

ANTICIPATION MECHANISM IN BODY SWAY CONTROL AND EFFECT OF MUSCLE FATIGUE

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Resumo: O objetivo deste trabalho foi quantificar a ocorrência do mecanismo antecipatório no controle do equilíbrio postural ortostático por meio de medição do atraso entre a atividade mioelétrica do gastrocnêmio lateral e o sinal estabilométrico, bem como determinar a influência da fadiga muscular neste processo. Os sinais estabilométricos e eletromiográficos (EMG) foram coletados de forma sincronizada a partir de 22 sujeitos. A fadiga do gastrocnêmio foi induzida por uma flexão plantar sustentada até a falência muscular. A aquisição dos sinais foi realizada por 120 s antes e após a fadiga induzida. Após a remoção da média, a raiz quadrada do valor médio quadrático do EMG (RMS-EMG) foi calculada por períodos de 20 ms. A função de correlação cruzada normalizada foi calculada para estimar o atraso temporal entre os sinais RMS-EMG e os estabilogramas. Tempos de antecipação de até 1,62 s foram encontrados antes e após a fadiga ($p < 0,05$), indicando que este mecanismo tem uma importante função no controle das oscilações posturais. A fadiga causou um aumento significativo da latência entre a atividade mioelétrica do gastrocnêmio e os movimentos do centro de pressão dos pés ($p < 0,05$).

Palavras-chave: Antecipação, Oscilações Posturais, Função de Correlação-cruzada, Fadiga, Eletromiografia.

Abstract: The aim of this work is to quantify the occurrence of an anticipatory mechanism in the control of quiet standing by measuring the lag between the myoelectric activity of the lateral gastrocnemius muscle and the stabilometric signal, as well as to determine the influence of the muscle fatigue on this process. Stabilometric and electromyographic (EMG) signals were synchronously collected from 22 subjects. Gastrocnemius fatigue was induced by a sustained plantar flexed posture until muscle failure. The data acquisition lasted for 120 s before and after the induced fatigue. After mean removal, the root mean square values of the EMG (RMS-EMG) were calculated for each 20 ms period. The normalized cross-correlation function was estimated to find the time delay between RMS-EMG and stabilometric signals. Anticipation values up to 1.62 s were found both before and after fatigue conditions ($p < 0.05$), indicating that this mechanism plays an important role in body sway control. The fatigue caused a significant increase in the latency between the myoelectric activity of the gastrocnemius muscle and the movements of the center of pressure ($p < 0.05$).

Keywords: Anticipation, Body sway, Cross-correlation Function, Fatigue, Electromyography.

Introduction

The maintenance of the orthostatic posture of the human body involves the integration of different control mechanisms, where the static and dynamic motor ability depends on a set of complex processes at medullary level, including postural tonic reflexes to maintain the ankle stiffness [42,43] and superior levels of the central nervous system (CNS) [16,19,22,26-28]. While standing, the body sways in all directions with variable muscular activity regulated by discrete neural stimuli [16] to maintain the projection of the center of mass (COM) within the limits of the support base. According to the stiffness theory [41-43], the postural control system is passive, with adjustments of muscular tension independent of the sensory inputs. These authors consider that ankle muscles control the anterior-posterior displacements by setting the stiffness, and support this hypothesis with the inverted pendulum model, by showing that COM and center of pressure (COP) signals are in-phase with a strong correlation between the acceleration of the COM and COM-COP difference. However, Morasso and Schieppati [28] disagree with ankle stiffness theory, as the sole explanation of the lack of time delay between COM and COP, pointing out that a delay in feedback loop would cause a global destabilization in this system. Using real data in a model, Fitzpatrick et al. [11] observed that a feedback mechanism is not sufficient to explain control of body sway. In accordance with their model, when removing the feedback the oscillations rise by a factor of two, which is not sufficient to affect the orthostatic posture. As patients with sensory dysfunction generally show postural balance difficulties, these authors suggested that sensory information participate in the postural control in addition to the feedback. Other theoretical [27] and experimental [20] studies confirm that stiffness contributes to balance control but isn't the only mechanism.

