. 2a). On the other hand, the duration of EPSP, i.e. MLR, in PSF was found to be 119.3 ± 54.3 ms which was significantly longer than the EPSP duration found using SEMG, PSTH and MMU methods ($p < 0.0001$). No significant difference was noted between other analysis methods (PSTH vs MMU: $p = 0.8594$, PSTH vs SEMG: $p = 0.6983$, MMU vs SEMG: $p = 0.3374$) (Fig. 2b).

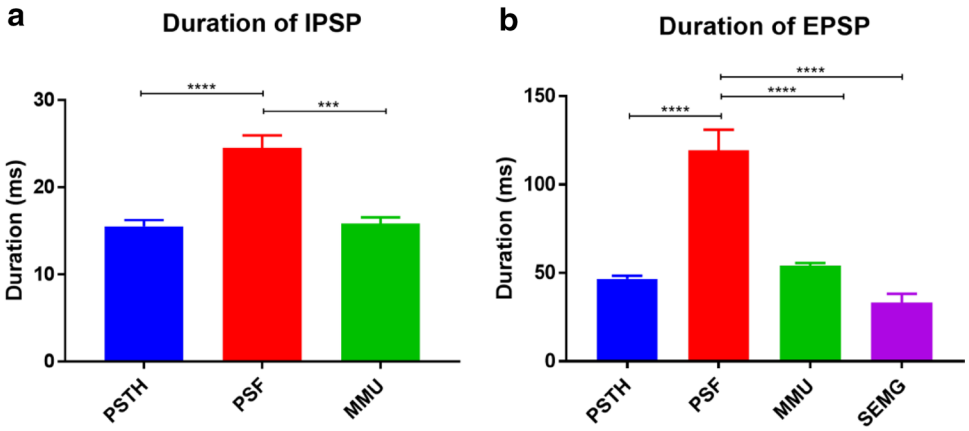

Fig. 2 Duration of the responses calculated using various techniques. **a** IPSP duration at PSTH, PSF, and MMU and **b** EPSP duration at PSTH, PSF, MMU, and SEMG are shown. Error bars are standard errors. ***$p<0.001$, ****$p<0.0001$

IPSP central delay and EPSP latency differs in various analysis methods

Conduction delay in the spinal cord and the latency of IPSP and EPSP was calculated (Fig. 3). IPSP latency calculated from PSTH and PSF was 36.43 ± 2.6 ms and that was significantly shorter than the latency of 38.59 ± 2.9 ms measured in MMU ($t=3164$; $df=22$, $p=0.0045$) (Fig. 3a). The central delay was found by subtracting the TA H-reflex latency from the latency of IPSP. The central delay of 4.92 ± 2.3 ms in PSTH was significantly shorter than that calculated in MMU of 7.1 ± 3.2 ms ($t=2905$; $df=17$, $p=0.0099$) (Fig. 3b). The latency of EPSP [$F(3, 71)=2087$] was found higher in PSF and SEMG as 68.3 ± 9.9 ms and 74.5 ± 9.7 ms, than PSTH and MMU which were 56.3 ± 4.6 ms and 57.6 ± 4.2 ms, respectively ($p<0.0001$) (Fig. 3c). When EPSP latency compared between PSF and SEMG ($p=0.1598$) as well as PSTH and MMU ($p=0.9447$), no significant difference was observed.

Both submaximal and supramaximal common peroneal nerve stimulation result in early IPSP followed by late EPSP

Since supramaximal stimulation of common peroneal nerve causes cross-talk in soleus muscle (see Fig. 1c), we investigated if submaximal stimulation for common peroneal nerve results in similar IPSP followed by EPSP phenomenon in all of the 4 different SMUs (one example in Fig. 4). Therefore, we stimulated the common peroneal nerve with an intensity that generated M-response in TA muscle about 50% of Mmax but no M-response in soleus muscle (Fig. 4a). Submaximal stimulation resulted in IPSP as a first event which is followed by EPSP. Then, we increased the stimulus intensity to the level that evokes maximal M-response in TA while the same SMU was recruited with the submaximal stimulation (Fig. 4b). Both approaches produced identical events in the soleus muscle, i.e. early IPSP followed by late EPSP.

