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Implementation of a Physiologically Identified PD Feedback Controller for Regulating the Active Ankle Torque during Quiet Stance

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Running Head: PD Control of Active Ankle Torque during Quiet Stance

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Abstract

Our studies have recently demonstrated that a proportional and derivative (PD) feedback controller, which takes advantage of the body's position and velocity information to regulate balance during quiet standing, can compensate for long neurological time delays and generate a control command that precedes body sway by 100 to 200 ms. Furthermore, PD gain pairs were identified that ensure a robust system behavior and at the same time generate dynamic responses as observed in quiet standing experiments with able-bodied subjects. The purpose of the present study was to experimentally verify that the PD controller identified in our previous study can: 1) regulate the active ankle torque to stabilize the body during quiet standing in spite of long neurological time delays; and 2) generate system dynamics, i.e., a motor command and body sway fluctuation, that successfully mimic those of the physiologic system of quiet standing. Our real-time closed-loop feedback circuit consisted of a center of mass position sensor and a functional electrical stimulator that elicited contractions of the plantar flexors as determined by the aforementioned PD controller. The control system regulated upright stance of a subject who was partially de-afferented and -efferented due to a neurological disorder called von Hippel-Lindau Syndrome (McCormick Grade III). While the subject was able to generate a motor command for the ankle joints, he could not *regulate* the resulting torque sufficiently due to a lack of sensory feedback. It is important to mention that a time delay was included in the closed-loop circuit of the PD controller to mimic the actual neurological time delay observed in able-bodied individuals. The experimental results of this case study suggest that the proposed PD controller in combination with a functional electrical stimulation system can regulate the active ankle torque during quiet stance and generate the same system dynamics as observed in healthy individuals. While these findings do not imply that the CNS actually applies a PD-like control strategy to regulate balance, they suggest that it is at least theoretically possible.

Index Terms

Active ankle torque, functional electrical stimulation, neurological time delay, PD feedback control, von Hippel-Lindau Syndrome.

I. INTRODUCTION

To date many studies have investigated the control strategy that healthy individuals apply to regulate balance during quiet standing [1]-[23]. It has been shown that the ankle joint torque needed to control the body during quiet stance can be evoked passively and actively. *Passive torque* components, which are the result of intrinsic mechanical properties of the joints, i.e., stiffness and viscosity, are not sufficient to generate the required torques to prevent the body from falling [1]-[4]. Therefore, additional *active torque* components, regulated by the central nervous system (CNS), are needed to ensure a stable system behavior.

If one assumes the involvement of the CNS during the upright balancing task as part of a classical feedback control scheme, three time delays have to be considered with respect to the active control task [5]:

1. A feedback time delay that represents cumulative time loss due to neural-transmission from the ankle somatosensory system to the brain (τ_F). This sensory modality has been chosen as it is the furthest away from the CNS, implying the longest feedback time delay among the sensory systems during quiet standing. Moreover, it has been shown that proprioceptive inputs from the lower legs and feet provide highly sensitive sensory information required for accurate perception of postural sway [6], being sufficient to allow a stable upright stance in the absence of other sensory modalities involved in the control of quiet standing [7].
2. A motor command time delay, which represents cumulative time loss due to the sensory-motor information process in the CNS and the neural transmission from the CNS to the plantar flexors (τ_M).

3. An electromechanical response time that represents the time difference between the moment when the electromyogram (EMG) signal is generated and the moment when the muscle force occurs (τ_E).

The cumulative value of these three delays, further called the *neurological time delay*, has been reported to be at least 80 ms [5]. A specific range for the neurological time delay cannot be given at this point, as the exact time needed for the sensory-motor information process in the CNS is not known. Note that, since the center of mass (COM) of the body is located high above a relatively small base of support during quiet standing, a longer neurological time delay implies a greater control challenge in stabilizing the system.

To compensate for this significant time delay or completely avoid its integration, it has been hypothesized that a feed-forward control mechanism is required to regulate balance during quiet standing [1], [3], [8]-[12]. This theory was especially appealing since experiments with able-bodied subjects demonstrated that the fluctuation of the motor command to the plantar flexors precedes the body sway fluctuation during quiet standing [12], [13]. Moreover, De Nunzio *et al.* [14] showed that a mechanical disturbance of the proprioceptive modality during quiet standing had little effect on body stabilization, suggesting that equilibrium control depends more on anticipation than on continuous proprioceptive feedback. However, this lack of effect of proprioceptive disturbance could also be explained by a dynamic re-weighting of the information provided by all the sensory modalities as reported during different test conditions [15]. As such, no conclusive physiologic evidence has been put forward that a feed-forward control mechanism is at work during quiet standing.

These considerations indicate that there is an ongoing debate whether a feed-forward or feedback control mechanism is predominantly responsible for stabilizing the body during quiet standing. As neither of these two strategies is universally accepted, both strategies need to be

challenged. Since a simple linear analysis can reveal a strong contribution of the sensory information to the balancing task during quiet standing [6], [7], [11], a linear feedback controller has already been considered for modeling the physiologic control mechanism of balance [16]. In order to determine the theoretical potential of a classical feedback system with respect to balance control, we investigated whether a proportional and derivative (PD) controller, which is assumed to work at a higher level of the CNS and utilizes the position and velocity information of the ankle joint, can provide the preceding active joint torque and ensure balance despite the neurological time delay [5], [13].

Our theoretical analysis has shown that a PD controller with various combinations of proportional (Kp) and derivative (Kd) gains is able to ensure balance during quiet standing [5]. Moreover, if relatively high velocity gains are applied, the PD controller can yield system dynamics (motor command and COM fluctuation) as observed in healthy individuals, despite neurological time delays of 80 to 135 ms [5]. This outcome complements the results published by Fukuoka *et al.* [24] and Jeka *et al.* [25], who have shown that the human somatosensory feedback system has, in fact, derivative characteristics [24] and that the sensory velocity information used for body stabilization is more accurate than the position or acceleration information [25]. Taking these findings into account, we concluded in [5] that a feed-forward mechanism is not implicitly needed to compensate for the neurological time delay as suggested by others.

The ability to perform stable quiet standing has various therapeutic and functional benefits for individuals with spinal cord injury (SCI) and other neurological disorders such as stroke and traumatic brain injury. A number of functional electrical stimulation (FES) systems have been proposed to date that are intended to allow these individuals to maintain balance and stand freely, i.e., without the use of their hands. These systems apply some sort of closed-loop

control strategy to regulate the output of the FES system. Based on studies by Matjačić and Bajd [26], [27], Jaime *et al.* [28] proposed a strategy that allowed a paraplegic subject with a complete lesion at T5 to maintain balance in a constricting multipurpose rehabilitation frame. Their control strategy implemented voluntary and reflex activity of the upper body while a closed-loop FES system regulated the active stiffness of the ankle joints. Using several control design approaches, Hunt *et al.* [29], Holderbaum *et al.* [30], and Gollee *et al.* [31] developed and evaluated a series of nested feedback systems that used FES to control the ankle torque of a paraplegic subject with a complete lesion at T7/8. During the experiments, the subject was standing in an apparatus that acts as a full body cast (*Wobbler*), allowing solely the ankle joints to move in the anterior-posterior direction. Abbas and Chizeck [32] compared the performance of a closed-loop feedback system with an open-loop stimulation strategy for two paraplegic subjects (T7 and T9) during standing, where both systems regulated the movement of the hip joints in the coronal plane. Note that all of these studies have demonstrated that FES has some potential to allow individuals with SCI to stand quietly. However, none of the proposed systems have considered mimicking the dynamics of the actual physiologic system during quiet standing and consequently did not implement its neurological time delay.

