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RESEARCH ARTICLE

Effects of vibration on cutaneous silent period

Şenay Aydın[1,2] · Markus Kofler[3] · Yeliz Bakuy[2] · Ayşegül Gündüz[2] · Meral E. Kızıltan[2]

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Abstract

Suppression of an ongoing muscle contraction following noxious digital stimulation is called cutaneous silent period (CSP) which is under the influence of several physiological factors. In this study, we aimed to evaluate the influence of group Ia afferents on the cutaneous silent period (CSP) by applying 2-min vibration. CSP was obtained from abductor pollicis brevis muscle after stimulating index finger. The recordings were repeated three times—before, during and after vibration—which was applied over the tendon of flexor carpi radialis muscle. Onset latency, duration and magnitude of total CSP, inhibitory phases I1 and I2, and of the long-loop reflex were measured and compared. Suppression indices of CSP, I1 and I2 increased significantly during and after vibration, indicating significantly less exteroceptive EMG suppression outlasting the time of vibration. Vibration also caused mild shortening of I2 end latency ($p = 0.048$) and I2 duration ($p = 0.019$). Our findings indicate that vibration exerts a powerful influence on CSPs and causes reduction in the magnitude of exteroceptive EMG suppression during and after vibration. Although vibration is known to activate Ia afferents, we cannot exclude contribution of other afferents, e.g. mechanoreceptors, as well as pre- or postsynaptic inhibitory effects on ensuing interneurons, or enhanced vibration-related excitatory influence.

Keywords Cutaneous silent period · Vibration · Group Ia afferent fibers · Alpha motoneuron · A delta fiber · Exteroceptive suppression

Introduction

Electromyographic (EMG) activity during voluntary muscle contraction can be suppressed temporarily by noxious electrical stimulation of a pure cutaneous nerve, i.e. the cutaneous silent period (CSP) (Inghilleri et al. 1997; Kofler and Poustka 2005; Uncini et al. 1991). Similar to cutaneomuscular reflexes following low-intensity electrical stimulation, three separate phases of EMG modulation can be distinguished: a first inhibitory phase (I1), a second inhibitory phase (I2), and an excitatory phase interrupting the periods of inhibition, which may contain a transcortical long-loop reflex (LLR).

Most authors agree that the presence of a CSP is dependent on intact small-diameter A-delta fibers, while the efferent reflex arm is formed by large-diameter alpha motoneurons (Leis 1998; Mota et al. 2015; Kofler et al. 2014). The CSP pathway is known to be devoid of μ-opiate receptors (Inghilleri et al. 2002). Despite its inhibitory nature, the circuit is not modulated by GABA-B agonists (Stetkarova and Kofler 2013), while monoaminergic modulation has been demonstrated by serotoninergic, noradrenergic and dopaminergic substances (Pujia et al. 2012, 2014; Serrao et al. 2002). High-frequency, low-intensity, transcutaneous electrical nerve stimulation (TENS), an antinociceptive strategy, which stimulates large-diameter afferent fibers and induces presynaptic inhibition, was reported to shorten the duration of CSP (Kofler 2004). CSP duration is under control of afferent proprioceptive impulses (Kofler 2004). According to Serrao et al. (2001), large-diameter afferents provide a small contribution to the CSP maintaining the excitability of interneurons and probably providing an additional security for the protective reflex (Serrao et al. 2001). A study recording CSPs in the foot after local anesthesia showed no change in end latency suggesting the role of large fibers

✉ Şenay Aydın
aydin.senay@hotmail.com

1 Department of Neurology, Yedikule Chest Diseases and Chest Surgery Training and Research Hospital, Istanbul, Turkey

2 Department of Neurology, Cerrahpasa Medical Faculty, Istanbul University-Cerrahpasa, Istanbul, Turkey

3 Department of Neurology, Hochzirl Hospital, Zirl, Austria

in determining end latency by reactivation of motor fibers after cessation of the effects of the inhibitory input (Mota et al. 2015).

