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Title: Body Movement Induced by Electrical Stimulation of Toe  
Muscles during Standing

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## Abstract

The purpose of this study was to investigate whether artificially induced muscle contractions of toe muscles using functional electrical stimulation (FES) would cause center of pressure (COP) displacement and corresponding body acceleration. Ten able-bodied subjects were asked to stand still on force plates. The flexor digitorum brevis and the flexor hallucis brevis in both legs were simultaneously stimulated using a transcutaneous FES device. The muscles were stimulated twenty times at random intervals with four different stimulation intensities. We demonstrated that the toe muscle activity induced by electrical stimulation evoked COP displacement, which generated body acceleration. As expected, a larger stimulation induced a larger COP movement and acceleration. Therefore, we propose the use of FES-induced contractions of the toe muscles as a means to **control** balance during FES-assisted quiet standing. Spinal cord injured and severe stroke patients **could benefit from this electrical stimulation technique for improving FES-assisted standing.**

**Key Words:** Balance, Toe, Quiet Standing, Functional Electrical Stimulation

## Introduction

Development of a neuroprosthesis for standing that applies functional electrical stimulation (FES) to keep the body in an erect position is currently an active field of research. It is envisioned that individuals with spinal cord injuries, stroke or traumatic brain injuries may benefit from this technology using it as a permanent rehabilitation system to assist them in standing or as a therapeutic device that would be applied to retrain them to balance during quiet standing. So far, a number of FES systems for standing have been proposed (1-8). Some of these systems have been successfully used in laboratory environments; however, there is limited evidence that suggests that these systems could be easily transferred into a clinical setting. In practice, FES-assisted standing is meaningful only if a user can stand with the device, without having to use his/her arms to maintain balance, allowing him/her to perform various activities of daily living while standing. To address this problem, a number of closed-loop FES systems for standing have been proposed, which have tried to address the active balance control issue (2-6). Most of these systems investigated the regulation of balance by controlling muscle contractions around the ankle joint. But quiet standing is a much more complex task involving multiple body segments and joints acting in a coordinated manner to regulate balance during standing (9).

One can easily experience situations in daily life in which, in order to maintain balance, toes are used to correct for postural disturbances such as slips, trips, and pushes. Even during quiet standing, we can frequently feel that the movement of the toes helps us to maintain balance. Since the foot is the only body part in contact with the surface during quiet standing, it is very likely that toe movements play an important role in balance control during quiet standing.

To date, few studies have suggested the role of toes in controlling balance. Tanaka et al. (10, 11) measured the sole pressure of the great toe and postural stability of one leg stance, and suggested that the strength of the great toe relates to the stability of one leg stance. For quiet standing, Schieppati et al. (12) focused on the relationship between toe muscle activity and center of pressure (COP) movement. They measured the electromyogram

from the flexor digitorum brevis (FDB) during quiet standing and the COP movement, and found that the toe muscle activity correlated with the COP movement. They suggested that FDB has a responsibility in the control the COP. However, since ankle extensors work together with the FDB as agonists during quiet standing, one can not know if the correlation observed by Schieppati et al. (12) indicates the causal relation between the FDB activity and the COP movement. For balance control ‘the center of mass (COM) is the controlled variable, whereas the COP is the controlling variable’ (13). Therefore, if one could contract the toe muscles in isolation and demonstrate that these contractions can regulate COP position, the toe muscles should be capable of controlling balance during quiet standing.

The purpose of this study was to investigate whether artificially induced muscle contractions of toe muscles using FES would cause COP displacement and **movement/acceleration of the COM**.

## Materials and Methods

### Subjects

Ten healthy subjects (age  $26.4 \pm 3.1$  yrs, mean  $\pm$  SD), of whom five were female, participated in this study. All subjects signed written informed consents, which were approved by the local ethical committee and conformed to the principles of the Declaration of Helsinki.

### Procedure

Two force platforms (Type 9366AB05, Kistler, Switzerland) were used to measure the COP position, according to Winter et al. (13). The horizontal force was also measured using the force platforms. To investigate the actual body movement in one subject, a laser displacement sensor (LK-2500, Keyence, Japan) was used (14). The subject wore an elastic belt at the waist with a  $10 \times 10$  cm plastic plate on the back around lumbar vertebra 3, which is the approximate COM location. The laser beam was aimed at the plastic plate to measure the distance from the ground fixed laser device to the plastic plate. Thus, the measured body displacement provided a very good approximation of the actual COM dynamics during quiet standing. Note that this COM estimate was not used for the quantitative analysis; it was

only used for the comparison of the behavior of the COP with the COM.

The subjects were asked to stand quietly for about 2 min with their eyes closed while the COP, the horizontal force, and the horizontal position of the lumbar point were measured. In the experiments, only the anterior-posterior direction of sway was considered. All data were sampled at 1 kHz and stored on a personal computer for subsequent analysis. All kinematic and kinetic signals were low-pass filtered using a fourth-order, zero-phase-lag Butterworth filter (15). The cut-off frequency of the filter was set to 15 Hz.