Additionally, the CNS recognizes movements, foreseeing imminent disturbances and commanding anticipatory muscle actions to minimize their effects [22,26-28,34]. Evidence of the brain's ability to predict movements and anticipate the appropriate task are being pointed out by various studies with the help of surface electromyography (EMG), e.g. relating the synergism of antigravitational muscles and the agonists of arm movements [1,2,4,8]. The anticipatory control of postural adjustments also have been studied with perturbations applied directly to the subject or to its base of support [1,4], resulting in anticipations from 50 to 100 ms for different muscles. During shoulder flexion or the task of pulling a rigid cable, some synergist postural muscles of the trunk and the leg are activated in anticipation of about 90 ms [4], likely with the objective of limiting the anterior sway related with the displacement of the COM generated by this movement.

Regarding the control of standing posture, Gatev et al. [13] observed peaks of myoelectric activity in the gastrocnemius muscle between 200 and 270 ms before the anterior-posterior COP movements, measured by the stabilograms. Moreover, Loram et al. [22] applied a cross-correlation function to show that α motor neuron activity is modulated approximately 160 ± 50 ms ahead of the muscle length modification in standing, with anterior-posterior movements of body in antiphase with muscle length alterations, thus suggesting anticipation as a mechanism of the body sway control. Thus, the anticipatory activity seems to play a relevant role in body sway control, which must be better investigated.

Muscle fatigue of the limbs is a well known factor that affects the postural control by increasing the body sway [37-39]. Nardone et al. [29,30] observed increased area of the COP and length of the sway path after fatigue. Additionally, Allison and Henry [1] observed that trunk muscle responses to sudden arm movements are affected by fatigue, requiring increased latency of the anticipatory postural adjustment. This leads to the hypothesis that anticipation on the postural control should also be increased by fatigue.

The purpose of this work is to quantify the occurrence of an anticipatory mechanism in quiet standing control by measuring the lag between the myoelectric activity of the lateral gastrocnemius muscle and the stabilometric signal, as well as verifying the influence of a muscle fatigue on this process.

Materials and Methods

Subjects

The sample was comprised of 22 subjects (15 males and 7 females), undergraduate students of the Physical Education School, with age 23.2 ± 3.6 years (mean \pm standard deviation), body mass 70.6 ± 10.9 kg and height 169.9 ± 7.0 cm, with no history of neurological disorders or orthopedic diseases. All subjects were voluntary and signed a free informed consent before inclusion in the study.

Stabilometric Data Recording

A three-point vertical force platform, developed by the authors [31] according to the specifications of the Association Française de Posturologie [3], was used for collecting stabilometric data. The three force-cells were disposed at the corners of an equilateral triangle with 40 cm side length. The signals from each force-cell were passed through a differential amplifier (adjustable gain, common mode rejection 120 dB at 60 Hz and input impedance 1 T Ω) with a second order anti-aliasing Butterwoth filter with 5 Hz cutoff frequency, and thus sampled at a rate of 50 Hz.

Data from the force platform were collected by a data acquisition board DacPad 1200 (National Instruments, Austin, USA), 12 bits resolution and dynamic range of ± 5 V, and stored in a personal computer.

Surface EMG Recording

Electromyographic signals were collected by superficial disposable Ag/AgCl electrodes (spherical, 10 mm diameter) Kendall MEDI-TRACE 2000 (The Ludlow Co., Chicopee, USA). The electrodes were fixed on the lateral head of the right gastrocnemius muscle, at one fourth of the distance between the fibula head and the calcaneus bone. Electrodes were placed following the lateral gastrocnemius fibers direction with 35 mm inter-electrodes distance with the reference electrode placed on the right lateral malleolus. The skin was prepared by shaving the hair, abrasion with sponge and alcohol cleansing.

The EMG amplifier (Biovision, Wehrheim, Germany) have a differential input, with gains 1000 and 5000, 120 dB common mode rejection, 1 T Ω input impedance, and is band limited between 10 and 1 kHz. After amplification, EMG signal was filtered by a first order anti-aliasing Butterwoth filter with 500 Hz cutoff frequency.

The EMG was digitized at 1 kHz through a data acquisition board DacCard 700 (National Instruments, Austin, USA), with 12 bits resolution and dynamic range of ± 5 V, and stored for further analysis.