Although vibration is not selective and may also activate other mechanoreceptors, it primarily triggers Ia afferent discharge (Fallon and Macefield 2007). In case, vibration-related influences on Ia afferent input, mechanoreceptors, or other pathways played a role at all in exteroceptive EMG modulation, we hypothesized that vibration would affect respective CSP parameters. Therefore, we aimed to investigate the effect of vibration on CSP in a population of healthy subjects.

Subjects and method

Subjects

Fourteen healthy individuals (8 females, mean age, 33.8 ± 6.7 years) without any neurological or systemic disease were included in the study. The study was approved by the local ethical committee and all participants gave informed consent.

Electrophysiological evaluation

All recordings were obtained using Ag-AgCl surface electrodes (Neuropack Σ-MEB-5504K, Nihon Kohden Corporation, Tokyo, Japan). Individuals sat on a comfortable armchair with the arms and hands held in a neutral position.

CSP recordings were performed in accordance with previous reports (Kofler 2003, 2004). CSP was recorded from abductor pollicis brevis (APB) muscle after noxious stimulation of digital nerves at the index finger while subject was performing a moderate contraction (Kofler et al. 2007; Rodi and Springer 2011). Stimulus intensity was 20 times sensory threshold, established in each individual at baseline before vibration. We used the same intensity for each condition (before, during, and after vibration). Twelve consecutive recordings were obtained at random intervals, rectified, amplified, and on-line averaged. Sweep duration was 300 ms. Auditory and visual feedback of the EMG signal was provided to facilitate the control of muscle activity. A prestimulus delay of 120 ms was set to enable the measurement of baseline EMG activity. Filter settings were 30 and 10,000 Hz. CSP recordings were repeated before, during and immediately after a 2-min period of vibration (Beurer Hand Held Massager, M70, Ulm, Germany), applied on the tendon of flexor carpi radialis muscle close to the wrist on the volar side of the right forearm with 100 Hz in frequency and 1 mm in amplitude. Care was taken to avoid as much as possible stimulating neighboring tendons or muscles. In all CSP recordings, three periods were distinguished (Fig. 1). We measured the following parameters similar to previously published reports (Kofler 2004).