As such, the purpose of the present study was to experimentally verify that the PD controller identified in our previous study [5] can: 1) regulate the active ankle torque to stabilize the body during quiet standing in spite of long neurological time delays; and 2) generate system dynamics, i.e., a motor command and body sway fluctuation, that successfully mimic those of the physiologic system of quiet standing. The PD controller was used to regulate FES induced contractions of the plantar flexors of a subject who was partially de-afferented and -efferented due to a neurological disorder called von Hippel-Lindau Syndrome. While the subject was able to generate a motor command for the ankle joints, he could not *regulate* the resulting torque due

to a lack of sufficient sensory feedback. To accurately mimic the actual physiologic system, a time delay was included in the closed-loop control circuit such that its cumulative time delay was in the range of the aforementioned neurological time delay of healthy individuals, i.e., longer than 80 ms. The results of this experimental case study confirm our theoretical findings that a PD controller with a relatively high velocity gain is able to stabilize the body and generate system dynamics that mimic those of the actual physiologic system as suggested in [5].

II. MATERIALS AND METHODS

A. Experimental Setup

In accordance with our theoretical study [5], the real-time closed-loop system was designed to investigate the stability of the anterior-posterior body sway. Fig. 1 shows the experimental setup schematically: The PD controlled system received its input from a laser displacement sensor (LK2500, Keyence, Japan), which recorded the anterior-posterior body sway fluctuation (1000 Hz). This measurement was used as an approximation for the COM fluctuation after confirming that the dynamics of quiet standing could be approximated by an inverted pendulum rotating around the ankle joint [12], [33]. The subject wore an elastic waist belt with a plastic plate (10 cm × 10 cm) that was positioned around the third lumbar vertebra (L3) on the subject's back. During the experimental trials, the laser beam was aimed at the plastic plate, and the distance between the plate and the laser sensor was measured. The subject stood on a force plate (Kistler, Switzerland) that recorded the fluctuation of the center of pressure (COP). While only the COM was used as sensory feedback, both COM and COP were used for stability analysis.

The laser measurements were sent to the PD controller, which calculated the required level of active ankle torque (see subsection *B: PD Control Kernel and Command Delivery*). After dividing the torque output into equal portions for each leg, an electrical stimulator (Compex Motion II, Compex Motion, Switzerland) provided the desired stimulation command (M_{STIM}) for both ankle extensors. The stimulation pulses had a constant frequency (f = 35 Hz) and pulse duration (Δp = 300 μs), and regulated the muscle contractions via amplitude variation (mA). Asymmetric, balanced biphasic pulses were applied during stimulation. Using these parameters, we empirically determined the stimulation amplitude as a function of torque (see subsection *C: Stimulation Amplitude as a Function of Torque*). The function was then

implemented in the real-time system and calculated the stimulation command M_{STIM} that was applied to the subject's plantar flexors in order to generate the required ankle torque. During the experiments, the anode (10 cm × 5 cm) was placed approximately two centimetres below the popliteal fossa with its horizontal center on the gastrocnemius muscle, while the cathode was located around the lower end of the gastrocnemius muscle belly. For safety reasons, the two inputs to the stimulator were optically isolated from the rest of the system, permitting only voltages up to 10 Volts to pass through (DSCA, Dataforth, USA).

B. PD Control Kernel and Command Delivery

Our real-time application implemented a PD controller with gains of Kp = 750 $Nm \cdot rad^{-1}$ and Kd = 350 $Nm \cdot s \cdot rad^{-1}$ that was theoretically capable of compensating for a neurological time delay of up to 135 ms [5]. Note that the gains were identified for a subject of average anthropometric parameters [5] and not specifically tuned for the subject of this study. This was due to the fact that the gain pairs proposed in [5] have passed both robustness and physiologic requirements and, hence, can be universally used for subjects with a wide range of anthropometric parameters. In order to ensure the validity of the 'universal PD controller' (Kp, Kd) = (750, 350) for this particular subject, we ensured its agreement with the empirically identified regression fit reported by Peterka [15].

The system was executed by a C++ based kernel (Visual C++ 5.0, Microsoft, USA) that communicated with an A/D and D/A converter at a frequency of 1000 Hz (PCI-MIO-16E-4, National Instruments, USA). Since the physical implementation of a PD controller enhances measurement noise in the sensory feedback loop [34], a third order Butterworth filter [35] with a cutoff frequency of 10 Hz was included.

Studies on human balance control have revealed that the tibialis anterior muscle rarely bursts during quiet standing as the COM is primarily located in front of the ankle joint, and

plantar flexing torque is continuously required to prevent the body from falling forward [22], [36]. Therefore, it was decided that only plantar flexors were subject to electrical stimulation in the present study.

C. Stimulation Amplitude as a Function of Torque

The stimulation amplitude versus torque function that is needed to translate each leg's control command into a respective level of stimulation was identified in the first preliminary experiment. This was done using a torque dynamometer (Biodex System 2, Biodex Medical Systems, USA) and the stimulator. The stimulation pulses were applied to the plantar flexors to determine the subject-specific relationship between the stimulation amplitude and the evoked ankle torque.

Fig. 2a shows the relation between the stimulation amplitude and the subject's ankle torque as measured in the preliminary experiment. For stimulation currents between 30 and 40 mA, the measurements revealed a rather linear relationship between the two variables (O). Due to the fact that stimulation currents lower than 30 mA did not reach the necessary threshold for reproducible tissue excitation, no ankle torque could be produced by these currents (Δ). We obtained the regression line from stimulation current to ankle torque using the stimulation range from 30 to 38 mA (Fig. 2a). Fig. 2b shows the stimulation amplitude as a function of torque obtained using the results of the preliminary experiment. For example, when the PD control kernel output generated a control command of 15 Nm per leg, the depicted function determined a stimulation command M_{STIM} of 34 mA that had to be delivered to the subject's plantar flexors. Note that the function's gradient range sufficiently covered the ankle torque fluctuation during quiet standing, which was reported to be approximately 15 to 20 Nm per leg [22].

D. Closed-Loop Time Delay

Due to the fact that we intended to keep the closed-loop time delay of the feedback system in the range of the neurological time delay (longer than 80 ms), the characteristic lag times of the experimental setup had to be investigated. They consisted of: 1) the information transmission time from the filtered COM measurement to the delivery of the calculated stimulation signal (τ_{TR}); 2) the electromechanical response time of the plantar flexors with respect to the stimulation (τ_{ST}); 3) the group delay implied by the Butterworth filter in the sensory feedback loop (τ_{BF}); and 4) the response time of the laser displacement sensor (τ_{LS}). Note that, for simplicity, these time delays were treated as constant delays, whereas the ones of the physiologic system may vary slightly.

The sum of τ_{TR} and τ_{ST} was determined in the second preliminary experiment that captured the time difference between a laser signal triggering the stimulation onset and the evoked ground reaction force as measured with the force plate. Note that we did not use the low pass filter in this experiment as to eliminate its effect on τ_{TR}. The applied stimulation current of 33 mA represented the mean of the theoretical stimulation range (≈ 28–38 mA) that provided the positive torque per ankle necessary for controlling balance during quiet stance. $\tau_{TR} + \tau_{ST}$ had an average value of 51.2 ± 11.2 ms (mean ± SD) that resulted from ten measurements using a randomized stimulation step. τ_{BF} depended on the characteristics of the applied Butterworth filter as well as the frequency spectrum of the COM fluctuation. Since frequencies up to 2 Hz dominated the spectrum of the COM signal, we determined the filter delay at the maximal frequency of 2 Hz (33 ms), and implemented it to approximate the constant time delay τ_{BF}. τ_{LS} was approximately 1 ms.