Software

The data acquisition software was built using Labview, version 5.0 (National Instruments, Austin, USA). All data analysis was performed off-line using Matlab, version 6.5 (The Mathworks, Natick, USA)

Experimental Protocol

Each subject was oriented to stay on the force platform, with the feet together and the arms relaxed. After 1 min of adaptation, stabilometric data was collected for 120 s. This

recording time was chosen according to previous recommendation [5], to effectively recognize the characteristic low frequency spectral components of this signal and avoid their recognition as non-stationarity in short time tests. At the beginning of stabilometric data acquisition, a pulse of synchronism is generated to trigger the beginning of the EMG recording, with gain 5000.

After the first collection phase, the subject was asked to perform a maximum plantar flexion contraction and maintain this position as much as he could until muscle failure, keeping the joint angle at its maximal amplitude, as proposed by Vuillerme et al. [39]. A metal stem was fixed on the floor, in front of the platform, in the direction of the individual's right hand to prevent falls during plantar flexion position. Subjects were oriented to put the right hand opened just in front of the stem, and to touch it only when necessary to maintain the equilibrium, but not using it as additional support. Only EMG was recorded during fatigue induction, with gain 1000 to avoid signal saturation.

After muscle failure, the subject returned to the initial (resting) position for another 120 s period of acquisition of both EMG (gain 5000) and stabilometric data.

Data Processing

The coordinates of the COP were calculated from the net force moments of the platform as follows:

$$x = \frac{F_2 \cdot l + F_3 \cdot (l/2)}{F_p} \quad (1)$$

$$y = \frac{F_3 \cdot l \cdot \sqrt{3}/2}{F_p} \quad (2)$$

where x and y correspond to mediolateral and anterior-posterior COP position, respectively, $l = 40$ cm, F_i are the forces at the force-cells i , being 1 and 2 the force cells in the mediolateral directions and 3 the one in front of the subject, and $F_p = F_1 + F_2 + F_3$.

The digitized EMG signals were band limited by a Butterworth filter, with cutoff frequencies of 10 and 300 Hz, each order 5, and five pairs of pole-zero (notch, adjusted to 2 Hz bandwidth) at 60 Hz and harmonics up to 300 Hz, to cut off mains noise which otherwise corrupts the low-amplitude EMG data. This filter was projected as the convolution of the coefficients of the seven digital filters, and applied in direct and reverse directions to avoid phase shifts. The root mean square values (RMS) of the EMG signals were calculated at windows of 20 ms to match the sampling rate with the stabilometric signals (50 Hz), as follows:

$$r[n] = \sqrt{\frac{1}{20} \cdot \sum_{i=(n-1)20-9}^{(n-1)20+10} (emg[i])^2} \quad (3)$$

where $r[n]$ is the RMS-EMG data and $emg[i]$ is the raw EMG data samples after mean removal.

To avoid a phase-drift between the $r[n]$ signal and respective stabilograms, the first RMS value was calculated over a window of 10 ms (10 samples), as follows:

$$r[1] = \sqrt{\frac{1}{10} \cdot \sum_{i=1}^{10} (emg[i])^2} \quad (4)$$

To quantify the delay between $r[n]$ and stabilograms, the normalized cross-correlation function (NCCF) [33] was estimated both before and after fatigue conditions, in anterior-posterior $y[n]$ (NCCF- y) and mediolateral $x[n]$ (NCCF- x) directions. The maximum value of the cross-correlation and the corresponding time lag were extracted from the NCCF- y and NCCF- x of each individual. This time lag was considered to be an estimate of the latency among signals, reflecting the average time between changes in EMG and corresponding COP displacement.

For monitoring the development of the fatigue, spectral median frequency (f_{median}) was estimated for the raw EMG collected during the isometric plantar flexion. The f_{median} was defined

as the frequency at which the power spectrum is divided into two regions with equal power [35]. This parameter was estimated for each 1 s epoch, starting 5 s after plantar flexion and finishing 5 s before this end, to avoid the non-stationarities due to ankle movements. Each power spectral density was estimated by a single periodogram with rectangular window and mean removal, by squaring the respective discrete Fourier transform (DFT), a procedure considered adequate by Farina and Merletti [10].