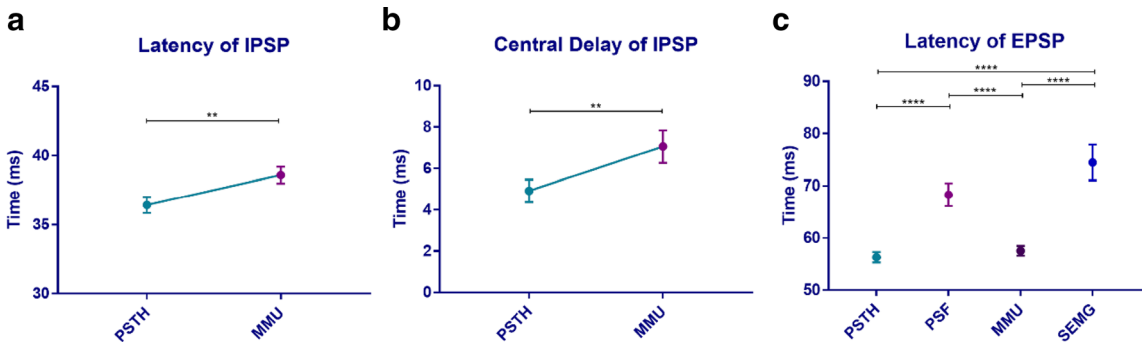

Fig. 3 Central delay of IPSP and latency of EPSP analysed with various methods. **a** The latency of the IPSP calculated using PSTH and MMU are shown. **b** Central delay of IPSP at PSTH and MMU are represented. **c** The latency of EPSP at PSTH, PSF, MMU, and SEMG are shown. Error bars are standard error. **$p<0.01$, ****$p<0.0001$

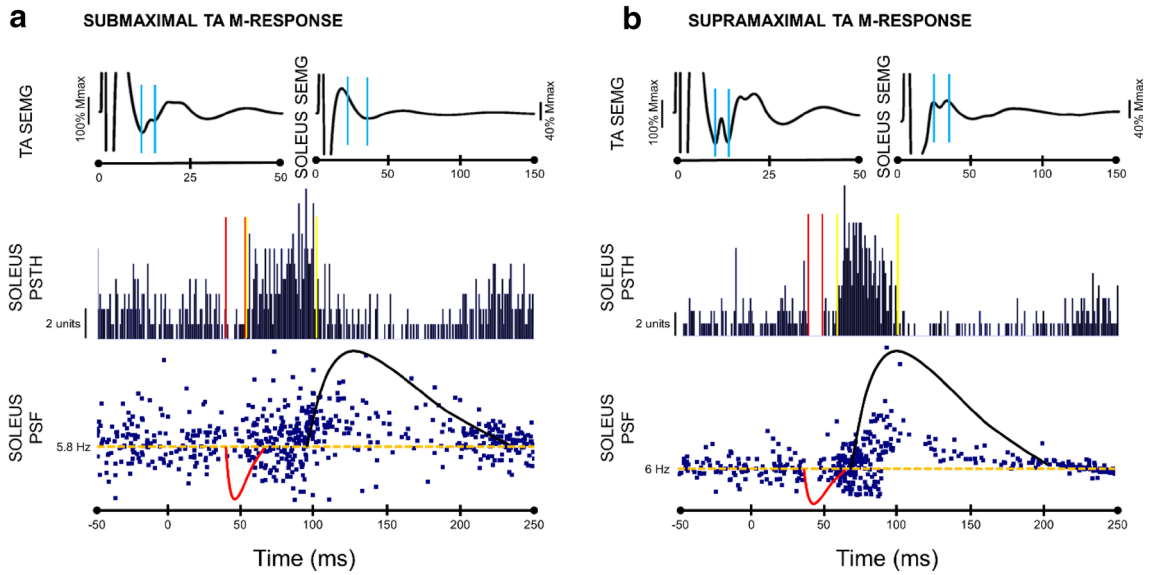

Fig. 4 Characteristics of IPSP and EPSP for sub- and supramaximal electrical stimulation. Stimulus intensity was determined according to TA SEMG at the top trace. **a** Submaximal intensity at 50% Mmax in TA muscle was free of soleus M-response, as shown in figure at the top. PSTH, in the middle, and PSF at the bottom revealed IPSP that is followed by EPSP in soleus. **b** Supramaximal intensity to produce Mmax in TA muscle was contaminated with soleus M-response, as shown in figure at the top. Although the stimulus intensity was higher compared to submaximal stimulation, the same IPSP and EPSP profile was observed. Blue lines at SEMG traces are M-response period. Red lines at PSTH are the duration for IPSP while yellow lines indicate the EPSP duration. Orange line at the PSF illustrates the background discharge rate of that particular SMU. Red and black curves represent imaginary synaptic profile for IPSP and EPSP, respectively

Discussion

We propose two novel findings in this study: Firstly, stimulation of common peroneal nerve evokes an early IPSP which is followed by a late EPSP in soleus muscle. Secondly, we obtained the profile of IPSP and EPSP (soleus MLR) using frequency-based analysis, PSF (Türker and Powers 2005).

