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SHORT COMMUNICATION

Monopolar surface electromyography: a better tool to assess motoneuron excitability upon passive muscle stretching

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Abstract Bipolar and monopolar surface electromyography (sEMG) are known procedures to measure the H-reflex. However, signal cancellation is a potential experimental problem of bipolar sEMG. The results of our study show that monopolar sEMG was the more sensitive procedure to differentiate motoneuron excitability at different passive muscle stretching speeds as it overcame signal cancellation.

Keywords Electromyography · Motoneuron excitability · Muscle stretching

H-reflex serves as a reliable tool to assess the excitability of spinal alpha motoneurons (α-MNs) through the monosynaptic reflex pathway [1–4]. The monosynaptic reflex pathway consists of the excitatory synapses between the Ia afferents of the muscle spindle and the spinal α-MNs of the corresponding homonymous muscle [2, 3].

Passive muscle stretching has been reported to inhibit the corresponding α-MNs in terms of the depression of H-reflex parameters [5–9]. However, little consideration has been given to the speed of the passive muscle stretching and to the surface electromyography (sEMG) methods used. Two sEMG methods (bipolar sEMG and monopolar sEMG) are standardly used to measure the H-reflex of the human muscle [10]. In addition, signal cancellation, where some part of the real signal amplitude is lost [11–13], is a potential problem of the bipolar sEMG that was used [5–9] in the measurement of H-reflex.

The primary aim of the study reported here was to examine and compare the effect of different sEMG methods (bipolar sEMG vs. monopolar sEMG) on detecting the modulation of α-MN excitability of human soleus muscle upon passive muscle stretching. The secondary aim was to gain a better understanding of the relationship between the speed of passive muscle stretching and the excitability of α-MNs.

Methods

Twelve healthy volunteers (one female and 11 males; age range 20–22 years) were tested. The subjects had no prior history of neuromuscular disorders, had a normal range of ankle motion (>10° of dorsiflexion), and gave their written informed consent to participate in this study, which was approved by the ethics committees of Hiroshima University and Hiroshima University hospital. The subjects assumed a comfortable prone position on the testing table, with the right knee supported and semi-flexed (10°). The right foot was placed and secured by a strap on the foot plate of a Biodex system3 dynamometer (Biodex Medical Systems, Shirley, NY) with the ankle joint at an angle of 90°. This position was then saved in a Biodex system3 computer as the starting position (0°). The untested leg of each subject was supported on a limb support. Subjects were asked to relax for the experiment, which lasted approximately 3 h. To start with, we measured the maximal passive ankle dorsiflexion for each subject by flexing the subject's ankle passively moving the Biodex system3

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dynamometer from the starting position up to the maximal comfortable and pain-free dorsiflexion. Thereafter, the angle was recorded (16.64 ± 1.5°) and saved as the ending position.

The H-reflex and the M-wave parameters of bipolar and monopolar sEMG were recorded at four conditions—the resting condition (relaxed soleus muscle with an ankle joint angle of 90°) and three other conditions—while the soleus muscle was at its maximal stretched length (pain-free maximal passive ankle dorsiflexion). The latter was achieved by using three passive stretching speeds of 2, 5 and 10° s^{-1}. There were two trials for each stretching maneuver at each testing condition. Subjects were given 5 min of resting time after each test condition or stretching maneuver trial, and 30 min of resting time after four tests were completed.

In the previous studies, the timing of the electrical stimulus employed to elicit the H-reflex and the M-wave was at the maximum tolerated dorsiflexion [5] and at ankle dorsiflexions of 0, 10 and 20° [8, 9], respectively. In this study, a single square wave pulse of 0.5 ms was applied to the tibial nerve at rest (0°), and the dorsiflexion was the maximum that could be tolerated by the subjects (16.64 ± 1.5°). The stimulus intensity was increased gradually, with a random inter-stimulus interval of at least 10 s, from an intensity below the H-wave threshold to the stable M-wave amplitude. Electrical stimulation was delivered using the bipolar stimulator of a Viking Quest portable EMG system (Viking Quest system with software V7.4; Nicolet Biomedical, Madison, WI). All sEMG signals were amplified (bandwidth 20–3 KHz), digitized (6 KHz) and recorded using the Viking Quest portable EMG system. The arrangement of the electrodes is illustrated in Fig. 1, and a circular disposable self-adhesive sEMG electrode with a diameter of 20 mm (VIASYS healthcare, Neurocare group, Conshohocken, PA) was used.