The area of COP displacement (*area*) [32] and velocities of anterior-posterior (*y-vel*) and mediolateral (*x-vel*) stabilograms were measured before and after fatigue to quantify the occurrence of stabilometric changes due to fatigue. All variables were calculated for the first 30 s of each test.

Statistical Analysis

Student's t -test was applied to test the presence of delays as well as the differences between the conditions before and after fatigue ($\alpha = 0.05$).

Monte-Carlo simulation [23] was applied to determine the critical value of the cross-correlation function, by simulating an ensemble of 1000 EMG and stabilograms signals pairs with equal spectral magnitude and random odd phase with uniform probability density function in the range from $-\pi$ to π . The simulated $r[n]$ was thus obtained as previously described for real data. The critical value for the significance level of 0.01 was then determined.

To confirm gastrocnemius fatigue, the linear dependency between f_{median} and time was tested by the Pearson correlation coefficient (r) with null hypothesis of no-correlation, applying Student's t -test after Fisher transformation, since the inverse hyperbolic tangent of r has Gaussian distribution. A linear regression was also performed for calculating the f_{median} slope. As the time until fatigue failure was different among individuals, only the initial 90 s were considered to describe the f_{median} behavior.

Results

A decrease of the median frequency along the first 90 s of plantar flexion was observed in 17 subjects (Table 1). The myoelectric signals of three subjects were not used in this analysis due to the poor quality of signals during plantar flexion. The average f_{median} showed significant negative correlation with time ($r = -0.74$, $p < 10^{-5}$). Its slope was equal to -0.13 Hz/s, with a coefficient of determination of 0.54.

Table 1. Slope of the linear model of each subject's median frequencies during the first 90 s of induction of the fatigue

Subject	Slope (Hz/s)	Time Until Fatigue (min)
FA1	-0.23	1.65
FA2	-0.07	4.94
FA3	0.04	2.06
FA4	-0.24	6.14
FA5	-0.08	4.03
FA6	-0.28	2.11
FA7	-0.13	2.27
FA8	(*)	2.46
FA9	(*)	1.98
FA10	0.37	7.04
FA11	-0.15	4.42
FA12	-0.11	2.30
FA13	-0.15	3.51
FA14	-0.24	5.84
FA15	-0.06	6.64
FA16	-0.28	3.55
FA17	-0.07	1.77
FA18	-0.13	1.73
FA19	-0.43	3.23
FA20	-0.01	2.75
FA21	(*)	1.92
FA22	-0.25	7.62
Mean \pm standard deviation	-0.13 ± 0.17	3.63 ± 1.92

(*) EMG discharged due to poor signal to noise ratio during the induction of the fatigue.

Local muscle fatigue caused significant increases in the measured stabilometric variables *area*, *x-vel* and *y-vel* (Table 2).

Table 2. Stabilometric changes due the induction of muscle fatigue

Parameter	Before Fatigue	After Fatigue (*)
Area of COP displacement (mm ²)	157 ± 70	281 ± 133
Mean-velocity in y direction (mm/s)	6.1 ± 1.5	8.6 ± 2.6
Mean-velocity in x direction (mm/s)	7.2 ± 2.3	8.6 ± 2.5

(*) all values presented significant differences ($p < 0.05$)

The cross-correlation function employed for measuring latency between muscle activity and body sway showed a single significant ($p < 0.01$) correlation peak between RMS-EMG data $r[n]$ and mediolateral displacement $x[n]$ both before (0.20 ± 0.14) and after fatigue (0.16 ± 0.09) conditions (Table 3), as well as anterior-posterior displacement $y[n]$ of 0.23 ± 0.11 and 0.25 ± 0.10 before and after fatigue, respectively. The positive cross-correlation values correspond to increases of muscle activity related to movements of the COP forward or to the right, and decreases backward and to the left.

