Relationships among Electromyogram, Displacement and Velocity of the Center of Pressure, and Muscle Stiffness of the Medial Gastrocnemius Muscle during Quiet Standing

Takanori UCHIYAMA,* # Gai KONDO**

Abstract Medial gastrocnemius muscle stiffness was estimated using a system identification technique. The medial gastrocnemius muscle was electrically stimulated using surface Ag-AgCl electrodes and the center of pressure fluctuation in the forward-backward direction was measured with a force plate. Electrically induced fluctuation of the center of pressure was classified according to the displacement and velocity of the center of pressure. The classified steps of displacement and velocity were 0.2 cm and 0.2 cm/s, respectively. The ranges of the classes were ±0.15 cm and ±0.15 cm/s. The classified fluctuations were synchronously averaged and the averaged fluctuation was regarded as an output signal of the transfer function from the electrical stimulation to the fluctuation. The transfer function was identified as an estimate of muscle stiffness using a singular value decomposition method. The average muscle stiffness of eight young male participants ranged from 56.7 to 75.9 N/m. Muscle stiffness was high when the displacement of the center of pressure was positive and the velocity was negative. These characteristics resembled the preceding 0.3 s of the electromyogram. This preceding high muscle activity probably contributed to muscle stiffness. Muscle stiffness was well approximated with a multiple linear regression plane, in which the explanatory variables were the displacement and velocity of the center of pressure.

Keywords: system identification, EMG, COP, lower leg.

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1. Introduction

Standing posture can be considered to be an inverted pendulum stabilized by contraction of the leg muscles. Muscle contraction is controlled by the central nervous system that also controls muscle stiffness. The central nervous system transmits signals to the muscles, causing muscle activation. The activated muscle then generates contractile force and becomes stiff. The activation level of the muscle can be evaluated using electromyography (EMG).

EMG signals are related to fluctuation of the center of pressure. The amplitude of EMG is known to be proportional to the displacement of the center of pressure [1]. In addition, the EMG signal has been found to precede the displacement of the center of pressure by 0.15–0.2 s [2, 3]. The velocity of the center of pressure is also important for maintaining the standing posture [2]. Standing posture can be regarded as an inverted pendulum stabilized by a proportional, integral and derivative (PID) feedback controller [4, 5] or a proportional and derivative (PD) feedback controller [2, 6]. Here, “D” corresponds to velocity, and is necessary to stabilize an inverted pendulum. In a previous study, a cross-correlation function between the smoothed rectified EMG signal and the velocity of the center of pressure exhibited a negative peak at −0.6 s (EMG preceding velocity) and a weak positive peak at 0.1 s [2]. EMG is related to both displacement and velocity of the center of pressure. The EMG signal is an index of the muscle activation level, measuring changes as the activated muscles become stiff. Therefore, we hypothesized that muscle stiffness is related to the displacement and velocity of the center of pressure. However, these relations have not been clarified. Currently, there is no method for estimating muscle stiffness.
stiffness during quiet standing, although ankle stiffness has been investigated by applying sudden movement to a plate on which a participant is standing [7–9].

We developed a technique for estimating muscle stiffness during quiet standing, using electrical stimulation and a force plate [10]. We applied this technique to various combinations of displacement and velocity of the center of pressure. The purpose of the current study was to examine muscle stiffness at various combinations of displacement and velocity of the center of pressure. Muscle stiffness and EMG in a displacement-velocity plane are important features of postural control analysis in a phase plane [11–13].

2. Methods

2.1 Measurements

We measured the center of pressure and EMG of eight young male volunteers aged 21–25 years. No participant had a history of neuromuscular disorders. The study was approved by our local ethics committee (No. 30–36; Faculty of Science and Technology, Keio University). All participants gave written informed consent, and all experimental protocols were in accordance with the Declaration of Helsinki.

Participants were instructed to stand with their feet aligned on a force plate (SS-FP40A0-SY*; Sports Sensing Co., LTD, Fukuoka, Japan), to cross their arms in front of their chest, and to look at a target at their eye height. Two experiments were conducted. In one experiment, electrically induced center of pressure fluctuations were recorded during quiet standing. In the other, the center of pressure and EMG were recorded.

2.1.1 Electrically induced center of pressure fluctuation

Electrically induced center of pressure fluctuation was measured to estimate muscle stiffness using a system identification technique. Electrical stimulation was applied to the medial gastrocnemius muscle every 10 s through disposable surface Ag-AgCl electrodes (F-150S; Nihon Kohden, Tokyo, Japan) using an electrical stimulator (SEN-3301; Nihon Kohden) and an isolator (SS-104J; Nihon Kohden). The electrodes were placed on the skin surface of the medial gastrocnemius muscle along the muscle fibers. The position of the electrodes was at L/3 from the popliteal fossa where L was the length from the popliteal fossa to the calcaneal tuber. The distance between the electrodes was 6 cm. The electrical stimulation was a mono-polar rectangular pulse of 500 µs width and an amplitude of approximately 20 mA. The center of pressure was measured for 300 s using a force plate and was sampled with a 16-bit analog-to-digital converter (cRIO-9215; National Instruments, Austin, TX, USA) at 2000 Hz. The measurements were repeated 10 times.