In equation (1), the system's closed-loop time delay τ_{CL} was estimated by combining the experimentally obtained values for $\tau_{TR} + \tau_{ST}$, τ_{BF}, and τ_{LS}.

$$\tau_{CL} = (\tau_{TR} + \tau_{ST}) + \tau_{BF} + \tau_{LS} = 51\,ms + 33\,ms + 1\,ms = 85\,ms \quad (1)$$

Since τ_{CL} was already in the range of the neurological time delay that the PD controller had to compensate in a physiologic manner, no additional time delay was needed.

E. Subject

The proposed system was tested with a subject who could not sufficiently regulate the active ankle torque due to a neurological disorder called von Hippel-Lindau Syndrome (VHL). VHL is a rare, genetic multi-system disorder characterized by the abnormal growth of tumors in certain parts of the body (*angiomatosis*). The tumors of the CNS are called *hemangioblastomas* and may develop in the brain, the retina of the eyes, and other areas of the nervous system. Symptoms of VHL vary among patients and depend on the size and location of the tumors. They may include headaches, problems with balance and walking, dizziness, weakness of the limbs, vision problems, and high blood pressure.

The male subject of the present study was 36 years of age, had height 173 cm, mass 59 kg, and suffered from VHL since birth. At the time of study, he had partial loss of sensation, proprioception and motor control caused by various hemangioblastomas in the cerebellum, the medulla, and the thoracic spinal cord (partially de-afferented and -efferented). As a result, he experienced balance problems and impaired gait, dizziness and significant muscle weakness in the legs. As the subject suffered under more severe neurological deficits and required two canes for walking, he was functioning at a Grade III level on the modified McCormick scale [37].

F. Stability Analysis

In order to determine whether the proposed system was capable of improving balance during quiet standing, the subject's performance was compared for three different treatments:

- NOstim: Natural performance without stimulation (0 mA)
- CONSTstim: Performance with constant stimulation (33 mA)
- PDstim: Performance with PD controlled stimulation (30 – 38 mA)

Note that the stimulation current used in CONSTstim was practically identical to the average stimulation current used during the PDstim treatments (33.9 mA).

In the literature, human balance during quiet standing has been characterized by analyzing the subject's COM and COP fluctuation. As such, the stability analysis of the present study focused on the behavior of these two time series. For each treatment, three trials of equal length were recorded. The order of the trial execution was randomized, whereas sufficient resting time of approximately ten minutes in between the trials was ensured to avoid accelerated fatiguing of the ankle muscles. During the 140 s of each trial, the subject was asked to stand still and maintain a balanced position with eyes open. The COM and COP recordings were logged at a sampling frequency of 1000 Hz and low-pass filtered using a fourth order, zero phase-lag Butterworth filter with a cutoff frequency of 5 Hz [35]. The latter 120 s of the recorded data were divided into two parts of 60 s each and analyzed by means of a one-way analysis of variance (ANOVA) with a significance level of $\alpha = 0.05$. The methodology of dividing the data into two equal parts for the analysis was chosen in agreement with Carpenter *et al.* [38], who have shown that a COP recording of 60 s is sufficient to capture the essential characteristics of human body sway.

In order to adequately characterize the performance for each treatment, the anterior-posterior fluctuations of the COM and COP were analyzed by means of measures of postural steadiness as suggested by Prieto *et al*. [39]:

I) Distance measures:

- the mean distance (MDIST)
- the root mean square distance (RDIST)
- the fluctuation range (RANGE)

II) Velocity measures:

- the mean velocity (MVELO)
- the root mean square velocity (RVELO)

Note that RVELO was the only measure not discussed by Prieto *et al.* [39]. The described measures were determined for each of the 60 s recordings and averaged for each treatment. Finally, we verified the validity of the applied ANOVA by examining the residuals for each measure with respect to the underlying assumptions.

G. Cross-Correlation Analysis

The subject's COM–M_{STIM} dynamics during the PDstim trials were compared with the ones of healthy individuals by means of cross-correlation analysis (CCF). Using the pre-processed COM and M_{STIM} data, two CCFs were calculated: 1) CCF between the COM position (COM_{POS}) and M_{STIM}; and 2) CCF between the COM velocity (COM_{VEL}) and M_{STIM}.

To calculate the CCFs, each of the 60 s long data sets was first divided into eight overlapping segments that were 2^{13} points, i.e., 8.192 s long. Then, a 13 bit FFT algorithm was applied before each segment's CCF was calculated. Finally, the CCFs were averaged for each trial and a single ensemble CCF was identified. Additionally, the group mean value and standard deviation of the time shifts were obtained for all trials.

The average time shifts from COM_{POS} to M_{STIM} and from COM_{VEL} to M_{STIM} were related to respective ranges of healthy subjects. According to Masani *et al.* [13], the time shift between COM_{POS} and the EMG of the right medial gastrocnemius muscle (M_{EMG}) was –155 ± 46 ms

(Mean ± SD). The CCF between COM_{VEL} and M_{EMG} had two peaks: one with a positive and one with a negative time shift. The positive time shift was 121 ± 134 ms and the negative time shift was –620 ± 134 ms [13]. Comparing the CCFs of the present study with these target ranges finally allowed us to evaluate whether the PD controller can generate body dynamics as seen in healthy individuals.

III. RESULTS

Fig. 3 shows examples of the COM fluctuation for three trials, each representing a different treatment. The dashed horizontal lines in each plot define the range of the mean ± 1 SD. A simple visual inspection of Fig. 3 suggests that the body sway in PDstim had a smaller magnitude than in NOstim and CONSTstim. The output generated by the PD controller in the PDstim trials did not enter the negative torque range or exceed the maximum permitted value of 26 Nm (38 mA) per leg at any time. Note that the average stimulation current for all PDstim trials was 33.9 ± 1.6 mA, whereas the constant stimulation level used in CONSTstim was 33 mA. Therefore, the PDstim and CONSTstim treatments delivered the same amount of charge to the muscles over time. The difference between the treatments was that during PDstim the intensity was dynamically regulated by the PD controller.

The marked 20 seconds of the PDstim treatment in Fig. 3 (black rectangle) are expanded in Fig. 4. Here, it can be clearly seen that the COM fluctuation (thick line) is closely related to the stimulation fluctuation (thin line) as determined by the PD controller. Also, we can observe that the controller effort stabilized the system by generating a stimulation command M_{STIM} that preceded the fluctuation of the COM. This observation was verified by CCF analysis, which revealed that M_{STIM} preceded the body sway fluctuation for all PDstim trials by 193 ± 21 ms.

Fig. 5a shows the average CCFs between COM_{POS} and M_{STIM} (left) and between COM_{VEL} and M_{STIM} (right) for each of the six PDstim recordings. The bold black curves indicate the group average CCFs for all recordings, whereas the gray vertical lines mark the target ranges for the respective time shifts obtained in [13]. It can be seen that the peaks of the average CCFs (one for COM_{POS}-M_{STIM} and two for COM_{VEL}-M_{STIM}) lie within the specified target ranges. For comparison, respective CCFs of a single able-bodied subject from [13] are shown in Fig. 5b (five trials of 30 s each).

Fig. 6 shows examples of the COP fluctuation in the anterior-posterior direction for each treatment. The dashed horizontal lines in each plot mark the range of the mean ± 1 SD. Similar to the COM fluctuation (see Fig. 3), the COP fluctuation in PDstim had a smaller magnitude than in NOstim and CONSTstim.

The results of the statistical analysis for both the COM and COP fluctuation are summarized in Table I. The PDstim trials had the smallest average value (bold font) for all COM measures, with differences between treatments being statistically significant (*) for all measures except RANGE. The COP analysis on the other hand revealed that the distance measures were smallest for PDstim, whereas the velocity measures were smallest for NOstim and largest for PDstim. The group difference was statistically significant for the COP distance but not the COP velocity measures.