Table 3. Maximum value of cross-correlation between the RMS value of the EMG and anterior-posterior and mediolateral stabilograms, before and after fatigue

Subject	Anterior-Posterior Displacement				Mediolateral Displacement			
	Before Fatigue		After Fatigue		Before Fatigue		After Fatigue	
	$NCCF_{max}$	Latency (s)	$NCCF_{max}$	Latency (s)	$NCCF_{max}$	Latency (s)	$NCCF_{max}$	Latency (s)
FA1	0.45	0.90	0.48	1.32	0.48	0.88	0.20	1.14
FA2	0.13	0.90	0.10	1.36	(*)			
FA3	0.16	1.36	0.20	1.30	0.14	1.34	0.13	1.26
FA4	0.42	1.18	0.36	1.12	0.39	1.10	0.13	1.12
FA5	0.29	1.26	0.38	1.18	(*)			
FA6	0.05	0.94	0.12	0.94	0.12	0.94	0.11	0.94
FA7	0.21	1.04	0.29	1.02	0.05	1.18	0.16	0.95
FA8	0.34	1.14	0.23	1.10	0.32	1.18	0.11	1.02
FA9	0.15	1.20	0.21	1.38	(*)			
FA10	0.18	0.80	0.20	1.22	0.05	0.86	0.07	1.08
FA11	0.12	1.20	0.25	1.22	0.11	1.22	0.20	1.32
FA12	0.26	0.94	0.32	0.96	0.10	0.96	0.13	0.96
FA13	0.13	1.20	0.17	1.16	0.07	1.12	0.15	1.26
FA14	0.13	1.14	0.12	1.30	0.24	0.88	0.20	1.30
FA15	0.34	1.16	0.22	1.14	0.09	1.52	0.09	1.60
FA16	0.36	1.18	0.38	1.10	0.40	1.16	0.35	1.10
FA17	0.22	1.20	0.32	1.18	0.21	1.20	0.33	1.20
FA18	(*)				0.37	1.06	0.15	1.46
FA19	0.15	1.24	0.14	1.40	0.36	0.82	0.10	1.30
FA20	0.15	1.02	0.26	1.50	0.25	0.86	0.32	1.30
FA21	0.38	1.30	0.30	1.62	(*)			
FA22	0.18	0.92	0.18	1.38	0.11	0.94	0.07	1.32
Mean	0.23	1.11 [‡]	0.25	1.23 [‡]	0.20	1.01 [‡]	0.16	1.14 [‡]
SD	0.11	0.15	0.10	0.17	0.14	0.19	0.09	0.18

(*) No significant $NCCF$ peak observed.

‡ Values before and after fatigue significantly different ($p < 0.05$)

The COP signal is delayed in relation to EMG, which can be well observed with the $r[n]$ signal (Fig. 1 and 2), and confirmed by the cross-correlation function (Fig. 3). The average latency of the COP displacement for the studied subjects was significantly different from zero ($p < 0.05$), for both directions and both fatigue conditions (Table 3).