The toe muscles of both feet, FDB and flexor hallucis brevis (FHB), were stimulated using a FES system (Complex Motion, Complex, Switzerland) (16). **Two surface stimulation electrodes** ( $1.5 \times 1.5$  cm) were located on the muscle bellies of each foot, and the indifferent electrodes were located behind the malleolus medialis (Fig. 1). All muscles were simultaneously stimulated **using biphasic asymmetrical pulses with constant current, a frequency of 35 Hz and a pulse duration of 300  $\mu$ s**. Four different levels of stimulation intensity were applied based on the motor threshold (MT); **0.8, 1.1, 1.3 and 1.7 MT**. The MT was determined as follows. Increasing the stimulation intensity gradually, the experimenter observed the movement of the hallux and the other toes for identification of the FDB's MT and for that of the FHB, respectively. The lowest intensity that caused one of the toe muscles to start contracting was identified as the MT intensity for the corresponding muscle. Each stimulation interval was 1 s long, and 20 trials for each stimulation intensity were set as a block of consecutive stimulations. The interval between each trial was about 4 s, but the experimenter randomized it to avoid the subject's anticipatory reactions, while visually checking whether the response disappeared. The inter-trial interval of 4.0 s was sufficient for subjects to recover their posture. The blocks of the different stimulation levels were randomized for individual subjects. Note that, to make their standing posture as natural as possible, the subject's basal position was not controlled.

## Analysis

The period of 0.2 s before each stimulation **and 2.0 s after the onset of the stimulation was used for the following analysis (note that stimulation lasted 1 s)**. The time stamp was set

from -0.2 to 2.0 s for each trial data. We subtracted the offset from each trial data using the value at 0.0 s. Each data set with a given stimulation intensity for each subject was obtained by ensemble averaging 20 trials.

The amplitude of the COP response was divided into three phases. The first phase was between 0.2 s and 0.6 s, where the initial reaction to the stimulation was seen, as shown in the Results section. As the COP dynamically moved forward in this phase, we quantified the response as the **maximum** value during this phase. The second phase was between 0.8 s and 1.0 s, where the COP was relatively steady as the response to the continuous stimulation. As the COP was steady, we quantified the response as the average value during this phase. The third phase was between 1.0 s and 1.6 s, where we saw a post stimulation response. As the COP moved backward, we quantified the response as the **minimum** value during this phase.

The horizontal acceleration (ACC) of the COM was estimated according to the Newton's law using the horizontal force ( $F$ ) measured by the force platform as,

$$ACC(t) = F(t)/m, \quad (1)$$

where  $m$  is the COM mass of the inverted pendulum.  $m$  was estimated as  $0.971M$  where  $M$  indicates the subject's body mass in kg (15). The responses of the ACC were also analyzed in the same three phases **as the COP**. As the ACC dynamically moved backward in the 1st phase, we quantified the response as the peak value during this phase. As the ACC was steady in the 2nd phase, we quantified the response as the average value during this phase. As the ACC moved backward in the 3rd phase, we quantified the response as the peak value during this phase.

According to the inverted pendulum model, the deviation of the COP from the COM position generates the ACC (13). **If the stimulation induces the COP movement but the COM does not move at the same time, a deviation of COP will be induced, which may generate the ACC accordingly. This assumption was verified in the following way.** Under this assumption (COM displacement = 0), the peak COP ( $COP_{peak}$ ) and the peak ACC ( $ACC_{peak}$ ) will satisfy the following equation:

$$ACC_{peak} = \frac{mgh}{I} COP_{peak}, \quad (2)$$

where  $I$  is the moment of inertia of the pendulum about the ankle joint,  $g$  is the gravitational constant, and  $h$  is the COM height from the ankle joint. We examined whether this equation was satisfied in the 1st peaks of COP and ACC or not. Regarding the 1st peak, we calculated the estimated peak ACC using the peak COP according to equation (2), and then we compared the estimated peak ACC with the actual peak ACC using linear regression analysis. In the calculation, we estimated each anthropometric values according to Winter (15):  $I = 0.678M(0.374l_1 + l_2)^2 + 0.192Ml_2^2$  kgm<sup>2</sup>:  $h = 0.261l_1 + 1.014l_2$  m where  $l_1$  indicates the length between the glenohumeral joint and the greater trochanter, and  $l_2$  indicates the length between the greater trochanter and the medial malleolus.

The difference of group mean values of the COP parameters among MT conditions was tested using one-way repeated measures ANOVA. The difference of group mean values of the maximum ACC parameters among MT conditions was also tested using one-way repeated measures ANOVA. Tukey-Kramer test was applied for post hoc analysis.  $P < 0.05$  was used as a level of significance to prevent excessive false-positive results.

## Results

The stimulation intensity for each muscle and each MT is shown in Table 1. The representative COP traces for one subject before ensemble averaging are shown in Fig. 2 for each stimulation intensity. In 0.8 MT traces (Fig. 2A), there was consistently no movement among the traces. However, we observed a consistent forward movement from 0.2 to 0.6 s (the 1st phase) with a peak at around 0.4 s in all other three MT conditions (Fig. 2B, C, and D). The forward movement in the 1st phase became more obvious if ensemble average traces were used, as shown in Fig. 3A. One can see that the amplitude of the forward movement depends on the stimulation intensity. The group means of the forward movement are shown in Fig. 3B. The COP displacements were significantly different among MT conditions as revealed by the ANOVA test ( $P < 0.0001$ ), and the displacements were larger for larger stimulation intensities as revealed by the post hoc test.