IV. DISCUSSION

A. Adequacy of the Closed-Loop Time Delay of the Experimental System

The neurological time delay of the physiologic system has been estimated by identifying the characteristic time delays within the sensory-motor loop of healthy individuals. The feedback time delay τ_F has been reported to be in the range of 35.1 and 40.1 ms [40]. Applegate *et al.* [40] measured τ_F by recording the latency between the instant the sensory stimulation is provided to the foot and the instant the sensory evoked potential is recorded in the somatosensory area I of the brain. The electromechanical response time τ_E has been measured as 10.54 ms by Isabelle *et al.* [41] and 11.5 ms by Winter *et al.* [42]. In contrast to τ_F and τ_E, a valid estimate for the motor command time delay τ_M cannot be found in the literature. Although the neural-transmission time between the cortex and soleus muscle during quiet standing has been reported to be in the range of 27 and 36.5 ms [43], [44], the exact value of the time needed for the sensory-motor information process in the CNS is not known. However, using constant values for τ_F (40 ms), τ_E (10 ms) and the neural transmission component of τ_M (30 ms) [5], the neurological time delay can be assumed to be at least 80 ms. Since the experimental feedback circuit of the present study implemented a closed-loop time delay of 85 ms (equation (1)), we conclude that a similar closed-loop time delay has been introduced to the system as in the physiologic case. It should also be noted that the experimental time delay of 85 ms is in the range of 80 to 135 ms for which the PD controller with gains of Kp = 750 $Nm \cdot rad^{-1}$ and Kd = 350 $Nm \cdot s \cdot rad^{-1}$ generated physiologic system dynamics in the simulations [5].

B. Practical Capability of the Feedback System to Regulate the Active Control Torque

The results of Prieto *et al.* [39] imply that multiple COP measures are necessary to adequately characterize performance differences between young and elderly adults during standing. It can be hypothesized that a similar variety of measures is needed to evaluate the

balance abilities of impaired subjects during quiet stance. Using the described measures, the analysis of the subject's standing performance revealed that all COM measures as well as the COP distance measures were smallest for the PDstim treatment (Table I). Since the differences between treatments were statistically significant for seven out of eight of these measures, it can be concluded that body sway has evidently been reduced during PDstim in comparison with the other two treatments. As such, the feedback control system coupled with an FES system represents an effective solution for improving balance during standing in subjects with certain neuromuscular disorders.

While the outcome for the COM measures directly translates to the subject's level of stability, the results for the COP distance and velocity measures require further explanation. COP distance measures have been related to the stability achieved by the postural control system; and COP velocity measures have been related to the amount of regulatory activity associated with this level of stability [45], [46]. Maki *et al.* [46] argued that subjects with high values for the velocity measures may be quite stable, in the sense that the COP does not approach the limits of the base of support, but may require frequent postural corrections to achieve this degree of stability. If this interpretation is correct, increased COP velocity measures may be indicative of a compensation for some underlying neural or sensorimotor dysfunction. Also Prieto *et al.* [39] hypothesized that in their experiments the elderly may have attained a similar level of steadiness as the young subjects (equal distance measures), but that the elderly may have required significantly more postural control activity to achieve this level of steadiness (higher velocity measures). In accordance with these implications, it can be concluded in the present study that, due to the lower COP distance measures during PDstim (Table I), the postural control system is achieving a higher level of stability for this treatment. The higher COP velocity measures on the

other hand signal a higher level of regulatory activity during PDstim as evoked by the implemented PD controller.

It was also observed that the CONSTstim treatment showed smaller values than the NOstim treatment for four out of ten measures. Since constant stimulation increases the level of stiffness around the ankle joint by providing additional muscle tonus, it improved the subject's balance to a certain extent. The constant stimulation is a type of open-loop control strategy mimicking the increase in muscle stiffness, which is fixed a priori. Indeed, Winter *et al.* [19] suggested that the muscle stiffness is set a priori depending on the task, such as a wide or narrow stance. However, the present results imply that a considerable part of the stability improvement for the PDstim treatment was not achieved by the increased passive torque but the dynamic activity induced by the PD controller. Therefore, the comparison of the CONSTstim and PDstim treatments emphasizes the role of the active torque component during balance control. We conclude that the implemented PD controller is capable of effectively improving balance during quiet stance. Namely, this outcome implies that a feedback control strategy is, in fact, a potential candidate for balance regulation even when long closed-loop time delays have to be compensated for.

C. Characterization of System Dynamics

Since the experimental feedback system included a closed-loop time delay of physiologic order (85 ms), the preceding time of the stimulation command M_{STIM} with respect to the body sway is a meaningful measure for evaluating the controller's ability to generate system dynamics that mimic the ones of the physiologic control system. On the one hand, experiments with able-bodied subjects have shown that M_{EMG} preceded the COM fluctuation by 155 ± 46 ms during quiet standing [13]. On the other hand, CCF analysis in the present study revealed that M_{STIM} preceded body sway by 193 ms during the PDstim trials. As such, the preceding time of the

stimulation command for the subject in the present study is not only within the specified target range (155 ± 46 ms), but is fairly large in comparison with healthy individuals. This is an important result since it shows that the PD controller is evidently capable of providing a motor command that sufficiently precedes body sway in spite of the closed-loop time delay.

Properties of body sway other than the phase advance of the motor command can be captured by means of CCF between the COM_{VEL} and M_{STIM}. Changes in body dynamics have an influence on the shape of the CCF between COM_{POS} and M_{STIM}, which in turn affects the peaks of the CCF between COM_{VEL} and M_{STIM}. Since these peaks were within the specified target ranges, M_{STIM} generated dynamics as seen in healthy subjects. Moreover, since a right shift of the CCF functions in Fig. 5a up to 90 ms would still keep all group peaks within the target ranges, it can be assumed that a closed-loop time delay of up to 85 + 90 ms will result in a system with physiologic dynamics.

D. Limitations of the Performed Study

The main goal of the present case study was to experimentally test our hypothesis that the PD control strategy can: 1) improve balance during quiet standing in spite of long neurological time delays; and 2) generate quiet standing dynamics as in healthy individuals. In what follows we are discussing the limitations of this study:

1) Presence of Natural Balance Control: Although the subject who participated in this study was partially de-afferented and -efferented, he still had some residual motor activity. Therefore, it has to be assumed that the subject applied a low, but inconsistent level of voluntary, but unregulated ankle muscle contractions during the experiments. Additionally, intact sensory systems such as neck proprioception, the vestibular and visual modalities contributed to the body stabilization.

Nevertheless, due to the impairment of the *efferent* motor channels, the ankle muscle contractions are still assumed to be unregulated in the case where these sensory channels are still functional. The fact that the subject's lower-extremity impairment was much higher than his upper-extremity impairment supports this assumption: He had to use two canes for walking, but had no significant difficulties with controlling upper-extremity movement (McCormick Grade III). Since our study only focused on the control of the ankle joint, we believe that the choice of the subject was appropriate for the experiments presented in this study.

2) Application of Positive Torque Component Only: In our experiments, the FES system was used to generate contractions of the plantar flexors only, and not of both the plantar flexors and the tibialis anterior muscle. Since the COM is naturally located in front of the ankle joints during quiet stance, a torque produced by the tibialis anterior muscle is rarely used to regulate balance during quiet stance. Instead, the torque around the ankle joints is almost entirely generated by the plantar flexors alone [36]. Therefore, we have decided to use only torques in our stability experiments that are generated by the plantar flexors. Nevertheless, future experiments should be performed in which the plantar flexors and the tibialis anterior muscle are controlled simultaneously.