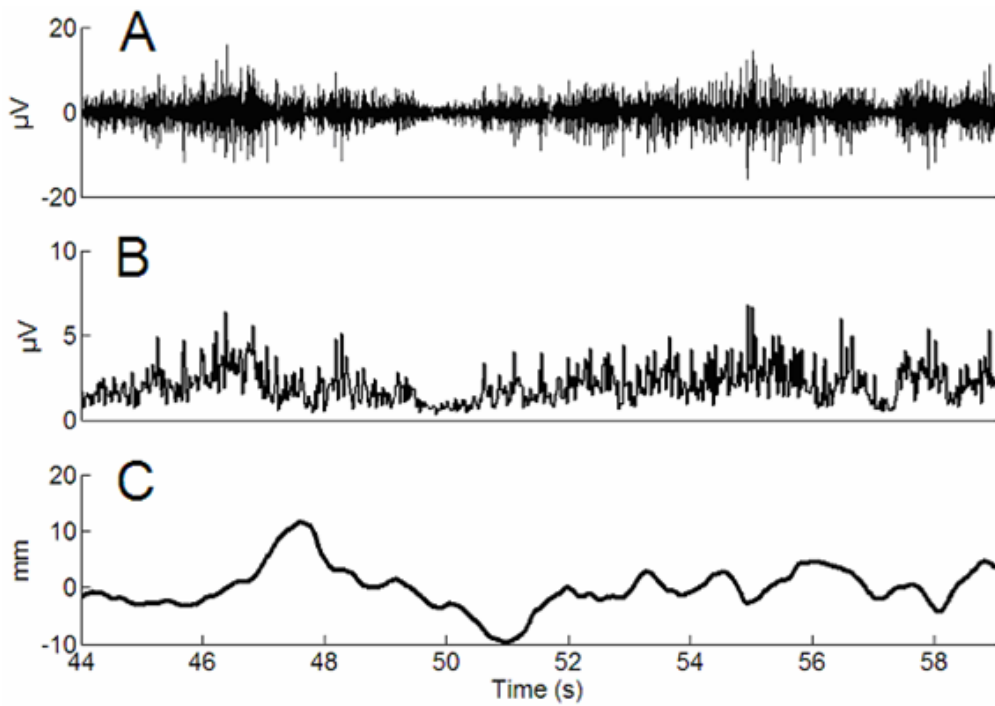


Fig. 1 Raw EMG signal from right gastrocnemius (A), the respective $r[n]$ data (B) and the synchronous anterior-posterior displacement of the center of pressure of the body (C). Data from subject FA1, before fatigue.

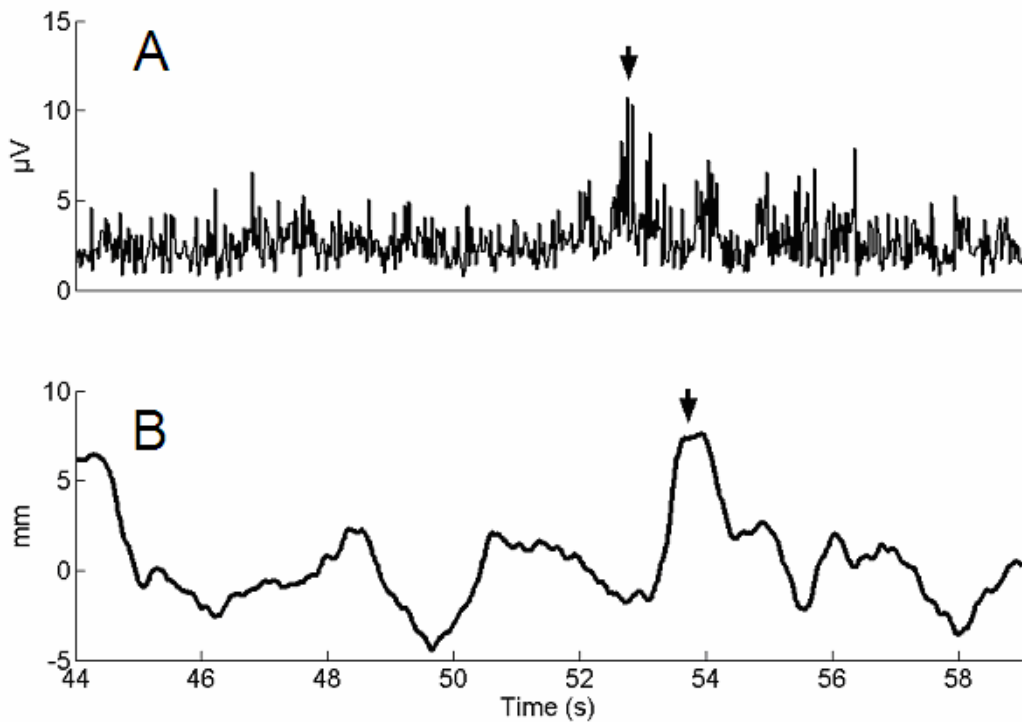


Fig. 2 $r[n]$ data (A) and the synchronous mediolateral displacement of the center of pressure of the body (B). The arrows indicate peaks in both signals and show that the stabilogram peak are delayed in relation to the $r[n]$ peak. Data from subject FA12, before fatigue.