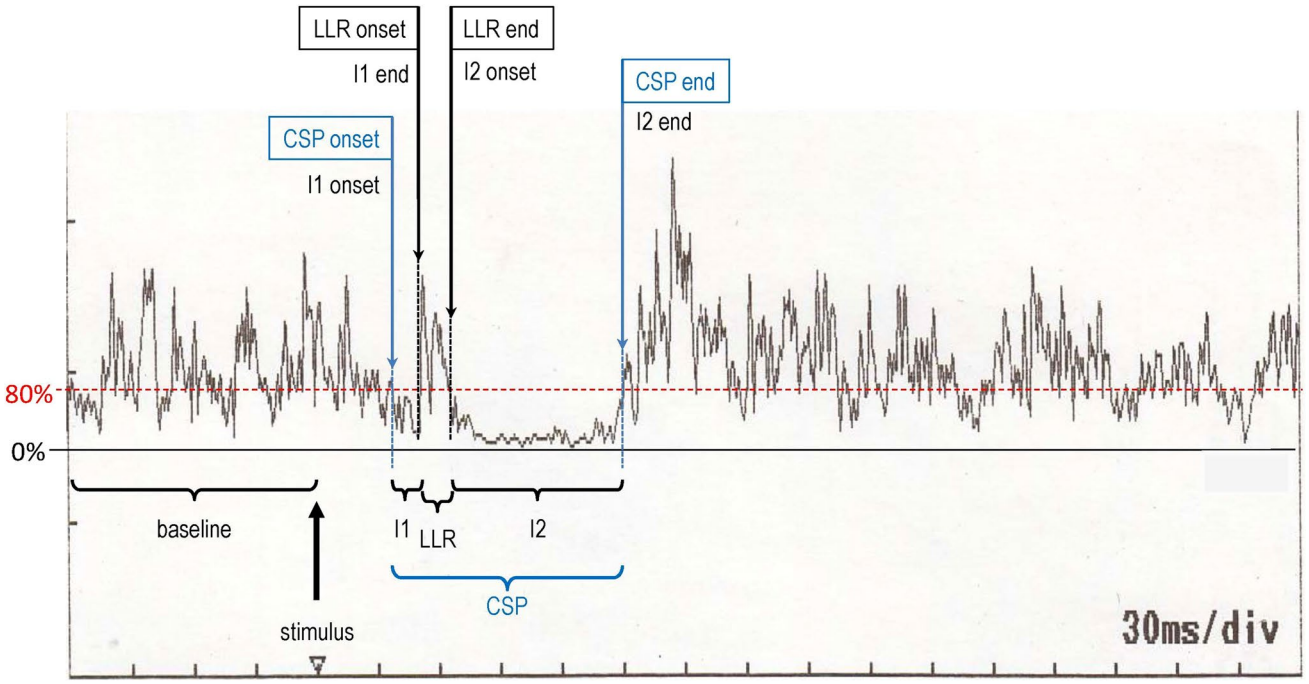

Fig. 1 An illustration showing a representative CSP. *ST* sensory threshold, *LLR* long-loop reflex, *I* inhibitory, *CSP* cutaneous silent period, *1 division* 200 microvolt)

- Mean baseline EMG amplitude during a 120 ms period preceding the stimulus;
- Onset latency, end latency, duration, and suppression index of the entire CSP,
- Duration and suppression index of I1; I1 onset latency being equal to CSP onset latency, and I1 end latency being equal to LLR onest latency;
- Onset latency, end latency, duration, and area of LLR, as well as absolute LLR amplitude and amplitude relative to mean baseline EMG amplitude;
- Duration and suppression index of I2; I2 onset latency being equal to LLR end latency, and I2 end latency being equal to CSP end latency.

To determine EMG suppression periods, all recordings were rectified and averaged. The onset latency of CSP (hence also of I1) was described as the first point in time when the EMG activity dropped below 80% of mean baseline EMG amplitude, whereas the CSP end latency (hence also I2 end latency) was the time when the amplitude returned above the 80% value (Kimura 2006; Kofler 2004). CSP duration was the time period between CSP onset and end latencies. Onset and end latencies of the transcortical long-loop reflex (hence end latency of I1 and onset latency of I2, respectively) were assessed by measuring onset and end latencies of EMG activity interrupting the early part of EMG suppression (Fig. 1) (Kofler 2004).

CSP suppression index was calculated as follows: mean EMG amplitude during the CSP divided by mean baseline EMG amplitude times 100. I1 and I2 suppression indices were calculated correspondingly by dividing either mean EMG amplitude during I1 or during I2 by mean baseline EMG amplitude times 100, respectively.

Statistical analysis

All parameters were compared between the three time points (before, during and after vibration). Data analyses were performed using the SPSS 20 software statistical package (SPSS Inc., Chicago, IL, USA). At first, normal distribution of data was analyzed using Shapiro–Wilk test. Comparisons were made using Friedman's test when distribution of data was non-homogenous. One-way repeated measures ANOVA was used when distribution of data was homogenous. Post-hoc comparisons were done using Wilcoxon signed-rank test or Bonferroni test depending on the normality of data. A p value < 0.05 was considered significant.

Results

Sensory threshold and stimulation intensity (mean ± standard deviation) were 2.0 ± 0.4 mA and 31.4 ± 5.0 mA, respectively. Prestimulus baseline amplitude did not differ significantly among the three conditions (Table 1).

Onset latencies of CSP (hence also of I1) and of I2 were not significantly different among the three conditions ($p = 0.797$ and $p = 0.161$ respectively).

End latency of I1 was not significantly different among the three conditions either ($p = 0.508$). However, there was a trend for shorter CSP end latency (hence also for shorter I2 end latency) during and after vibration compared to baseline ($p = 0.056$). Post-hoc comparisons revealed a significantly shorter CSP (hence I2) end latency during vibration compared to baseline recordings ($p = 0.048$).

Durations of entire CSP and of I1 were not significantly different among the three conditions ($p = 0.566$). I2 duration, however, was significantly shorter during vibration vs baseline ($p = 0.019$).