2.1.2 EMG

The electrodes were placed on the skin surface of the medial gastrocnemius muscle in the same position as for the electrical stimulation, but the distance between the electrodes was 4 cm. The EMG was amplified (× 500) and filtered (10–1000 Hz) with an amplifier (EMG-025; Harada Electronics Industry, Sapporo, Hokkaido, Japan). EMG signals of the soleus and the tibialis anterior muscles were also measured. EMG signals and center of pressure were recorded for 300 s at a sampling frequency of 2000 Hz.

2.2 Analysis

2.2.1 Electrically induced center of pressure fluctuation

The measured center of pressure in the forward and backward direction was smoothed using a moving averaging technique with a window width of 0.1 s. The smoothed center of pressure was differentiated to obtain its velocity. Both the smoothed displacement and velocity of the center of pressure were decimated at a sampling frequency of 200 Hz.

Electrical stimulation was applied to the medial gastrocnemius muscle every 10 s. Therefore, the displacement and velocity of the center of pressure for electrical stimulation were scattered, as shown in Fig. 1. Circles denote the displacement and the velocity of center of pressure in the forward-backward direction. The displacement was that at 0.05 s before the electrical stimulation and the velocity was the average velocity between 0.1 and 0 s before the electrical stimulation.

We classified the electrically induced center of pressure fluctuation according to the displacement and velocity of the center of pressure. The displacement was classified every 0.2 cm (from −10 to 10 cm) within 0.15 cm. There was an overlap of 0.1 cm between the adjacent displacement classes. The velocity was classified every 0.2 cm/s (from −10 to 10 cm/s) within 0.15 cm/s. There was an overlap of 0.1 cm/s between the adjacent velocity classes. The ranges of the displacement and velocity, ±10 cm and ±10 cm/s, were sufficiently large to involve the displacement and velocity of the center of pressure.

The classified center of pressure fluctuation was synchronously averaged to estimate muscle stiffness when the number of classified center of fluctuation values was greater than six. The synchronous averages of the classified electrically induced fluctuation are shown.

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*Resolution: Fx/Fy/Fz 0.305 N, Mz/My/Mx 0.0305 N/m; linearity error: less than 3% (full scale); hysteresis: less than 1% (full scale)
in Fig. 2. The black solid line denotes the synchronous average of the electrically induced fluctuation classified in the black solid line square (0.6 ± 0.15 cm and 0 ± 0.15 cm/s) in Fig. 1. The black solid line was flat at 0.6 cm of displacement before the electrical stimulation (0 s). Other examples of the classified fluctuation are shown with black dotted, red solid, and red dotted lines in Fig. 2. The black dotted line was flat at −0.6 cm of displacement before electrical stimulation. The red solid and dotted lines show the linear increase (0.6 cm/s) and decrease (−0.6 cm/s) before 0 s, respectively.

2.2.2 Stiffness estimation
Muscle stiffness was estimated using a system identification technique that was slightly modified from that used in our previous study [10]. The induced fluctuation in this duration showed a sharp peak, primarily caused by muscle contraction, although a small amplitude of the center of pressure fluctuation was caused by backward sway, and intrinsic fluctuation was also involved. We assumed that the backward sway and intrinsic fluctuation were negligible in this short duration.

The system was identified as a second-order model (spring-mass-damper model) using a singular value decomposition method. The spring-mass-damper model has an undamped natural angular frequency. The square of the undamped natural angular frequency is equal to $k/m$, where $m$ is mass and $k$ is spring constant (stiffness) of the spring-mass-damper model. Therefore, the muscle stiffness was calculated by multiplying the undamped natural angular frequency by the muscle weight. Each participant’s muscle weight was estimated from the percentage of the muscle weight and participant’s weight (average 63.7 kg). The percentage of muscle weight was calculated from the volume of the medial gastrocnemius muscle (285 cm$^3$) [14] and muscle density (1.03 g/cm$^3$) [15].

Multiple linear regression was used to explain the relationships between stiffness ($k$), displacement ($y$) and velocity ($v$) of the center of pressure using the following equation:

$$k = b_0 + b_1y + b_2v. \quad (1)$$

The regression equation was calculated from the classified data, which consisted of the average muscle stiffness of eight participants, the classified displacement and the velocity of the center of pressure.
2.2.3 EMG
The root mean square of EMG (rmsEMG) was calculated using a moving window with a width of 0.1 s. The rmsEMG was decimated at 200 Hz. The rmsEMG was also classified based on the displacement and velocity of the center of pressure in similar manner to the electrically induced center of pressure fluctuation. The rmsEMG at $t, t - 0.1, t - 0.2, \text{and } t - 0.3$ s were investigated, where $t$ is the time at which the displacement and velocity of the center of pressure were classified. The preceding rmsEMG signals were investigated because the center of pressure showed a strong correlation with EMG signals in the preceding 0.15–0.2 s [2, 3].