V. CONCLUSION

The findings of the performed case study demonstrate that a PD controller in combination with an FES system can be applied to augment stability in disabled individuals during quiet standing. A velocity-accentuated PD controller can compensate for a long neurological time delay and generate system dynamics that accurately mimic those observed in able-bodied subjects during quiet standing. While these findings do not imply that the CNS actually applies a PD-like control strategy to regulate balance, one cannot ignore the possibility that it is at least theoretically possible.

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REFERENCES

[1] P. G. Morasso and M. Schieppati, "Can muscle stiffness alone stabilize upright standing?," *J. Neurophysiol.,* vol. 83, pp. 1622-1626, Sept. 1999.

[2] P. G. Morasso and V. Sanguineti, "Ankle muscle stiffness alone cannot stabilize balance during quiet standing," *J. Neurophysiol.*, vol. 88, pp. 2157-2162, Oct. 2002.

[3] I. D. Loram and M. Lakie, "Direct measurement of human ankle stiffness during quiet standing: the intrinsic mechanical stiffness is insufficient for stability," *J. Physiol.*, vol. 545, pp. 1041-1053, Dec. 2002.

[4] M. Casadio, P. G. Morasso, and V. Sanguineti, "Direct measurement of ankle stiffness during quiet standing: implications for control modeling and clinical application," *Gait and Posture*, vol. 21, pp. 410-424, June 2005.

[5] K. Masani, A. H. Vette, and M. R. Popovic, "Controlling balance during quiet standing: Proportional and derivative controller generates preceding motor command to body sway position observed in experiments," *Gait and Posture,* vol. 23, pp. 164-172, Feb. 2006.

[6] R. Fitzpatrick and D. I. McCloskey, "Proprioceptive, visual and vestibular thresholds for the perception of sway during standing in humans," *J. Physiol.*, vol. 478, pp. 173-186, July 1994.

[7] R. Fitzpatrick, D. K. Rogers, and D. I. McCloskey, "Stable human standing with lower-limb muscle afferents providing the only sensory input," *J. Physiol.*, vol. 480, pp. 395-403, Oct. 1994.

[8] I. D. Loram, C. N. Maganaris, and M. Lakie, "Paradoxical muscle movement in human standing," *J. Physiol.*, vol. 556, pp. 683-689, May 2004.

[9] I. D. Loram, C. N. Maganaris, and M. Lakie, "Active, non-spring-like muscle movements in human postural sway: how might paradoxical changes in muscle length be produced?," *J. Physiol.*, vol. 564, pp. 281-293, Apr. 2005.

[10] I. D. Loram, C. N. Maganaris, and M. Lakie, "Human postural sway results from frequent, ballistic bias impulses by soleus and gastrocnemius," *J. Physiol.*, vol. 564, pp. 295-311, Apr. 2005.

[11] R. Fitzpatrick, D. Burke, and S. C. Gandevia, "Loop gain of reflexes controlling human standing measured with the use of postural and vestibular disturbances," *J. Neurophysiol.*, vol. 76, pp. 3994-4008, Dec. 1996.

[12] P. Gatev, S. Thomas, T. Kepple, and M. Halett, "Feedforward ankle strategy of balance during quiet stance in adults," *J. Physiol.*, vol. 514, pp. 915-928, Feb. 1999.

[13] K. Masani, M. R. Popovic, K. Nakazawa, M. Kouzaki, and D. Nozaki, "Importance of body sway velocity information in controlling ankle extensor activities during quiet stance," *J. Neurophysiol.*, vol. 90, pp. 3774-3782, Dec. 2003.

[14] A. M. De Nunzio, A. Nardone, and M. Schieppati, "Head stabilization on a continuously oscillating platform: The effect of a proprioceptive disturbance on the balancing strategy," *Exp. Brain Res.*, vol. 165, pp. 261-272, Aug. 2005.

[15] R. J. Peterka, "Sensorimotor integration in human postural control," *J. Neurophysiol.*, vol. 88, pp. 1097-1118, Sept. 2002.

[16] R. J. Peterka, "Posture Control: Simplifying the complexities of maintaining balance. Insights provided by simple closed-loop models of human postural control," *IEEE Eng. Med. Biol. Mag.*, vol. 22, pp. 63-68, Mar. 2003.

[17] S. Grillner, "The role of muscle stiffness in meeting the changing postural and locomotor requirements for force development by the ankle extensors," *Acta Physiol. Scand.*, vol. 86, pp. 92-108, Sept. 1972.

[18] V. S. Gurfinkel, M. I. Lipshits, and K. Y. Popov, "Is the stretch reflex the main mechanism in the system of regulation of the vertical posture of man?," *Biophysics*, vol. 19, pp. 761-766, 1974.

[19] D. A. Winter, A. E. Patla, F. Prince, M. Ishac, and K. Gielo-Perczak, "Stiffness control of balance in quiet standing," *J. Neurophysiol.*, vol. 80, pp. 1211-1221, Sept. 1998.

[20] P. G. Morasso, L. Baratto, R. Capra, and G. Spada, "Internal models in the control of posture," *Neural Networks*, vol. 12, pp. 1173-1180, Oct. 1999.

[21] D. A. Winter, A. E. Patla, S. Rietdyk, and M. G. Ishac, "Ankle muscle stiffness in the control of balance during quiet standing," *J. Neurophysiol.*, vol. 85, pp.2630-2633, June 2001.

[22] I. D. Loram and M. Lakie, "Human balancing of an inverted pendulum: Position control by small, ballistic-like, throw and catch movements," *J. Physiol.*, vol. 540, pp. 1111-1124, May 2002.

[23] D. A. Winter, A. E. Patla, M. Ishac, and W. H. Gage, "Motor mechanisms of balance during quiet standing," *J. Electromyogr. Kinesiol.*, vol. 13, pp. 49-56, Feb. 2003.

[24] Y. Fukuoka, T. Nagata, A. Ishida, and H. Minamitani, "Characteristics of somatosensory feedback in postural control during standing," *IEEE Trans. Neural Sys. Rehab. Eng.*, vol. 9, pp. 145-153, June 2001.

[25] J. Jeka, T. Kiemel, R. Creath, F. Horak, and R. Peterka, "Controlling human upright posture: Velocity information is more accurate than position or acceleration," *J. Neurophysiol.*, vol. 92, pp. 2368-2379, Oct. 2004.

[26] Z. Matjačić and T. Bajd, "Arm free paraplegic standing: Part I – Control model synthesis and simulation," *IEEE Trans. Rehab. Eng.*, vol. 6, pp. 125-138, June 1998.

[27] Z. Matjačić and T. Bajd, "Arm free paraplegic standing: Part II – Experimental results," *IEEE Trans. Rehab. Eng.*, vol. 6, pp. 139-150, June 1998.

[28] R.-P. Jaime, Z. Matjačić, and K. J. Hunt, "Paraplegic standing supported by FES-controlled ankle stiffness," *IEEE Trans. Neural Sys. Rehab. Eng.*, vol. 10, pp. 239-248, Dec. 2002.

[29] K. J. Hunt, H. Gollee, and R. P. Jaime, "Control of paraplegic ankle joint stiffness using FES while standing," *Med. Eng. Phys.*, vol. 23, pp. 541-555, Oct. 2001.

[30] W. Holderbaum, K. J. Hunt, and H. Gollee, "H_∞ robust control design for unsupported paraplegic standing: experimental evaluation," *Control Eng. Practice*, vol. 10, pp. 1211-1222, Nov. 2002.

[31] H. Gollee, K. J. Hunt, and D. E. Wood, "New results in feedback control of unsupported standing in paraplegia," *IEEE Trans. Neural Sys. Rehab. Eng.*, vol. 12, pp. 73-80, Mar. 2004.

[32] J. J. Abbas and H. J. Chizeck, "Feedback control of coronal plane hip angle in paraplegic subjects using functional neuromuscular stimulation," *IEEE Trans. Biomed. Eng.*, vol. 38, pp. 687-698, July 1991.