The suppression index of the entire CSP was higher during both vibration and post-vibration sessions compared to baseline, indicating less powerful EMG suppression ($p = 0.000$, Table). Likewise, I1 and I2 suppression indices were higher during both vibration and post-vibration sessions compared to baseline, indicating less suppression ($p < 0.001$ each, Fig. 2).

None of the LLR parameters (onset and end latencies, duration, area, as well as absolute LLR amplitude and amplitude relative to mean baseline EMG amplitude) differed among conditions.

Discussion

The major finding of this study was a significant and marked reduction in exteroceptive EMG suppression during and immediately following a 2-min period of vibration to the forearm. Suppression indices were significantly higher for each inhibitory phase, I1 and I2, and for the entire CSP during and after vibration. I2 duration and I2 end latency were mildly, but significantly shorter during vibration as compared to baseline. There was no significant effect on LLR amplitudes.

Various parameters have been measured on a given CSP. Different physiological conditions such as age and temperature or different recording settings may affect the CSP parameters. In the present study, all these conditions remained constant, and subjects served as their own controls.

Although vibration is not selective and may also activate mechanoreceptors other than primary spindle endings

Table 1 CSP parameters during each condition

	Pre-vibration	Vibration	Post-vibration	*P*
Baseline amplitude (mV)	0.4 ± 0.2	0.4 ± 0.2	0.5 ± 0.4	0.296
CSP onset latency (ms)	41.2 ± 8.7	40.2 ± 8.9	38.8 ± 6.7	0.797
CSP end latency (ms)	122.9 ± 10.3	116.4 ± 10.8	116.9 ± 9.7	0.056[a]
CSP duration (ms)	81.6 ± 13.5	76.2 ± 12.6	78.2 ± 11.1	0.508
I1 duration (ms)	11.9 ± 4.2	14.0 ± 5.6	14.7 ± 6.3	0.566
I2 duration (ms)	52.8 ± 14.3	42.8 ± 12.8	45.8 ± 12.8	0.018[b]
LLR onset latency (ms)	53.2 ± 9.6	54.2 ± 8.6	53.5 ± 8.1	0.840
LLR end latency (ms)	70.1 ± 11.5	73.2 ± 6.9	71.2 ± 7.7	0.161
LLR duration (ms)	16.9 ± 7.1	19.4 ± 7.6	17.7 ± 6.6	0.606
LLR area (mVms)	1.197 ± 1.0	1.833 ± 1.531	1.661 ± 1.015	0.257
LLR rectified amplitude (mV)	0.298 ± 0.153	0.403 ± 0.252	0.416 ± 0.362	0.319
Relative LLR amplitude (%)	87.4 ± 41.5	97.1 ± 51.8	101.8 ± 50.3	0.526
SI-CSP	17.1 ± 9.7	43.2 ± 8.9	44.2 ± 7.2	0.000*
SI-I1	23.9 ± 21.6	55.8 ± 20.8	54.9 ± 27.2	0.001**
SI-I2	14.6 ± 10.7	50.2 ± 15.2	49.9 ± 18.1	0.000***

CSP cutaneous silent period, *LLR* long-loop reflex, *I* inhibitory, *SI* suppression index

[a]Trend for significance (repeated measures one-way ANOVA). Post-hoc Bonferroni test: baseline vs vibration $p = 0.048$; baseline vs postvibration $p = 1.0$

[b]$p < 0.05$ (repeated measures one-way ANOVA). Post-hoc Bonferroni test: baseline vs vibration $p = 0.019$; baseline vs postvibration $p = 0.308$

*$p < 0.005$ (repeated measures one-way ANOVA). Post-hoc Bonferroni test: baseline vs vibration $p = 0.000$; baseline vs postvibration $p = 0.000$

**$p < 0.005$ (Friedman test). Post-hoc Wilcoxon signed-rank test: baseline vs vibration $p = 0.004$; baseline vs postvibration $p = 0.001$

***$p < 0.005$ (repeated measures one-way ANOVA). Post-hoc Bonferroni test: baseline vs vibration $p = 0.000$; baseline vs postvibration $p = 0.000$