3. Results

3.1 Electrically induced center of pressure fluctuation
Figure 1 shows an example of electrically induced center of pressure fluctuation (displacement and velocity). The standard deviation of the displacement was 0.38 cm, and that of the velocity was 0.50 cm/s. Other participants exhibited relatively large standard deviations. The ranges of the standard deviation of displacement and velocity for the eight participants were 0.38–0.84 cm and 0.50–0.85 cm/s, respectively.

3.2 System identification
The first peak of the synchronously averaged induced fluctuation was well approximated using the second-order model, as shown in Fig. 3. Other induced fluctuations in all classes of all participants were also approximated well using the second-order model.

3.3 Stiffness
The induced center of pressure fluctuations were classified according to the displacement and velocity. The classified fluctuation was synchronously averaged, and the muscle stiffness was then estimated. The average muscle stiffness of eight participants is shown in Fig. 4. The top panel, a, shows the contour plot of the muscle stiffness. Stiffness ranged from 56.7 to 75.9 N/m, and increased as the displacement of the center of pressure increased. Stiffness also increased as the velocity decreased. These findings indicated that muscle stiffness was high when the displacement of the center of pressure was positive and the velocity was negative.

Muscle stiffness was explained well using the displacement and velocity of the center of pressure, as shown in Fig. 4b. The multiple linear regression coefficients, $b_0, b_1, \text{and } b_2$, in Eq. 1 were $65.1 \text{ N/m}, 4.53 \times 10^2 \text{ N/m}^2, \text{ and } -1.12 \times 10^3 \text{ Ns/m}^2$, respectively. The multiple coefficient of determination, $R^2$, was 0.925. The multiple linear regression coefficients should be interpreted...
with caution because the coefficients depend on the type of muscle, and the individual's physical constitution.

### 3.4 EMG

The rmsEMG data were classified according to the displacement and velocity of the center of pressure. The average data for the classified rmsEMG of eight participants are shown in Fig. 5. The top panels, a and b, show the rmsEMG at $t = -0.3$ s and $t = -0.2$ s, where $t$ is the time at which the center of pressure was classified. The rms-EMGs were large when the displacement of the center of pressure was positive and the velocity was negative. The bottom panels, c and d, show the rmsEMG at $t = -0.1$ s and $t = 0$ s. The rmsEMG was large at positive velocity.

The center of pressure in the positive displacement and negative velocity showed high muscle activity at $-0.3$ s, and muscle activity then decreased. In contrast, the center of pressure in positive displacement and positive velocity showed low muscle activity at $-0.3$ s but high activity at $-0.1$ and $0$ s.

EMG signals of the soleus and tibialis anterior muscle did not show such large changes (see Appendix) related to the velocity of the center of pressure. The contour plots of EMG signals are shown in the Appendix.

### 4. Discussion

#### 4.1 Stiffness and center of pressure

Muscle stiffness ranged from 56.7 to 75.9 N/m. These values are similar to those in our previous study (approximately 70 N/m [10]) although the estimation method was slightly modified, as described in 2.2.2. The muscle stiffness is also close to that estimated in a previous study using mechanomyography (52 N/m [16]). Muscle stiffness was estimated from the undamped natural angular frequency (approximately 16 rad/s ≈ 2.5 Hz). This finding is similar to that reported in a previous study (approximately 2.8 Hz [17]). The modified method used in the current study (short duration of output signal and neglecting backward sway and intrinsic fluctuation) provides a measure of medial gastrocnemius muscle stiffness.

Muscle stiffness was high when the displacement of the center of pressure was positive (Fig. 4). This result potentially supports the notion that ankle stiffness is high while standing with the center of pressure moving forward [18].

The muscle stiffness was also high when the velocity the center of pressure was negative (Fig. 4). The negative velocity indicates that the center of pressure moved backward. The highest stiffness was observed at approximately 0 cm displacement of the center of pressure and $-0.5$ cm/s of the velocity of the center of pressure. This displacement and velocity imply that the body was at the origin and swayed backward. This characteristic may contribute to stabilization of standing posture during paradoxical muscle movement [19]. In paradoxical movement, the soleus and gastrocnemius muscles lengthen during backward body sway. Muscle elongation during backward sway (negative velocity) and high stiffness can provide strong elastic force.