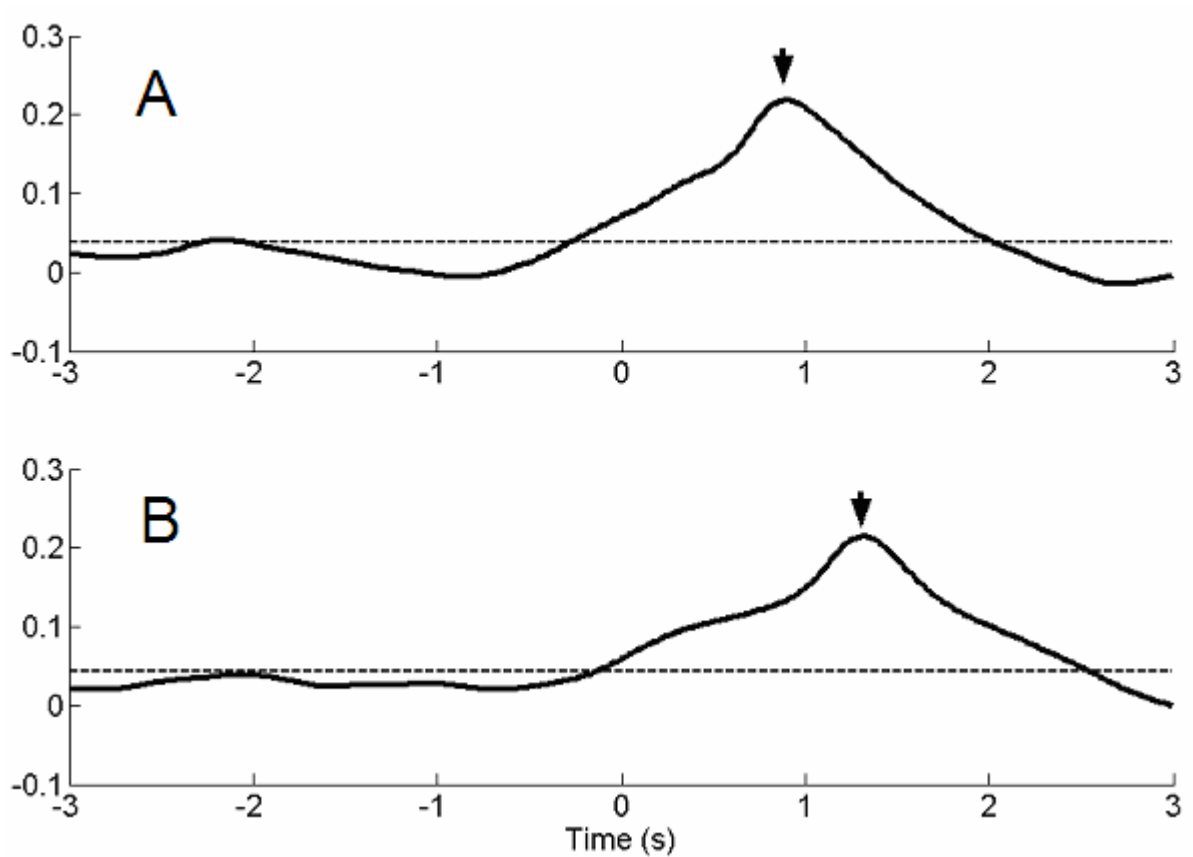


Fig. 3 Normalized cross-correlation function between gastrocnemius muscle $r[n]$ and the anterior-posterior displacement of the center of pressure of the body, before (A) and after (B) fatigue induction. The arrows indicate the maximum values and the dashed lines indicate the critical value ($\alpha = 0.01$) obtained with Monte Carlo simulation. Data from subject FA1.

The fatigue caused a significant increase ($p < 0.05$) in the ensemble average latency between $r[n]$ and both anterior-posterior and mediolateral stabilograms.

Discussion

In this work, two questions were addressed concerning anticipatory activity in the control of body sway: 1) Does it play a relevant and coherent role in postural control, and 2) if so, what is the effect of the muscle fatigue on this process?

Evidence and Role of Anticipation

The time delays obtained in the present study clearly cannot be explained solely by the electromechanical delay (EMD), since COP movements are delayed at least 800 ms in relation to the $r[n]$. Depending on the motor unit characteristics, its muscle fibers reach maximal force after 20 to 150 ms (EMD) from the onset of the depolarization, with the force decreasing gradually after that [14].

The present results support the hypothesis that there is a predictive strategy of control, previously pointed out by Gatev et al. in a similar study [13]. The anticipation between soleus activity and stabilograms was observed by Fitzpatrick et al. [12], with a low magnitude random perturbation applied at the pelvis. These authors observed a phase advance of the EMG related to the stabilogram, corresponding to 100 and 300 ms of anticipation. The anticipatory mechanism was also observed when a sinusoidal translation was applied to the platform [9]. Dietz et al. [9] suggested that there was no significant contribution of the spinal-vestibular reflex for the compensatory actions of the gastrocnemius and anterior tibialis muscles during this translation, as the integrated EMG signal presented a very small delay in relation to the head movement.