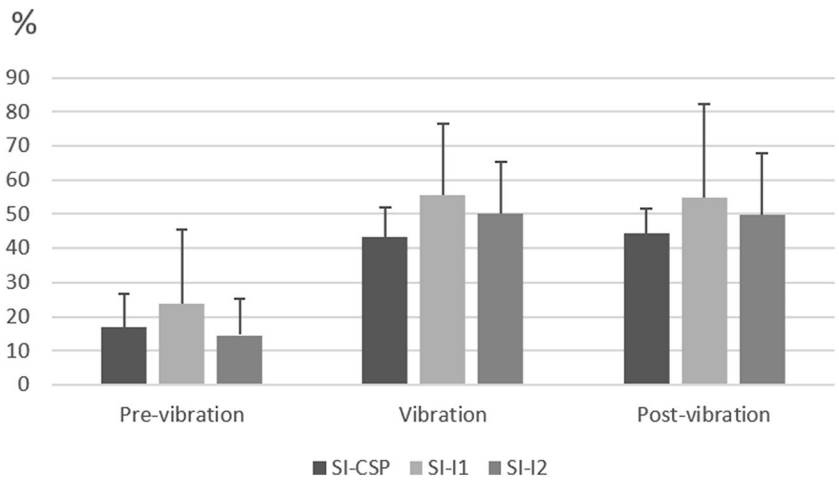

Fig. 2 Graphic presentation of suppression indices of entire CSP, I1 and I2. *SI* suppression index, *I* inhibitory, *CSP* cutaneous silent period, % suppression index

(Pierrot-Deseilligny and Burke 2012), when applied to a relevant tendon, it was shown to suppress H-reflex, despite an increase in the amplitude of M response (Gillies et al. 1969). Therefore, the authors concluded the effect of vibration was on the afferent volley rather than on large-diameter efferent motor fibers. Suppression of H-reflex excitability following vibration was largely attributed to hyperpolarization of group Ia fibers (Fetz et al. 1979). Vibration at low amplitude, as used in the present study, does not influence muscle contraction (Claus et al. 1988) or EMG amplitude (Mosier et al. 2017). Vibration superimposed on low-level isometric contraction may alter the motor unit recruitment strategy, activating larger and faster motor units (Xu et al. 2018) with high firing frequency (Griffin et al. 2001) which could exert a confounding effect on CSP. However, we compared prestimulus baseline

amplitudes and found no difference among conditions. Thus, we can exclude a significant effect of vibration on ongoing prestimulus EMG activity in the present study. Although a short-lived enhancement of motor evoked potentials (MEPs) occurs after muscle vibration, an absolute long-lasting enhancement of alpha motoneurons develops after 120 ms of vibration (Claus et al. 1988). Furthermore, relatively long-lasting (15 min vs 2 min in the present study) low amplitude muscle vibration increases MEP amplitudes in the vibrated muscle while suppressing MEPs in non-vibrated muscles of the same hand (Rosenkranz and Rothwell 2003).

Several lines of evidence suggest an essential role of A-delta-fibers in the generation of CSP. In the upper limbs, the nociceptive stimulus is conveyed to the spinal cord subsequently inhibiting motor nuclei of the intrinsic hand muscles (Leis 1998). Exteroceptive EMG suppression may occur at the level of corticospinal tract neurons, inhibitory interneurons, spinal motoneurons, or a combination of all. To delineate this issue, Leis et al. (1995) studied H-reflexes and F-waves during CSPs and found that spinal motoneurons remained excitable throughout the CSP, suggesting a prominent role of presynaptic inhibition in the generation of CSPs. In contrast, Inghilleri et al. (1997) reported suppressed F-waves during the time period corresponding to the CSP, suggesting postsynaptic inhibition. Manconi et al. (1998) demonstrated a similar temporal change of H-reflexes and MEPs during the CSP, favoring postsynaptic inhibition of motoneurons, whereas Priori et al. (1998), who also assessed MEPs and H-reflexes during the CSP, provided experimental evidence of dissimilar behavior of H reflexes and MEPs, concluding that there was no evidence of postsynaptic inhibition.