[33] W. H. Gage, D. A. Winter, J. S. Frank, and A. L. Adkin, "Kinematic and kinetic validity of the inverted pendulum model in quiet standing," *Gait and Posture*, vol. 19, pp. 124-132, Apr. 2004.

[34] Z. Matjačić, K. Hunt, H. Gollee, and T. Sinkjaer, "Control of posture with FES systems," *Med. Eng. Phys.*, vol. 25, pp. 51-62, Jan. 2003.

[35] D. A. Winter, *Biomechanics and motor control of human movement.* Toronto, ON: John Wiley & Sons Inc., 1990, pp. 36-41.

[36] J. W. Smith, "The forces operating at the human ankle joint during standing," *J. Anat.*, vol. 91, pp. 545-564, Oct. 1957.

[37] V. Van Velthofen, P. C. Reinacher, J. Klisch, H. P. Neumann, S. Gläsker, "Treatment of intramedullary hemangioblastomas, with special attention to von Hippel-Lindau disease," *J. Neurosurg.*, vol. 53, pp. 1306-1314, Dec. 2003.

[38] M. G. Carpenter, J. S. Frank, D. A. Winter, and G. W. Peysar, "Sampling duration effects on centre of pressure summary measures," *Gait and Posture*, vol. 13, pp. 35-40, Feb. 2001.

[39] T. E. Prieto, J. B. Myklebust, R. G. Hoffmann, E. G. Lovett, and B. M. Myklebust, "Measures of postural steadiness: Differences between healthy young and elderly adults," *IEEE Trans. Biomed. Eng.*, vol. 43, pp. 956-966, Sept. 1996.

[40] C. Applegate, S. C. Gandevia, and D. Burke, "Changes in muscle and cutaneous cerebral potentials during standing," *Exp. Brain Res.*, vol. 71, pp. 183-188, June 1988.

[41] M. Isabelle, Q.-B. Sylvie, and P. Chantal, "Electromechanical assessment of ankle stability," *Eur. J. Appl. Physiol.*, vol. 88, pp. 558-564, Feb. 2003.

[42] E. M. Winter and F. B. Brookes, "Electromechanical response times and muscle elasticity in men and women," *Eur. J. Appl. Physiol. Occup. Physiol.*, vol. 63, pp. 124-128, Aug. 1991.

[43] H. Ackermann, E. Scholz, W. Koehler, and J. Dichgans, "Influence of posture and voluntary background contraction upon compound muscle action potentials from anterior tibial and soleus muscle following transcranial magnetic stimulation," *Electroenceph. Clin. Neurophysiol.*, vol. 81, pp. 71-80, Feb. 1991.

[44] B. A. Lavoie, F. W. J. Cody, and C. Capaday, "Cortical control of human soleus muscle during volitional and postural activities studied using focal magnetic stimulation," *Exp. Brain Res.*, vol. 103, pp. 97-107, Jan. 1995.

[45] A. Hufschmidt, J. Dichgans, K. H. Mauritz, and M. Hufschmidt, "Some methods and parameters of body sway quantification and their neurological applications," *Arch. Psychiur. Nervenkr.,* vol. 228, pp. 135-150, May 1980.

[46] B. E. Maki, P. J. Holliday, and G. R. Fernie, "Aging and postural control: A comparison of spontaneous- and induced-sway balance tests," *J. Amer. Geriatr. Soc.,* vol. 38, pp. 1-9, Jan. 1990.

TABLE I. COMPARISON OF AVERAGE STABILITY RESULTS FOR EACH TREATMENT (MEAN ± SD)

	COM	**NOstim**	**CONSTstim**	**PDstim**
I	$MDIST_{AP}$* [cm]	0.832 ± 0.166	1.052 ± 0.243	**0.690** ± **0.154**
	$RDIST_{AP}$* [cm]	1.083 ± 0.231	1.253 ± 0.269	**0.871** ± **0.217**
	$RANGE_{AP}$ [cm]	6.003 ± 1.382	5.701 ± 1.194	**4.498** ± **1.030**
II	$MVELO_{AP}$* [cm/s]	0.939 ± 0.115	0.882 ± 0.102	**0.776** ± **0.086**
	$RVELO_{AP}$* [cm/s]	1.237 ± 0.152	1.131 ± 0.136	**1.019** ± **0.091**
	COP	**NOstim**	**CONSTstim**	**PDstim**
I	$MDIST_{AP}$* [cm]	1.009 ± 0.136	1.158 ± 0.229	**0.857** ± **0.126**
	$RDIST_{AP}$* [cm]	1.308 ± 0.181	1.423 ± 0.270	**1.095** ± **0.169**
	$RANGE_{AP}$* [cm]	8.399 ± 0.898	7.767 ± 1.390	**6.723** ± **0.862**
II	$MVELO_{AP}$ [cm/s]	**2.607** ± **0.444**	2.691 ± 0.478	2.759 ± 0.740
	$RVELO_{AP}$ [cm/s]	**3.542** ± **0.550**	3.582 ± 0.608	3.761 ± 1.099

I) represents distance measures, II) represents velocity measures.

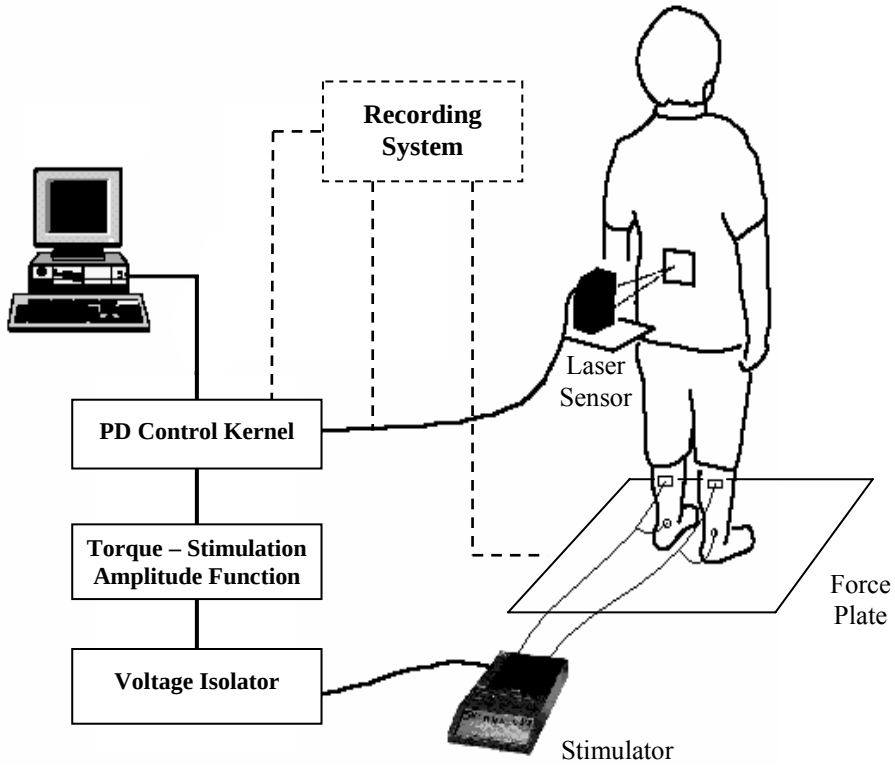

Fig. 1. Schematic of the experimental setup. The laser measurements representing the fluctuation of spontaneous body sway were sent to the controller, which determined the level of active ankle torque that was needed to stabilize the system.