In Fig. 3A, one can see that the COP moved backward after the forward movement and

remained in the back position during the entire duration of the 2nd phase. Then, the COP remained in the backward position for about 0.2 s after the stimulation was terminated, followed by further displacement backwards (peak at 1.4 s). After the backward peak, the COP moved to its initial position. The group means of the average displacement in the 2nd phase and the peak displacement in the 3rd phase are shown in Fig. 3C and in Fig. 3D, respectively. The COP displacements were significantly different among MT conditions in the 2nd phase ( $P < 0.0001$ ) and in the 3rd phase ( $P < 0.0001$ ). However, it should be noted that the variations of the responses in the 2nd phase and 3rd phase were much larger than in the 1st phase (Fig. 2 and Fig. 3).

In Fig. 4A, we compared the traces of COP, laser measurement (COM estimate) and ACC of 1.7 MT condition calculated using equation (1), for one subject. While the COP started moving about 0.2 s after the onset of the stimulation, the laser measurement did not move at that moment and started moving only after 0.4 s. Therefore, it is assumed that the deviation of the COP from the COM started at 0.2 s, and reached the largest deviation at around 0.4 s. At the same time as the deviation between the COP and the laser measurement was observed (1st phase), a backward ACC was observed as well, and reached a peak at around 0.4 s. At around 0.7 s, the COP and the laser measurement coincided again, and the ACC returned to zero. At around 1.2 s (i.e. 0.2 s after the cessation of the stimulation), the COP started deviating from the laser measurement again but this time backwards, while the laser measurement stayed at the same position and the forward ACC was generated at the same moment. At around 1.4 s, the laser measurement indicated forward movement of the COM. At the same moment, the COP position showed a peak of backward movement and the ACC also showed a forward peak. The group means of the peak displacement in the 1st phase, the average displacement in the 2nd phase and the peak displacement in the 3rd phase of the ACC are shown in Fig. 4B, 4C, and in 4D, respectively. All parameters were significantly different among MT conditions, while the average in the 2nd phase were close to zero for all MT intensities and did not show the clear and monotonic dependency on the MT intensity.

We compared the actual ACC with the ACC estimated from the COP regarding the

1st peaks in Fig. 5. The data from all subjects and all MT levels were grouped together. Linear regression analysis provided the regression lines of  $Y = 0.000424 + 0.993X$ ,  $R^2 = 0.940$  ( $X$  indicates the actual ACC, and  $Y$  indicates the estimated ACC). The regression line is quite close to the line of identity, despite inherent errors in estimating the anthropometric parameters. A 95 % confidence interval for the slope resulted in values from 0.911 to 1.075, which included 1, and a 95 % confidence interval for the intercept resulted in values from -0.002 to 0.003, which included 0.

## Discussion

The toe muscle stimulation was able to evoke the COP forward movement with a latency of about 0.2 s (Fig. 3 and Fig. 4A). Simultaneously with the COP displacement, a backward ACC was generated (Fig. 4A). The ACC was equivalent to the COP deviation from the COM as predicted by the equation (2), which assumes that the COM is stationary until the COP displacement reaches its peak (Fig. 5). Thus, we successfully demonstrated that by stimulating toe muscles it is possible to induce COP movement, and an ACC as a result of this COP movement.

### Feasibility of the Toe Muscle Stimulation for FES Standing

Studies that have evaluated FES systems for quiet standing, have focused on control of the ankle joint via electrical stimulation of gastrocnemius, soleus and tibialis anterior (2-8). However, the present results indicate that toe muscle stimulation is capable of regulating COP dynamics as well, and may be a good candidate for controlling balance in FES-assisted standing applications. Our results suggest that the COP displacement and the ACC, induced by electrically stimulated FHB and FDB, are functions of the stimulation intensity (Fig. 3B, Fig. 4B and Fig. 4C), i.e. the results imply that body acceleration can be controlled during quiet standing by regulating the stimulation intensity applied to FHB and FDB. Therefore, it might be feasible to use this technique to regulate balance during standing. When one sways in stance, one can easily feel the toe movement only when the body moves forward from its natural body location. This implies that the toe movement has some responsibility

in producing backward movement of the body **as a reaction to the forward movement**. Therefore, electrical stimulation of toe muscles should be considered as a potential mechanism for balance regulation during FES-assisted quiet standing. One of the problems of using the toe muscle stimulation for the purpose of artificial balance control is the latency in the system response to electrical stimulation, i.e. the COP movement was produced about **0.2 s after the onset of stimulation**. The actual reasons for this latency are not known yet, but can be attributed to a combination of foot-joint mechanics and muscle force generation latency. One could also suggest that the latency is due to the neural feedback loop. Regardless of the source of the latency, one has to take it into consideration when an FES-system for standing is being designed that will stimulate FHB and FDB. Further systematic studies are required to determine how electrical stimulation of FHB and FDB could assist balance control during FES-assisted quiet standing.

After the initial response to the toe muscle stimulation (1st phase), we observed the steady state shift (2nd phase) and the post stimulation response (3rd phase) (Fig. 4A). In the 2nd phase, the COP was located in the back, compared to its initial location without the prominent body acceleration. **In the 3rd phase, after the electrical stimulation stopped contracting the toe muscles, the foot deformation was released and the opposite reaction to the 1st phase occurred, i.e., a backward movement of COP. The backward movement of COP generated the deviation of COP from COM again but in the opposite direction to the one in the 1st phase. Then the forward body acceleration was induced, and the body moved forward to the initial position.** Although the responses in the 2nd and 3rd phases were clearly observed in ensemble averaged traces (Fig. 4A), the variance of the responses was larger than in the 1st phase (Fig. 2, Fig. 3A). This suggests that, when the toe muscle stimulation is introduced in the FES-assisted standing system, one could predict the response of the 1st phase but would have difficulties predicting the responses in the 2nd and 3rd phases. This phenomenon can present an additional technical problem in implementing toe muscle stimulation for the purpose of balance control during FES-assisted standing.