We found no change in CSP onset latency between the sessions, suggesting that normal conduction of afferent A-delta fibers is not affected by vibration. Corticospinal projections also modulate CSPs, as upper motor neuron lesions were shown to delay CSP onset (Gilio et al. 2008). Although vibration may affect corticospinal output (Rosenkranz and Rothwell 2003), no change in CSP onset latency may serve to exclude a significant effect of vibration on corticospinal output in the present study. Furthermore, Rosenkranz and Rothwell (2003) applied much longer durations of vibration as compared to the present study. A larger suppression index, hence less suppression, with shorter CSP duration, suggests either reduced inhibition or increased excitation during vibration. Thus, reduced EMG suppression may mean less inhibition of active motoneurons, especially no inhibition in a number of high-frequency motoneurons, during CSP in the presence of vibration. Or, we may speculate, in the presence of vibration, the effects of the inhibitory input is interrupted earlier due to faster reactivation of motor fibers. However, the second hypothesis is less likely, because theoretically a CSP can be generated by synchronously "knocking out" only one single discharge per motoneuron.

Concerning possible causes of inhibition of reduced number of motoneurons during vibration, we may claim presynaptic inhibition of pain afferents, reduced activity of inhibitory interneurons, increased activity of excitatory interneurons, reduced recurrent inhibition or earlier resynchronization of motoneurons due to vibration.

Although vibration does not exert any major effects on nociceptive pathways either (Pazzaglia et al. 2017), an effect of vibration on pain has been documented (Hollins et al. 2014; Weerakkody et al. 2003). According to the gate control theory, large-diameter afferents close the gate of pain transmission whereas small-diameter afferents open it (Melzack and Wall 1965). In the presence of vibration, the domination of large-diameter afferents may hinder the transmission of A-delta fibers to the spinal cord segments. However, there are several arguments about this hypothesis in our case. First of all, vibration largely ameliorates pressure-induced pain and it does not affect pain induced by stimulation of small-diameter afferents (Pazzaglia et al. 2017; Weerakkody et al. 2003). Some studies using a similar vibration frequency did not show any effect on pain (Park et al. 2014). Second, in our study, there was no evidence of altered conduction in A-delta fibers (e.g. CSP onset latency). The crucial role of A-delta fibers in CSP generation has recently been confirmed in various forms of polyneuropathy, revealing that a reduction of A-delta input delays CSP onset (Lopergolo et al. 2015). A similar study using 15 min of high-frequency TENS led to shortened CSP duration whereas the amount of exteroceptive suppression based on the index of suppression was slightly increased (Kofler 2004). In that study, index of suppression was merely changed because of the suppression in LLR amplitude, whereas in the present study, LLR did not change, but the "purely" inhibitory phase I2 was slightly shortened and its suppression index profoundly increased. The effect of TENS on the CSP was attributed to TENS-associated presynaptic inhibition of nociceptive A-delta fibers at the spinal segmental level. Last, in a previous study of nociceptive flexor reflex and vibration, segmental vibration facilitated the response on biceps femoris muscle (Ertekin and Akçali 1978). Then, is it possible for muscle vibration, as in our study, to facilitate a nociceptive flexor response on APB during CSP? There are flexor responses on thenar muscles after painful stimuli to upper extremities with latencies corresponding to CSP (Syrovegin et al. 2000). Then, one explanation for reduced exteroceptive suppression during vibration may be facilitation of a response similar to flexor reflex on thenar muscles. However, we cannot rule out different mechanisms of pain modulation in cervical and lumbar segments.

Traditionally, each interneuron is devoted to a dominant group of sensory input. However, each individual

interneuron has been suggested to receive a broad convergence from a large variety of sensory modalities, as well as inputs from one or more descending tracts (Hultborn 2001). Hence, inhibitory interneurons may receive inputs from group Ia fibers as well as from nociceptors. After hyperpolarization of group Ia fibers, due to "busy-line theory", the activity of inhibitory interneurons may decrease leading to less exteroceptive suppression throughout the CSP (Pierrot-Deseilligny and Burke 2012). However, the CSP circuit is probably oligosynaptic, as paired stimuli with interstimulus intervals between 100 and 500 ms failed to show significant CSP habituation (Inghilleri et al. 1997), thus rendering it unlikely that a large number of interneurons might be involved in our findings.