Muscle stiffness is controlled by muscle activity and is affected by the shape of the muscle. The muscle shape varies depending on the ankle angle. The ankle angle did not exhibit substantial changes during quiet standing. The ankle angle change was less than 1° [20] when the center of fluctuation was less than approximately ±1 cm, as shown in Fig. 1. A previous study has shown that the muscle-tendon unit length changes by 0.83 mm/° for a gastrocnemius muscle with a 39-cm leg length [21]. The current study indicates that the muscle shape change is small during quiet standing. Therefore, we propose that the muscle shape depends on the ankle angle and does not affect muscle stiffness, and that muscle stiffness is primarily affected by muscle activity.

The contour plot of the rmsEMG at $-0.3$ s (Fig. 5a) showed distribution similar to that of the muscle stiffness data shown in Fig. 4, although there were slight differ-
ence such as a difference in direction of the gradient. The preceding EMG may make the muscle stiff, considering that the EMG preceded the muscle length change (0.2 s) [3]. From the perspective of the control mechanisms involved in quiet standing, the fourth quadrant of the phase plane was stable. The fourth quadrant is also an inactive region according to intermittent postural control theory [11–13]. The rmsEMG at 0 s (Fig. 5d) was low in the fourth quadrant. This result is in accord with intermittent postural control theory.

Multiple linear regression explained the dependence of muscle stiffness on the displacement and velocity of the center of pressure. This regression may be useful for investigating the control mechanism of quiet standing in the phase plane. One limitation of the current study was that the measured ranges of the displacement and velocity of the center of pressure were small. It was difficult to record electrically induced fluctuation at large displacement and high velocity of the center of pressure.

4.2 EMG and center of pressure
The EMG signal increased as the displacement of the center of pressure increased, as shown in all panels of Fig. 5. The increase in the EMG signal is consistent with the results of previous studies [1, 18].

The EMG signal increased or decreased as the velocity of the center of pressure increased, depending on the preceding time of the EMG. The EMG signal was high in the fourth quadrant at the preceding time of 0.3 and 0.2 s (Fig. 5a and b). The EMG signal at the preceding time of 0.3 s exhibited a contour plot resembling that of stiffness (Fig. 4a). This high level of muscle activity may make the muscle stiff, as described in section 4.1.

In contrast, the EMG signal was high in the first quadrant at the preceding time of 0.1 and 0 s (Fig. 5c and d). This also supports the notion that the first quadrant is an active region in the intermittent postural control theory [11–13]. The high level of muscle activity during positive velocity may be related to the shortening of the muscle at forward sway in paradoxical movement [19]. These high levels of muscle activity did not affect the muscle stiffness at time $t$ at which the center of pressure was classified, because the preceding times (0.1 and 0 s) were short.

5. Conclusion
Electrically induced fluctuation of the center of pressure was measured and the transfer function from the electrical stimulation to the fluctuation was identified as a second-order (spring-mass-damper) model. The transfer function provided muscle stiffness depending on the displacement and velocity of the center of pressure. The muscle stiffness was high when the displacement was positive and the velocity was negative. The stiffness exhibited a contour plot resembling that of the rmsEMG at a preceding time of 0.3 s. The muscle stiffness was approximated well by multiple linear regression of the displacement and velocity of the center of pressure.

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Conflict of Interest
We have no conflicts of interest relationship with any companies or commercial organizations based on the definition of the Japanese Society for Medical and Biological Engineering.

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Appendix

The contour plots of the soleus and the tibialis anterior muscles are shown in Figs. A and B, respectively. The ranges of the rmsEMG of the soleus and the tibialis anterior muscles were expanded twice and four times, respectively, compared with that of the medial gastrocnemius muscle. The rmsEMG of the soleus muscle was related to the displacement of the center of pressure and was weakly related to the velocity of the center of pressure. The rmsEMG of the tibialis anterior muscle was maintained at an approximately constant level.

Fig. A Soleus rmsEMG. (a) RmsEMG at −0.3 s of classified displacement and velocity of the center of pressure. (b) −0.2 s, (c) −0.1 s, (d) 0 s.

Fig. B Tibialis anterior rmsEMG. (a) RmsEMG at −0.3 s of classified displacement and velocity of the center of pressure. (b) −0.2 s, (c) −0.1 s, (d) 0 s.
Takanori UCHIYAMA
Takanori UCHIYAMA received his Bachelor of Engineering, Master of Engineering, and PhD degrees from Keio University, Japan, in 1987, 1989, and 1992, respectively. In 1997, he joined Keio University, where he is currently a professor of Faculty of Science and Technology. His research interests include biological measurement. He is a member of JSMBE, SICE, IEICE, IEEJ, and IEEE.

Gai KONDO
Gai KONDO received his Bachelor of Engineering and Master of Engineering from Keio University, Japan, in 2017 and 2019, respectively. His research interests include biological measurement and signal processing.