## Mechanism of the Response to Toe Muscle Stimulation

The mechanism of the body movement can be explained as follows. Since toe muscle activity induces deformation of the foot and redistribution of the forces under the foot, which affect the COP position, toe muscle activity is able to induce COP movement without a need to contract the ankle muscles. Since COP deviation from COM generates ACC, it is logical to conclude that the toe muscle stimulation generates ACC of the body during quiet standing.

Another possible explanation for the body movement due to electrical stimulation of the toe muscles is an indirect stimulation of cutaneous afferents. The contracted toe muscles increase the tactile sensation at the toe, which indirectly induces the cutaneous afferent activity. Kavounoudias and his colleagues demonstrated in a series of studies (17-19) that the cutaneous information contributes to balance control during quiet standing. They demonstrated that the vibration to the foot sole around the toe induces a soleus activity at the latency of  $0.119 \pm 0.028$  s, which generates a small forward movement of the COP at the latency of  $0.251 \pm 0.111$  s followed by the COP backward movement with the latency of  $0.9 \pm 0.4$  s (18). They also monitored body sway reaction by measuring ankle angle displacement that occurs at the latency of  $0.434 \pm 0.170$  s (19). These latencies are identical to those observed in our experiments.

Besides indirect stimulation of cutaneous afferents discussed above, cutaneous afferents can be stimulated directly. It has been demonstrated that electrical stimulation in the same areas as those stimulated in this study induces soleus excitatory activity in an interval of long-latency reflex (about 0.12-0.22 s) (20, 21). Andersen et al. (20) also investigated the response of COP, and found that a small forward COP movement (0.1-0.44 cm) occurred with a latency of about 0.14 s using the same location of stimulation. Thus, the direct cutaneous stimulation can induce the soleus activity, and the soleus activity induces COP forward movement at about the same latency and same amount as those in our experiment. Therefore, although the stimulation methods in those studies was different from this study, and was used for direct stimulation of cutaneous afferents, it is possible that the stimulation used in this study could elicit the calf muscle activities via direct stimulation of the cutaneous

afferent, which could induce the COP forward movement at the latency around 0.2 s. Future analysis involving EMG recordings may help determine to what extent electrical stimulation of cutaneous afferents affects the observed COP displacement.

Most of the muscles that have motor points on the sole of the foot are only actuating joints within the foot. The only muscle that spans the ankle joint and the foot is the lumbricalis muscle. Therefore, if the lumbricalis muscles had been stimulated, they might have exerted the ankle torque in addition to the toe muscle activity. However, we believe that potential contributions of the lumbricalis muscle contractions in this particular case are insignificant. The reported anthropometric parameters of the lumbricalis muscle shows the following: The moment arm of the lumbricalis muscle in the neutral position is 15 mm (22): The pennation angle is 12.6 deg (23): The physiological cross-sectional area is 1.04 cm<sup>2</sup> (24). Therefore, if we estimate the specific tension of the muscle as 22.5 N/cm<sup>2</sup> (25), the maximum torque contribution of the muscle will be 0.34 Nm ( $22.5 \times 1.04 \times \cos 12.6 \times 0.015$ ). If the COP displacement is equal to 5 mm, the required ankle torque needed to make this displacement has to be 3 Nm for a person whose weight is 60 kg. Therefore, even if the lumbricalis muscles had been erroneously activated to their full contraction capacity in our experiments, they could not have generated sufficient torque to cause ankle movements that would result in the COP displacements observed in our experiments.

## Conclusions

In this article we have shown that the FES-induced contractions of the flexor digitorum brevis and the flexor hallucis brevis (toe muscles) evoked COP displacement, which generated body acceleration. As expected, a larger stimulation induced a larger COP movement and acceleration. This finding suggests that FES of the flexor digitorum brevis and the flexor hallucis brevis could be used to regulate balance during FES-assisted quiet standing. Spinal cord injured and severe stroke patients could potentially benefit from such FES-assisted standing.

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	Stimulation Intensity [mA]			
	Left		Right	
	FHB	FDB	FHB	FDB
	6.6±2.0	7.8±2.0	7.3±1.3	7.0±2.3
0.8MT	5.6±2.0	6.8±2.0	6.3±1.3	6.0±2.3
1.1MT	7.5±2.5	8.9±2.6	8.3±1.3	7.8±2.9
1.3MT	8.7±3.1	10.6±3.2	9.8±1.8	9.3±3.5
1.7MT	11.2±4.0	13.6±4.1	13.6±3.2	12.0±4.5

Table 1: Stimulation intensity for each motor threshold (MT) for each muscle.

## Figure Legends

### Fig. 1 Locations of the stimulation surface electrodes

Two stimulation surface electrodes ( $1.5 \times 1.5$  cm) were located on the muscle bellies of the flexor digitorum brevis and the flexor hallucis brevis. The indifferent electrodes were located behind the malleolus medialis.