These findings are quite unlike our previous findings using similar methodologies. Our previous studies using a similar protocol suggested that MLR in soleus SEMG was due to a delayed excitation response induced by activation of spindle secondary afferents (Uysal et al. 2009, 2011, 2012). Using single motor unit activity from the soleus muscle and peristimulus frequencygram technique we have now shown that the soleus MLR is a combination of two responses; an early Ia reciprocal IPSP and a late EPSP induced by a combination of circuitries including Group Ia + Group II EPSPs from soleus muscle receptors, and Group Ib EPSP from the TA muscle.

Soleus MLR response and its origin

Soleus MLR response can be elicited in two different ways. Firstly, using electrical stimulation of the common peroneal nerve and secondly using transcranial magnetic stimulation (TMS) which is evoked by shortening of TA muscle, thus, stretching the soleus muscle. Since both are referred to as MLR response of the soleus muscle we need to discuss the two responses separately.

Using electrical stimulation of the common peroneal nerve method, similar to the current protocol, Uysal et al. (2009) suggested that the MLR in soleus SEMG was due to a delayed excitation response induced by the activation of spindle secondary (Group II) afferents. This claim was due to its late latency and its responses to cold and ischemia manoeuvres (Boyraz et al. 2009; Uysal et al. 2011, 2012).

Using TMS methodology, Sammut et al. (1995) found that the TMS induced a direct motor response (M-response) in the TA muscle after about 30 ms and MLR response in soleus muscle with a latency of about 75 ms. Therefore, they have suggested that the soleus late response elicited by TMS is a soleus stretch reflex resulting from dorsiflexion of the foot due to activation of TA (when ankle position is not stable) following cortical stimulation. In the same year Ertekin et al. (1995) described the same MLR response by transcranial stimulation and concluded that soleus MLR response was a polysynaptic extensor response related to postural mechanisms and originating through convergence of descending motor commands and peripheral sensory feedback (Ertekin et al. 1995). Suga et al. (2001) described the same soleus MLR response elicited by TMS. They suggested that the response may thus be a polysynaptic response