Uncini and colleagues (1991) suggested motor neuron inhibition from nociceptive stimulation may be mediated by Renshaw cells directly activated by high-threshold cutaneous afferents (Uncini et al. 1991). When an individual Renshaw cell is activated, motoneuron innervating agonist muscle as well as inhibitory interneuron receiving group Ia innervation are inhibited (Pierrot-Deseilligny and Burke 2012). Renshaw cells get excitatory and inhibitory inputs from ipsilateral cutaneous afferents, group II and III muscle afferent fibers (Pierrot-Deseilligny and Burke 2012). Group II afferents inhibit Renshaw cells (Pierrot-Deseilligny and Burke 2012). Thus, inhibition of Renshaw cells by group II activation during vibration may in theory cause less exteroceptive suppression. However, the role of Renshaw cells in CSP generation remains to be elucidated, as motoneurons innervating distal muscles were shown to lack Renshaw cell inhibition (Illert and Wietelmann 1989; Illert and Kümmel 1999; Katz et al. 1993).

Post-CSP EMG rebound activity is mainly secondary to resynchronization of motoneurons. There is also sufficient evidence that post-CSP EMG rebound activity may contain an excitatory reflex component, which, based on the latency and the nature of the underlying stimulus, is compatible with a somatosensory startle reflex (Kumru et al. 2009). Vibration itself is a potent stimulator of startle response in animals (Friedel 1999). However, stimuli evoking a startle response should be sudden and unexpected. Instead, we used continuous stimulus in this study and we did not see any change in the prestimulus EMG activity. Therefore, an earlier startle-related post-CSP response due to vibration seems unlikely. Additionally, continuous vibration inhibits the auditory startle response in humans suggesting that there should rather be longer CSPs in the presence of vibration (Hill and Blumenthal 2005). In our opinion, the contribution of post-EMG startle response is minor in reduced suppression or shorter CSP duration in the present study.

On a neuronal level, pharmacological studies have attempted to clarify underlying physiological mechanisms. To date, only monaminergic substances, such as levodopa, pramipexole, tramadol, and escitalopram have been shown to influence CSP parameters in healthy subjects or in patients with various neurological disorders (Pujia et al. 2012, 2014; Serrao et al. 2002). In idiopathic Parkinson's disease, CSP duration was shortened after levodopa intake (Serrao et al. 2002), while in patients with multiple system atrophy, who had no clinical benefit from levodopa, also CSPs did not change (Stetkarova et al. 2015). In addition, the antihistaminergic drug cetirizine had no influence on CSP parameters (Kofler et al. 2009). Activity in dopaminergic pathways or presynaptic inhibition are other likely causes. However, the effect of vibration on CSP duration occurs during its application and is short-lived. Absence of any modulation of LLRs by vibration also excludes the possible role of levodopa. Thus, suprasegmental control or effect of levodopa is unlikely because levodopa is known to change gain of LLR (Johnson et al. 1994).

The most important limitation of our study is the lack of analyzing additional electrophysiological parameters showing motoneuron activation such as compound muscle action potential. Furthermore, it is technically virtually impossible to limit vibration with the apparatus and stimulation parameters used to one tendon only. Therefore, although we aimed at stimulating only the tendon of flexor carpi radialis muscle close to the wrist, it is likely that also other tendons, and even more proximally located muscles, were mildly affected by vibration.

Conclusion

Exteroceptive EMG suppression in APB is markedly reduced, and I2 duration is mildly shortened, by forearm muscle vibration, which is known to be a powerful stimulus for Ia afferent fibers. Thus, based on the present data, we propose that Ia afferent input is capable of exerting powerful inhibition on inhibitory interneurons in the CSP pathway. We cannot, however, exclude effects due to activation of other mechanoreceptors, nor can we differentiate between possible presynaptic inhibition of A-delta afferents and pre- or postsynaptic inhibitory effects on ensuing interneurons. Based on the literature, there may even be a contribution to increased EMG activity from concomitant excitatory influence as shown by vibration-related enhancement of MEPs, while aninfluence on Renshaw cells seems rather unlikely.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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