### Fig. 2 Representative examples of the center of pressure (COP) traces

A; 0.8 motor threshold (MT), B; 1.1 MT, C; 1.3 MT, D; 1.7 MT. Each graph shows 10 superimposed COP traces for a subject to illustrate the variation of the response. The stimulation was applied from 0.0 s to 1.0 s. The origin of the COP position was shifted to 0.0 cm at the moment of the stimulation in all traces. **The positive value of the COP indicates forward and the negative value indicates backward direction of motion.**

### Fig. 3 Representative examples of the ensemble averaged center of pressure (COP) trace and the group result of the COP displacement

A; Ensemble averaged COP traces for all stimulation **intensities** for a subject. The thin line, the dotted line, the dashed line, and the thick lines indicate 0.8 motor threshold (MT), 1.1 MT, 1.3 MT, and 1.7 MT, respectively. The grey areas show the range between the ensemble average plus SD and minus SD for each stimulation level. **The positive value of the COP indicates forward and the negative value indicates backward direction of motion.** B; The group mean values of the maximum COP displacement in the 1st phase for each stimulation level. C; The group mean values of the average COP displacement in the 2nd phase for each stimulation level. D; The group mean values of the minimum COP displacement in the 3rd phase for each stimulation level. Error bars show the standard deviation of the mean value. The *P* value indicates the result of the ANOVA test. \* indicates  $P < 0.05$  for the post hoc test.

### Fig. 4 Representative example of the ensemble averaged center of pressure (COP),

**the laser measurement that represents center of mass (COM) and the COM acceleration (ACC), and the group result of the ACC amplitude**

A; Ensemble averaged COP, laser measurement and ACC traces for 1.7 motor threshold (MT) for a subject. The thin line and the thick line in the upper traces indicates the COP and the laser measurement, respectively. The lower trace indicates the ACC. **The positive value of the COP indicates forward and the negative value indicates backward direction of motion.**

B; The group mean values of the minimum ACC in the 1st phase for each stimulation level.

C; The group mean values of the average ACC in the 2nd phase for each stimulation level.

D; The group mean values of the maximum ACC in the 3rd phase for each stimulation level.

Error bars show the standard deviation of the mean value. The  $P$  value indicates the result of the ANOVA test. \* indicates  $P < 0.05$  for the post hoc test.

**Fig. 5 Comparison between the actual ACC and the estimated ACC from COP-COM for each trial**

Linear regression analysis provided the regression lines (thick line) of:  $Y = 0.000424 + 0.993X, R^2 = 0.940$  ( $X$  indicates the actual ACC, and  $Y$  indicates the estimated ACC).

**The peak ACC during the 1st phase is shown and compared to the estimated peak ACC calculated according to equation (2).** Note that regression line is close to the line of identity (thin line).

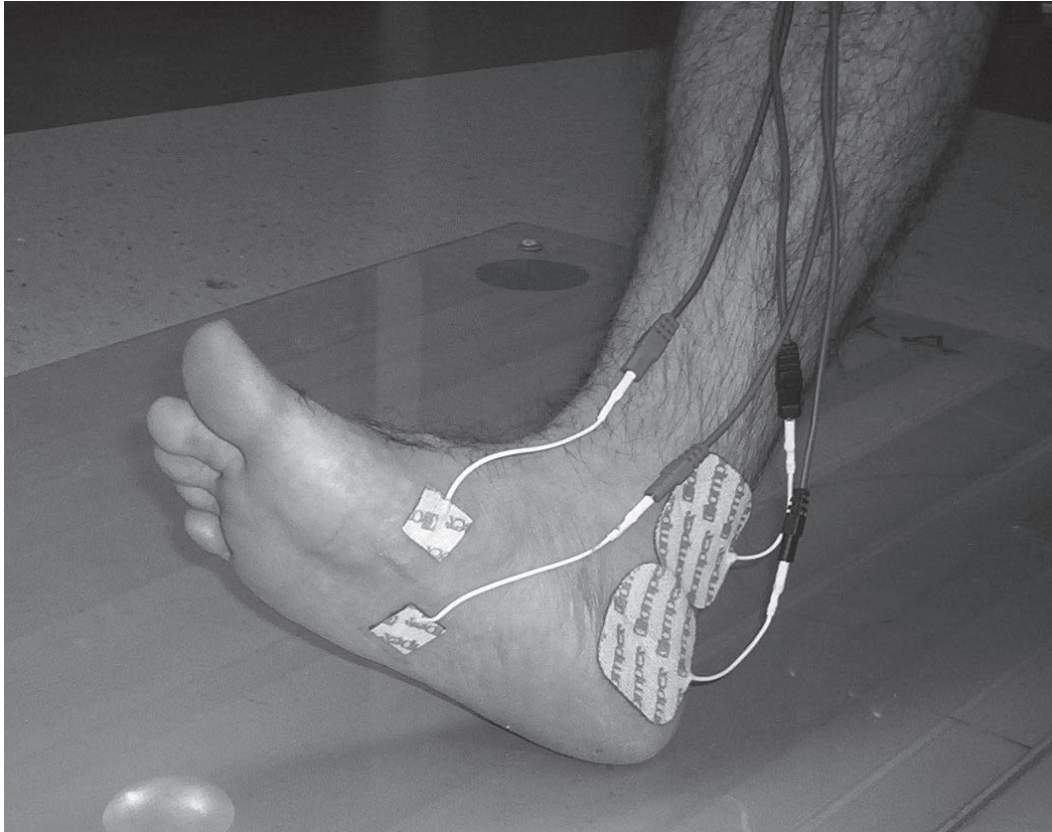


Figure 1: **Locations of the stimulation surface electrodes** Two stimulation surface electrodes ( $1.5 \times 1.5$  cm) were located on the muscle bellies of the flexor digitorum brevis and the flexor hallucis brevis. The indifferent electrodes were located behind the malleolus medialis.