To quantify the corresponding α-MNs excitability, several H-reflex and M-wave parameters were measured, including maximal H-wave (Hmax), maximal M-wave (Mmax) and the Hmax/Mmax ratio. The peak-to-peak amplitudes of all responses were recorded and plotted as a percentage of Mmax amplitude to obtain a standardized H–M recruitment curve. To confirm the reliability and the consistency of our data, we measured the H-wave with a stimulus intensity that corresponded to 50% of the Mmax amplitude of the resting condition [14]. At this intensity, 14 H-wave responses, peak-to-peak, at each testing condition were recorded and averaged for five subjects. The recording was performed by three sEMG methods: the bipolar sEMG (G1-BG2) [10], the ordinary monopolar sEMG (G1-MG2) [10, 13, 15] and a proposed second monopolar sEMG method (BG2-MG2) (Fig. 1). The statistical

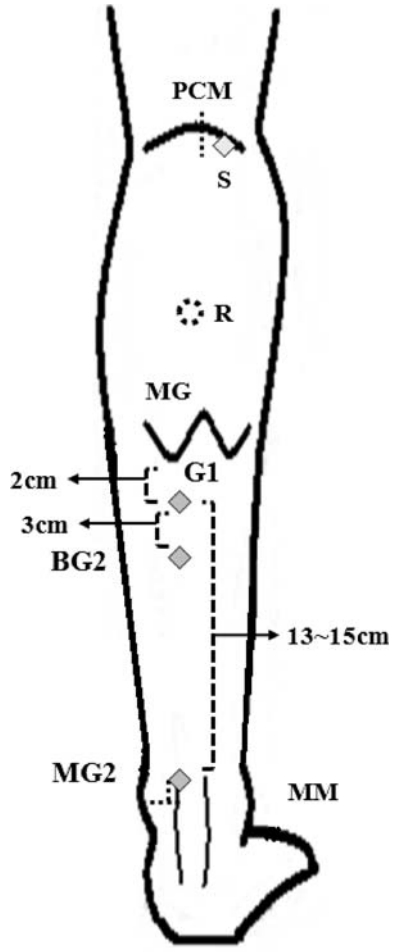

Fig. 1 Arrangement of surface electromyography (sEMG) electrodes. The G1 electrode position is 2 cm distal to the insertion of the medial gastrocnemius muscle (*MG*) into the Achilles tendon and is used for both sEMG methods (bipolar and monopolar). In the bipolar sEMG, the G2 electrode (*BG2*) is placed 3 cm distal to G1. In the monopolar sEMG, the G2 electrode (*MG2*) is placed 3 cm medially and 2 cm superiorly to medial malleolus (*MM*) and over the Achilles tendon. The reference electrode (*R*) of both methods is placed anterior over the tibial bone at a distance between the stimulus site and G1 electrode (*dashed circle*).The stimulating site (*S*) is approximately 1 cm lateral to the popliteal crease mid-point (*PCM*)

significance was determined using a repeated measures analysis of variance (ANOVA) followed by a post hoc analysis (Dunnett test). The significance was accepted at $P < 0.05$.

Results

Compared with those in the resting condition, the means of the Hmax amplitude and Hmax/Mmax ratio in the bipolar sEMG decreased significantly at all passive stretching speeds (2, 5 and 10° s^{-1}) (Figs. 2a, 3a). In contrast, in the ordinary monopolar sEMG (G1-MG2), the means of the

Hmax amplitude and Hmax/Mmax ratio decreased significantly only at a passive stretching speed of 2° s^{-1}, and the passive stretching speeds of 5 and 10° s^{-1} did not show any significant differences (Figs. 2b, 3b). The means of the Mmax amplitude did not show any significant differences ($P > 0.05$) within each sEMG method (Fig. 2).

Again, with the stimulus intensity fixed at 50% of Mmax, the means of the H-wave amplitude in the bipolar sEMG had decreased significantly at all stretching speeds when compared with the resting condition. Despite the difference in the magnitude, the mean of the H-wave also decreased significantly at a passive stretching speed of 2° s^{-1} in a similar manner in both the monopolar sEMG (G1-MG2, and BG2-MG2), and there were no significant differences at stretching speed of 5 and 10° s^{-1}. In addition, there were no significant differences in the recorded amplitudes between the bipolar sEMG (G1-BG2) and the suggested monopolar sEMG (BG2-MG2); nor were there any significant differences between the suggested monopolar sEMG (BG2-MG2) and the ordinary monopolar sEMG (G1-MG2) at all testing conditions. There were also no significant differences in the recorded amplitudes between the bipolar sEMG (G1-BG2) and the ordinary monopolar sEMG (G1-MG2) at resting and passive stretching speed of 2° s^{-1} conditions, whereas, significant differences were detected at passive stretching speed of 5 and 10° s^{-1} (Fig. 4).

There were no significant differences ($P > 0.05$) in the signal latency between the sEMG methods and the testing conditions.