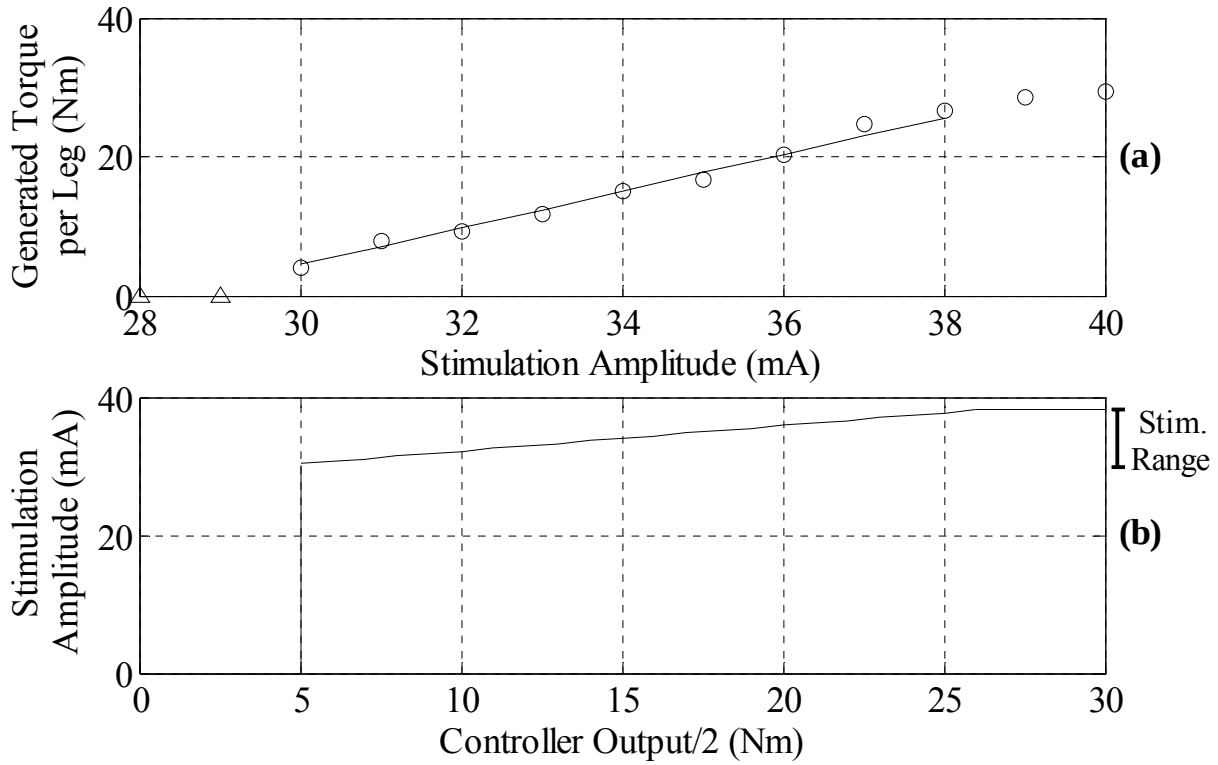

Fig. 2. Result of the first preliminary experiment (a), and the stimulation amplitude as a function of torque (b). Fig. 2a depicts the relation between the stimulation current and generated ankle torque as determined in the preliminary experiment with the subject. Fig. 2b shows the function from the controller output to the stimulation level as implemented in the PDstim trials.

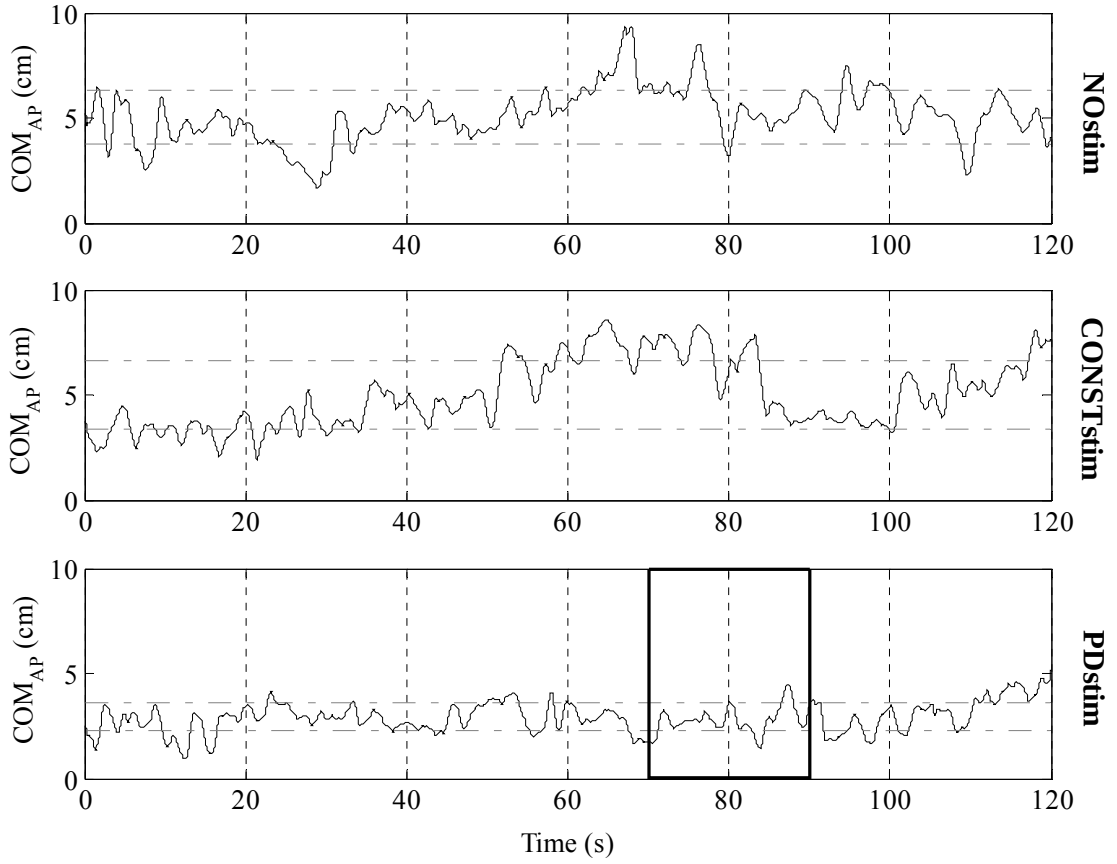

Fig. 3. Fluctuation of COM position during quiet stance. COM fluctuation without stimulation (NOstim), COM fluctuation with constant stimulation (CONSTstim), and COM fluctuation with controlled stimulation (PDstim). The dashed horizontal lines in each plot define the range of the mean ± 1 SD. The black rectangle in PDstim marks an excerpt that is also shown in Fig. 4.

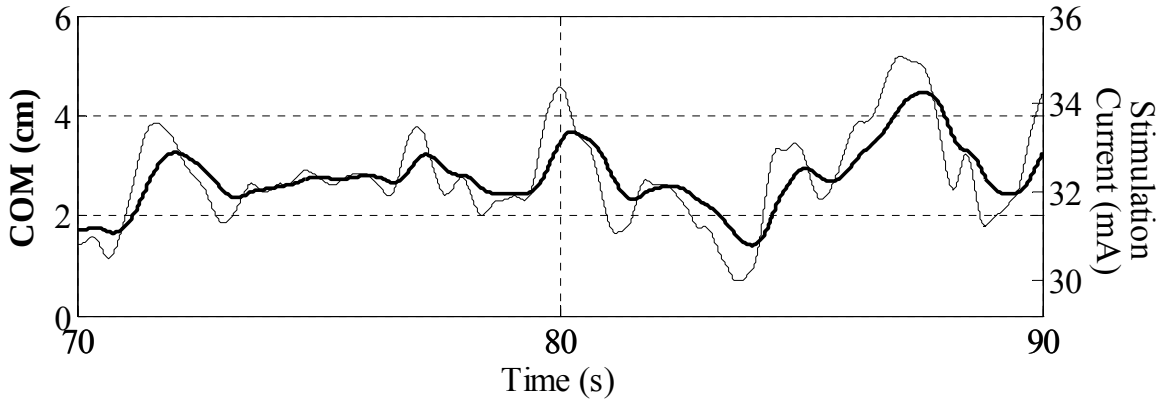

Fig. 4. Excerpt from the COM fluctuation in PDstim (Fig. 3), related to the PD controlled level of stimulation current (M_{STIM}). The stimulation fluctuation (thin line) preceded the COM fluctuation (thick line).