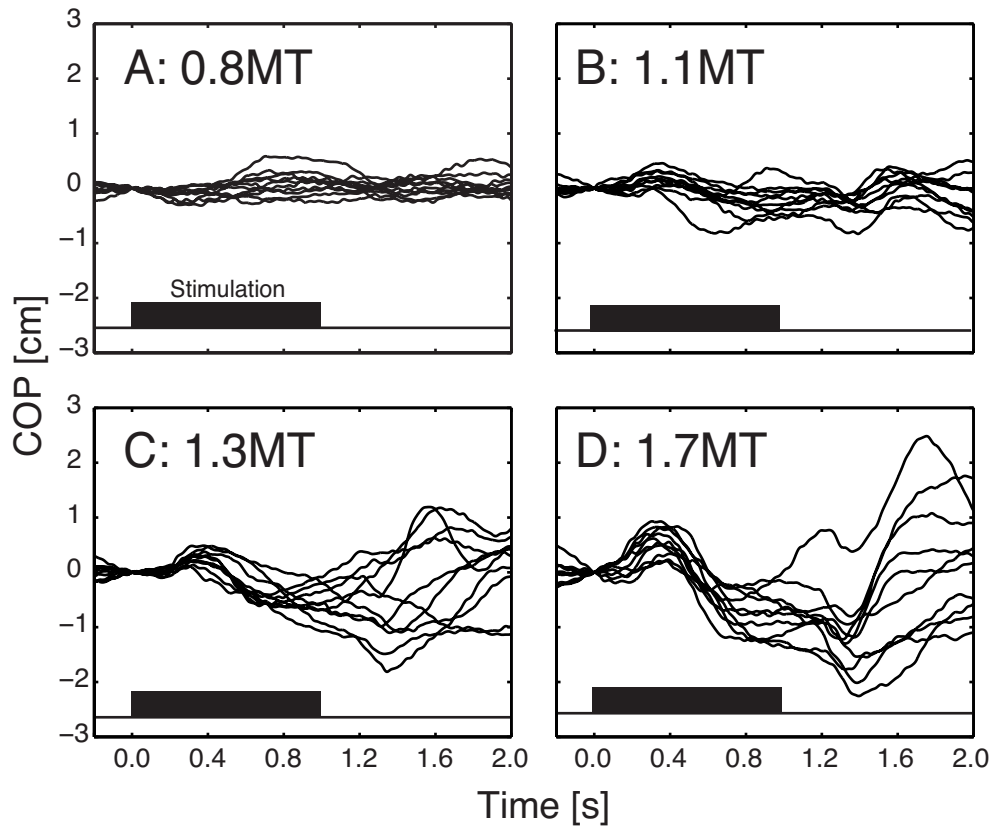


Figure 2: **Representative examples of the center of pressure (COP) traces** A; 0.8 motor threshold (MT), B; **1.1 MT**, C; **1.3 MT**, D; **1.7 MT**. Each graph shows 10 superimposed COP traces for a subject to illustrate the variation of the response. The stimulation was applied from 0.0 s to 1.0 s. The origin of the COP position was shifted to 0.0 cm at the moment of the stimulation in all traces. **The positive value of the COP indicates forward and the negative value indicates backward direction of motion.**