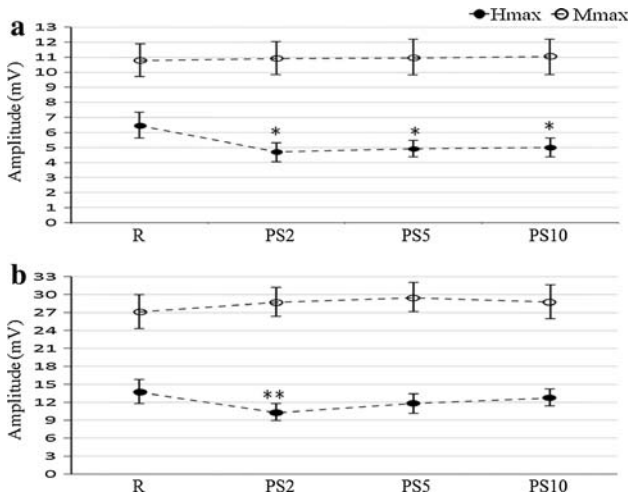

Fig. 2 Absolute maximal H-wave (*Hmax*) and maximal M-wave (*Mmax*) values. Mean ± standard error (SE, $n = 12$) of the bipolar sEMG (**a**) and ordinary monopolar sEMG (**b**) at a resting (R) condition and passive stretching (PS) speed of 2, 5 and 10° s^{-1} (*R*, *PS2*, *PS5*, and *PS10*, respectively). $*P < 0.001$, $**P = 0.011$

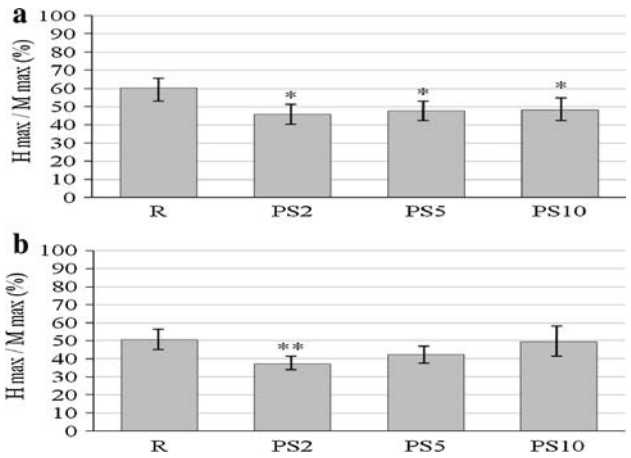

Fig. 3 Hmax/Mmax ratio. Mean ± SE ($n = 12$) at the bipolar sEMG (**a**) and the ordinary monopolar sEMG (**b**). Abbreviations are the same as in Fig. 2. $*P < 0.001$, $**P = 0.028$

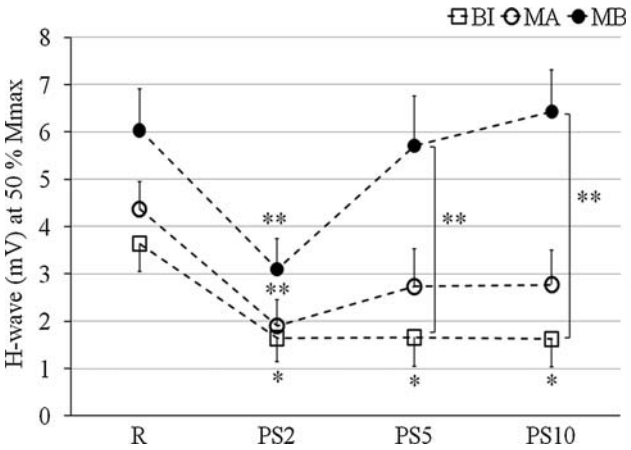

Fig. 4 H-wave at 50% of Mmax (mean ± SE; $n = 5$) at bipolar sEMG (G1-BG2, *BI*), suggested monopolar sEMG (BG2-MG2, *MA*) and the ordinary monopolar sEMG (G1-MG2, *MB*). Abbreviations are the same as in Fig. 2. $*P = 0.002$, $**P < 0.05$

Discussion

In the bipolar sEMG, the corresponding α-MNs of the soleus muscle were inhibited significantly at all passive stretching speeds (2, 5 and 10° s^{-1}). Given that the potential problem of the bipolar sEMG is that of signal cancellation, where some part of the real signal amplitude is lost as a result of a temporal summation and overlapping of the recorded amplitude of two opposite polarities. Such overlapping occurs when an action potential starts to be recorded from the G2 electrode before it has been completely passed and recorded by the G1 electrode [11–13]. Therefore, our primary consideration was that these inhibitions of α-MNs with respect to passive stretching speeds were not due to purely physiological mechanisms, as we

suspected that the results were influenced or contaminated by the signal cancellation (an experimental attribute).