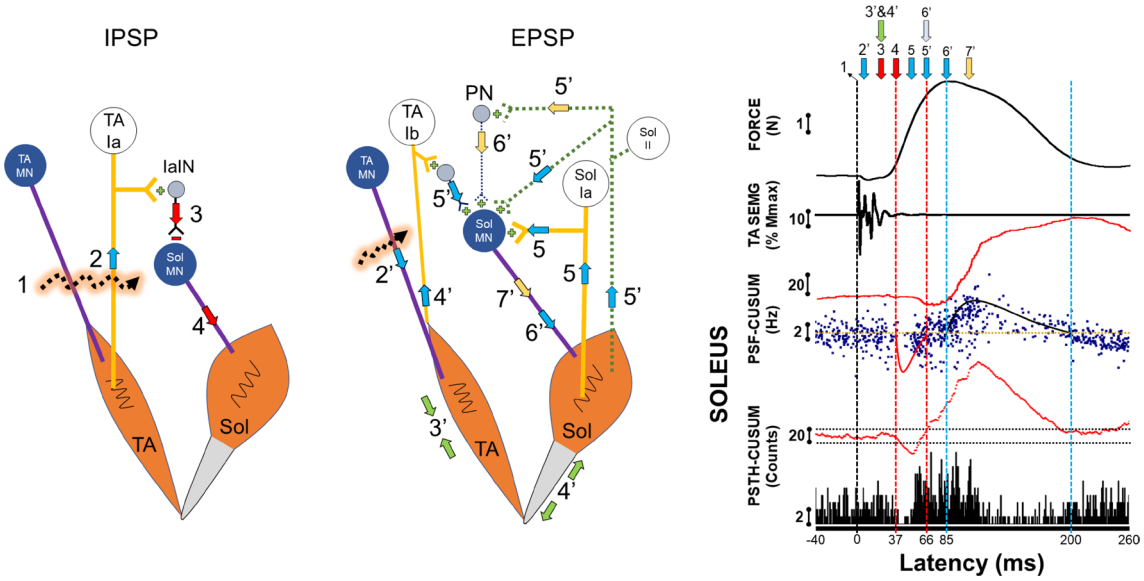

Fig. 5 Proposed mechanisms behind the IPSP followed by soleus (Sol) MLR. Left figure: is the proposed wiring diagram of the circuitry for IPSP and the middle figure is the proposed diagram of the circuitry for EPSP. Electrical stimulation of the common peroneal nerve (1) stimulates both primary afferents (2) and TA motoneurons (MN) (2′). TA motor axon stimulation causes TA muscle to contract (3′) which leads to tendon organ to be activated (4′) and may involve reciprocal Ib excitation (5′). Moreover, TA motor axon stimulation also leads to soleus muscle to be stretched (4′) thus activating group I (5) and maybe group II (green dashed line) spindle afferents (5′) after some electro-mechanical delay. Simultaneously, activation of primary afferents of TA muscle (2) initiates reciprocal inhibition circuitry (3) through Ia inhibitory interneurons (IaIN). Inhibition at soleus muscle is the first observable event (4). Following the inhibition, TA twitch mediated stretch reflex excites the soleus motoneurons via primary afferents (Ia) (6′). The late loop of group II afferents may also be involved in the excitation through propriospinal neurons (PN) which may increase the duration of MLR (7′). Green arrows are muscle shortening and lengthening. Red arrows are inhibitory systems. Blue arrows are short and yellow arrows are the late circuitries. Right Figure: is the time course of the wiring diagram recorded from the TA and soleus muscles and analysed with different methods. The arrows at the top of force recording are the proposed timings of the circuitry. Force recording revealed muscle twitch following TA M-response. This resulted in decreased soleus motor unit discharge rate followed by a slightly delayed increase in discharge rate of the units in PSF-CUSUM. In addition, reduction in the probability of firing is observed in PSTH-CUSUM at the bottom. This reduction is immediately followed by an increase in firing probability of the units. The vertical black dashed line is the onset of the stimulation (1), red dashed line is the onset of the inhibition, blue dashed line is the onset of excitation in PSF-CUSUM. Red and black parabolic curves in PSF are the proposed profile of IPSP and EPSP, respectively. The yellow horizontal dashed line in PSF is the average discharge rate, and black horizontal dashed lines in PSTH-CUSUM are the error bars

related to the postural control of the agonist and antagonist organization between the TA and soleus (Suga et al. 2001).

As can be seen there are two different views on the origin of MLR response in soleus muscle. One is the suggestion by Sammut et al. (1995) who claim that TMS-induced soleus MLR is a secondary stretch reflex response induced by contraction of the TA muscle. Therefore, TMS-induced MLR may have originated from stretch sensitive group Ia fibres in the soleus. On the other hand, the origin of MLR induced by the electrical stimulation of the common peroneal nerve has been reported to be group II fibres with respect to the response to cold manoeuvres and response to ischemia and tizanidine (Boyraz et al. 2009; Uysal et al. 2011, 2012).

Our results indicated that electrical stimulation of the common peroneal nerve-induced soleus MLR is not a pure excitatory reflex but made up of two distinct components. First is an inhibition originating possibly from antagonist Ia IPSPs and second is a long latency EPSP originating from stretch reflex of the soleus and reciprocal excitation by TA Ib afferents due to stimulus-induced contraction (Pierrot-Deseilligny et al. 1981).

Stimulation of common peroneal nerve evoked an IPSP which was followed by an EPSP in soleus muscle

In the current study, using regularly-active SMUs from the soleus muscle and discharge-rate analysis (PSF), we found that there was a genuine Ia reciprocal inhibition followed by a stretch induced reflex response in the soleus. The first response is due to the well-known Ia-IPSP pathway connecting antagonist muscles (Hultborn et al. 1976; Katz et al. 1991; Laporte and Lloyd 1952).

In this study, the latency for this inhibitory potential was about 36 ms with a central delay of 5 ms, calculated from SMU probability. This central delay may be due to a number of interneurons that Ia-IPSP volleys have to pass before reaching soleus motoneurons. Also, the conduction distance is slightly longer than the H-reflex arch in the soleus.

Electrical stimulation of the common peroneal nerve generated TA muscle contraction hence generating a movement of the foot in the dorsiflexion direction. This movement acts as a stretch stimulus for the triceps surae muscles and hence it results in the stretch reflex of the soleus muscle. In Fig. 5, we detailed the steps of these two events one by one. It is important to note here that the latency of the excitation in this study (75–80 ms for SEMG) was similar to the soleus MLR latency in the literature.

Limitations of the study

We have observed that suprathreshold stimulation of the common peroneal nerve also generated an M-response in the soleus SEMG (Figs. 1c, 4b). This is most likely to be a cross-talk. However, we are confident that both sub- and supramaximal activation of the common peroneal nerve generates two responses in soleus motor units, an IPSP followed by an EPSP. IPSP is very much likely to be of Ia-IPSP of origin and EPSP or MLR is due to soleus stretch reflex. In cases where soleus motor axons are also activated (observed in some cases where suprathreshold stimulation of the common peroneal nerve is used), it is possible that the early IPSP may be caused by a combination of Ia-IPSP from the TA muscle and Renshaw IPSP from the soleus muscle motoneuron pool, even though Özyurt et al. (2019) showed the duration of recurrent inhibition that is longer than Ia-IPSP (20–25 ms vs 30–45 ms calculated using PSF).

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Compliance with ethical standards

Conflict of interest Authors declare that they have no conflict of interest to disclosure.

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