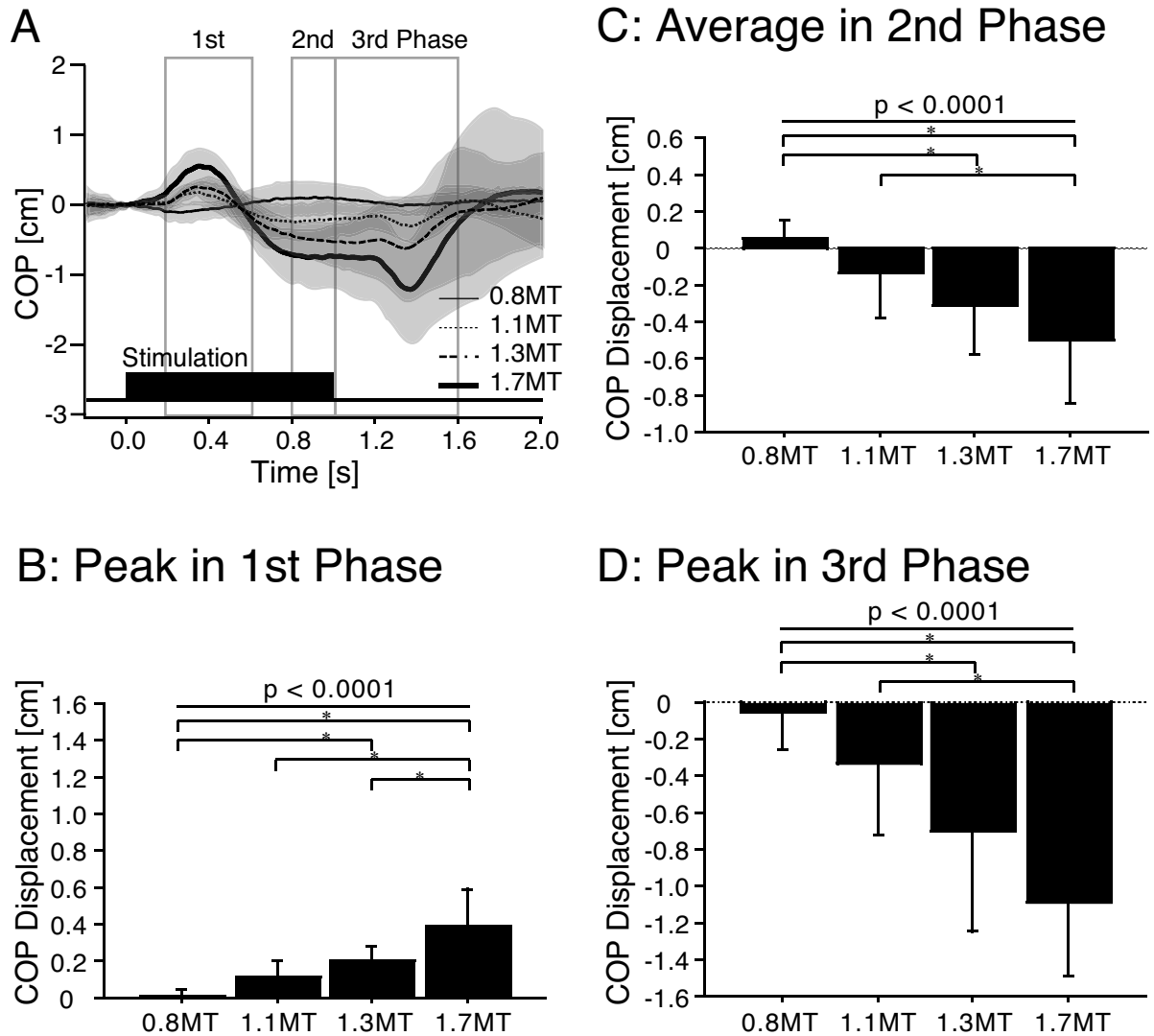


Figure 3: **Representative example of the ensemble averaged center of pressure (COP) trace and the group result of the COP displacement** A; Ensemble averaged COP traces for all stimulation intensities for a subject. The thin line, the dotted line, the dashed line, and the thick lines indicate 0.8 motor threshold (MT), 1.1 MT, 1.3 MT, and 1.7 MT, respectively. The grey areas show the range between the ensemble average plus SD and minus SD for each stimulation level. **The positive value of the COP indicates forward and the negative value indicates backward direction of motion.** B; The group mean values of the maximum COP displacement in the 1st phase for each stimulation level. C; The group mean values of the average COP displacement in the 2nd phase for each stimulation level. D; The group mean values of the minimum COP displacement in the 3rd phase for each stimulation level. Error bars show the standard deviation of the mean value. The  $P$  value indicates the result of the ANOVA test. \* indicates  $P < 0.05$  for the post hoc test.