In the monopolar sEMG, the corresponding α-MNs of the soleus muscle were significantly inhibited only at a passive stretching speed of 2° s^{-1}. This observation provides an important clarification for any meaningful comparison of the results of the monopolar and bipolar sEMG methods. The underlying principle of the monopolar sEMG system is to place the G2 electrode over the tendon of the tested muscle, which is unexcitable tissue [10, 13, 15]. The hypothesis is, therefore, that signal cancellation is avoided at the monopolar sEMG. This placement of the electrodes may introduce far-field potentials [16] that may be involved in the amplitude recorded by the monopolar system. However, it is assumed that the monopolar sEMG in this study has the same technical principle of "near-field type montage" [17]. Of particular importance in this system is the appearance of positive and negative wave forms (far-field potentials) with constant latency and at location coincident with and beyond the propagating action potential [18]. Therefore, it is hypothesized that the respective dipolar far-field components cancel each other out, yielding only near-field potential [19]. Support for this hypothesis can be seen in Fig. 4, where the results indicate that the far-field potential has no significant effect on the recorded potential amplitude of the monopolar sEMG methods at all testing conditions and that those significant differences between the ordinary monopolar sEMG and bipolar sEMG at passive stretching speeds of 5 and 10° s^{-1} were due to the large degree of signal cancellation that occurs due to nature of the bipolar recording electrodes—and not to the far-field potential amplitude added to the monopolar evoked potential amplitudes, since there were no significant differences at resting and a passive stretching speed of 2° s^{-1}. As a result, we suggest that the monopolar sEMG results were due to physiological factors rather than to experimental ones.

Another point worth noting is that assessing α-MN excitability indirectly, as the mean of H-reflex, using the sEMG recording is not an approach that will enable the existence of different α-MN excitability information from the different monopolar sEMG methods to be denied or confirmed, or that will determine if the G1 and BG2 electrodes are placed on the same muscle fibre. However, the electrode configuration in this study is the same as that reported elsewhere [10, 13, 15]. In addition, in terms of the exist data, there were no significant differences between the G1-MG2 and BG2-MG2 (Fig. 4), which suggests that the G1 and BG2 are probably located at the same distance from the generator source.

We have limited ourselves to the identification of a definite physiological mechanism in terms of the ordinary monopolar sEMG (G1-MG2) results. However, to exclude the role of presynaptic inhibition of the soleus muscle, we applied the electrical stimulation to the tibial nerve only, with no conditioning volleys on any other nerve; the subjects' hips and knees were kept stationary and relaxed, and the soleus muscle was relaxed during the experiments, with no active contraction [20, 21]. Also, to avoid the post-activation depression or homosynaptic depression, the electrical stimulation was applied with a random inter-stimulus interval of at least 10 s [22, 23]. Furthermore, to ensure that the changes in the H-reflex can be assumed to be a result of alterations in the corresponding α-MN excitability rather than changes in the spatial relationship between the nerve and stimulating electrode or changes in the angle of the ankle [9, 24], the Mmax amplitudes remained stable during all testing conditions (as shown in Fig. 2).

Peripherally, Ib inhibitory afferents of Golgi tendon organs were first considered to be responsible for reflex inhibition. However, it has been reported that when the reflex is recorded at rest and with no voluntary muscle contraction, the role of Ia–Ib inhibitory interneurons can be considered to be at a minimal and less active [25]. Furthermore, during ankle dorsiflexion, neither joint receptors nor cutaneous mechanoreceptors contribute significantly to the decrease in reflex excitability by comparing reflex excitability before and after skin anaesthesia [5, 26].

One possible explanation is changes in the synchronization pattern of the motor units. This becomes particularly important when the motor units are being voluntary activated because of the nature of their un-synchronized firing [27]. In addition, in this study, there were no significant differences in the onset latency and the inter-peak time interval of the monopolar signal at all testing conditions. Therefore, we hypothesize that both monopolar sEMGs had the same synchronization pattern. However, we cannot exclude the possibility of changes in the synchronization pattern of the motor units because the surface electrode detects only the temporal sum of action potential trains [12].

An alternative possible explanation is shown in a cat muscle experiment [28], in which the discharge rate of Ia afferents declines as the muscle is stretched slowly (1.25 mm s^{-1}), and it increases at a more rapid stretching speed of 10 mm s^{-1}. These results are explained later on as the intrafusal muscle fibre capsule enhancing the direct impact of stretching force on Ia afferents if the former is stretched directly by the surrounding muscle tissue [29]. Also, increased compliance of the extrafusal or intrafusal muscle fibres may decrease the resting discharge of the Ia afferents entering the motoneuronal pool in lengthened position [6].

In conclusion, the detection of changes in α-MN excitability upon passive muscle stretching demonstrates that a

great attention should be paid to the sEMG method and the speed of stretching. We found that the monopolar sEMG was the more sensitive method to differentiate MN excitability at different passive muscle stretching speeds.

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