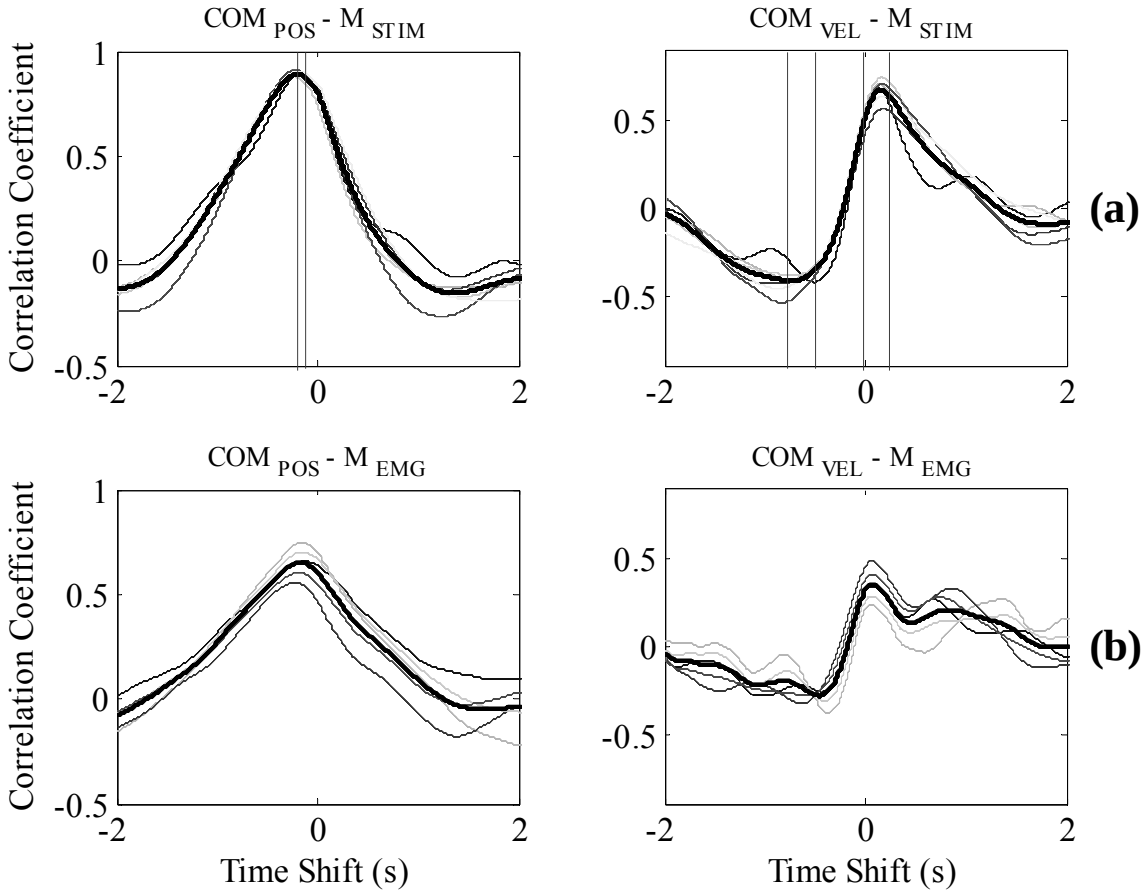

Fig. 5. Cross-correlation comparison: (a) CCFs between COM_{POS} and M_{STIM}, and between COM_{VEL} and M_{STIM} for the PDstim recordings. The bold black curves indicate the group average CCFs for all six PDstim recordings, whereas the gray vertical lines mark the target ranges for the respective time shifts; (b) CCFs between COM_{POS} and M_{EMG}, and between COM_{VEL} and M_{EMG} for one able-bodied subject (5 trials of 30 s) [13].

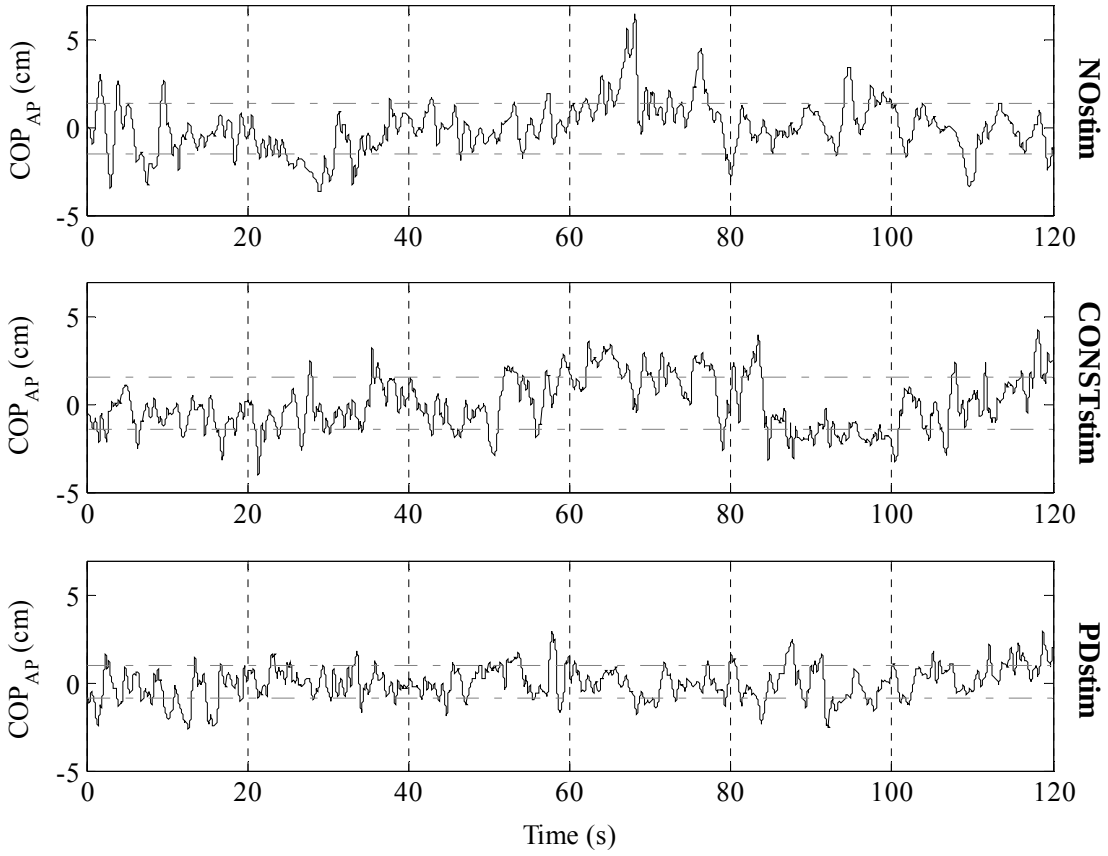

Fig. 6. Fluctuation of COP position during quiet stance. COP fluctuation without stimulation (NOstim), COP fluctuation with constant stimulation (CONSTstim), and COP fluctuation with controlled stimulation (PDstim). The dashed horizontal lines in each plot define the range of the mean ± 1 SD.