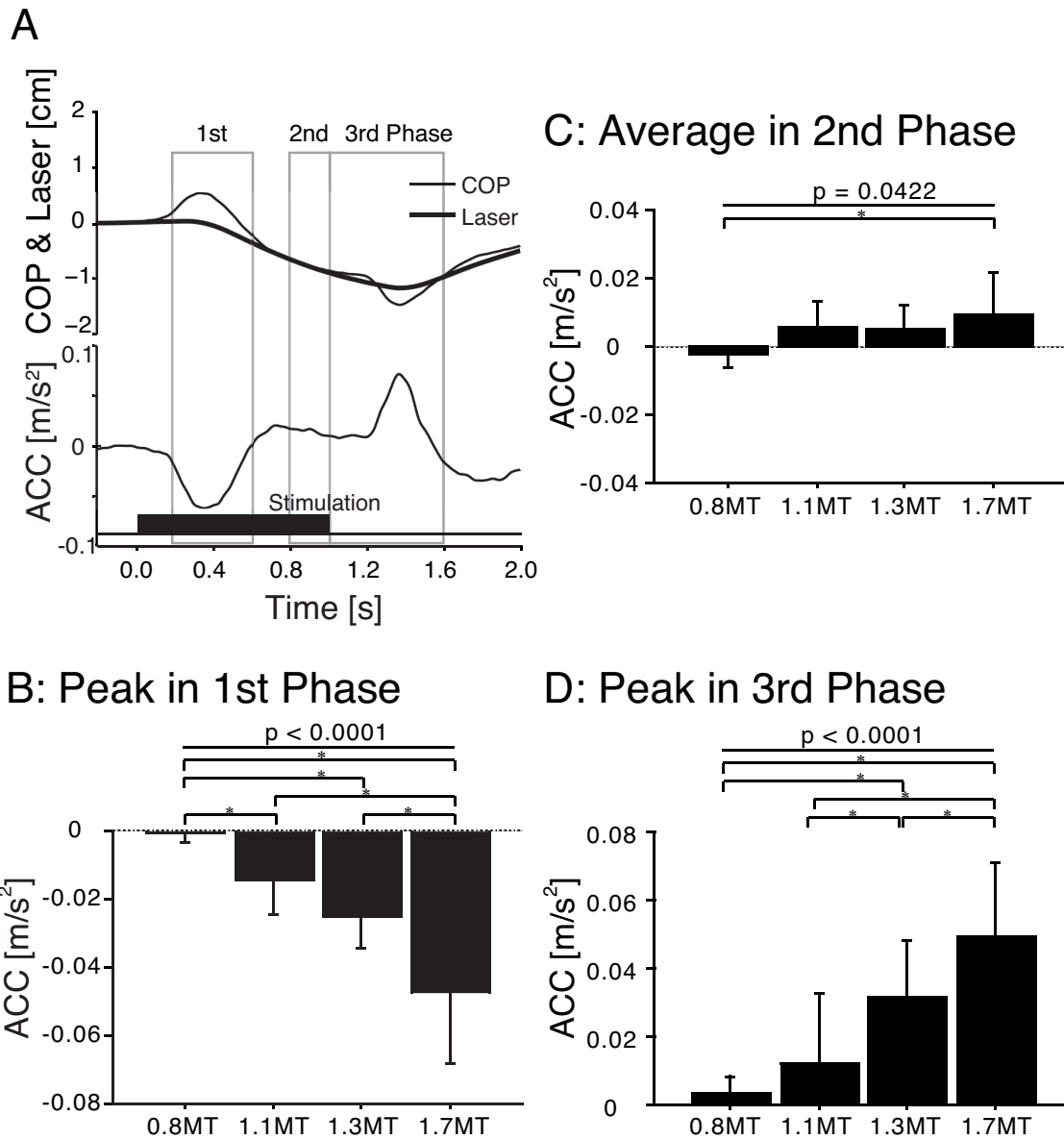


Figure 4: **Representative example of the ensemble averaged center of pressure (COP), the laser measurement that represents center of mass (COM) and the COM acceleration (ACC), and the group result of the ACC amplitude A; Ensemble averaged COP, laser measurement and ACC traces for 1.7 motor threshold (MT) for a subject. The thin line and the thick line in the upper traces indicates the COP and the laser measurement, respectively. The lower trace indicates the ACC. The positive value of the COP indicates forward and the negative value indicates backward direction of motion.** B; The group mean values of the minimum ACC in the 1st phase for each stimulation level. C; The group mean values of the average ACC in the 2nd phase for each stimulation level. D; The group mean values of the maximum ACC in the 3rd phase for each stimulation level. Error bars show the standard deviation of the mean value. The  $P$  value indicates the result of the ANOVA test. \* indicates  $P < 0.05$  for the post hoc test.

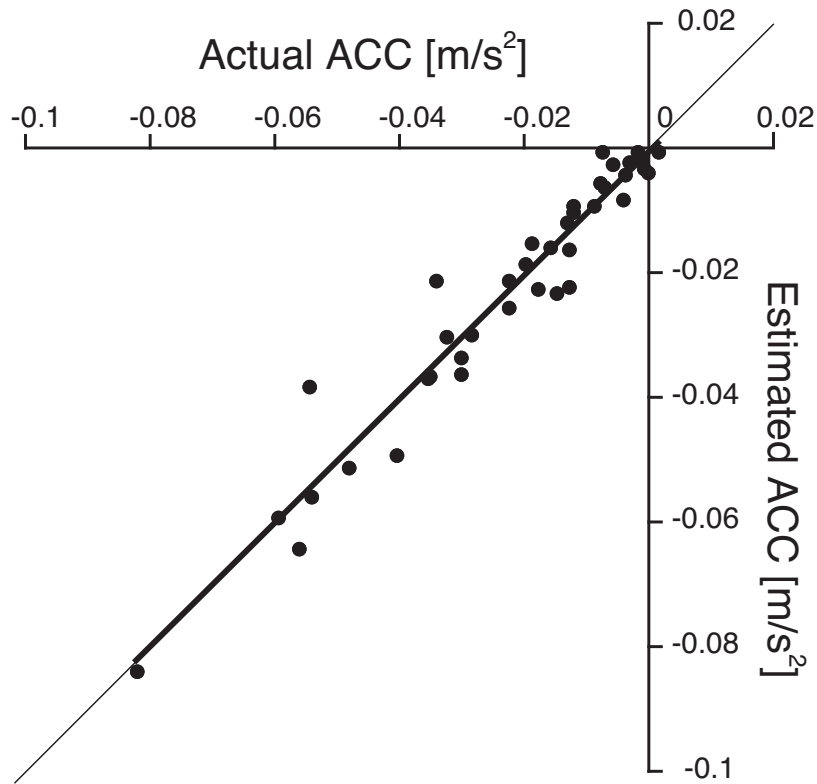


Figure 5: Comparison between the actual ACC and the estimated ACC from COP-COM for each trial. Linear regression analysis provided the regression lines (thick line) of:  $Y = 0.000424 + 0.993X$ ,  $R^2 = 0.940$  ( $X$  indicates the actual ACC, and  $Y$  indicates the estimated ACC). The peak ACC during the 1st phase is shown and compared to the estimated peak ACC calculated according to equation (2). Note that regression line is close to the line of identity (thin line).