Using a realistic simulation of the inverted pendulum, Loram et al. [18] observed that significant changes in the pendulum sway size were obtained only by modification in the CNS control and not in the stiffness, suggesting the actuation of an anticipatory control mechanism. Soon after, Loram and Lakie [19], using a mathematical model, proposed that quiet standing control presents a “throw and catch” pattern, which it is assumed that the resting equilibrium position of the human inverted pendulum is unstable and temporary. These authors suggest that movements from a different resting equilibrium position can only be accomplished by a ballistic-like “throw and catch” pattern of torque, and again the anticipatory mechanism would be responsible for sway size reduction.

Moreover, Loram and Lakie [20] showed that ankle stiffness is determined by various elastic and more compliant components than muscle (foot, aponeuroses and Aquilles' tendon) in series, and noted that the CNS cannot control the ankle stiffness, which is a biomechanical constant. Therefore, the authors proposed that triceps surae maintains balance by predictively controlling the proximal offset (myotendinous junction) of the spring-like element (tendon) in a ballistic-like pattern. To elucidate the active control of human standing, this hypothesis was supported by showing how a large real inverted pendulum coupled to a limb by a soft spring was manually balanced [16]. This setup with low intrinsic stiffness revealed that satisfactory stability is obtained with movements in opposition to the pendulum load. Therefore a hypothesis emerged that during standing position the triceps surae muscle shortens in forward sway and lengthens in backward sway. The paradoxical muscle movement hypothesis was confirmed for soleus and gastrocnemius muscles by using dynamic ultrasound imaging *in vivo*, with subjects making voluntary sway [21] or in quiet standing [22]. Thus, the studies presented by Loram's group are in agreement with the present results and would predict that gastrocnemius muscle would be activated in advance of maximum COP displacement.

The question that arises from the present study is “why the measured delay between muscle activity and COP displacements are higher than other values from literature?” In the case of experiments involving sudden and unexpected perturbations, smaller anticipatory control may reflect the fact that brain is not as trained for sudden perturbations as it is for natural body oscillations [36].

The measured delay around 1 s is consistent with the median period of oscillation of the body described by Lakie et al. [16], who pointed out that the unidirectional movement of the body is always around 1 s. Thus, the present results suggest that the gastrocnemius muscle is electrically activated immediately after the beginning of a COP movement in the forward or right direction, acting more to prevent than to compensate a larger displacement. This reasoning is also consistent with the studies of Collins and De Luca [6], who observed that the autocorrelation of the COP signals are positive and decrease for lags from zero to 1 s, approximately, becoming negative for higher lags.

On the Use of Cross-Correlation to Measure Delays

The cross-correlation values obtained are low, although significant, suggesting that the myoelectric activity of gastrocnemius is not the only variable that explains postural control, mainly in the mediolateral direction, thus explaining the absence of significant peaks in some cases. The reduced *NCCF* values are partially explained by the reduced time window (20 ms) employed for calculating RMS-EMG data. With the chosen value, the resulting signal remains with high frequency oscillations, which are not expected to have response in the COP displacements. However, the option for a more smoothed RMS-EMG data would imply in decimating both $r[n]$ and stabilometric signals. Consequently, the reduced *NCCF* time-resolution would affect the measurement of changes in the peak delay due to fatigue. To assure that *NCCF* peaks are significant, the Monte Carlo simulation was adopted [23], with a significance level of 0.01. This procedure guarantees that significant values represent a linear dependence between both EMG activity and COP displacements.

The use of *NCCF* for measuring latencies between correlated signals is a classical procedure in digital signal processing [33], although its results should be carefully analyzed in the context of control systems with feedback and feedforward control mechanisms [15]. This becomes an open field for further investigation.

Effect of Fatigue

The changes observed in the power spectrum during sustained isometric contraction reflect the non-stationarity of the EMG, as pointed out by Merletti and Lo Conte [25]. The decrease of the f_{median} during the isometric contraction of the lateral gastrocnemius shows the development of the fatigue process, and the fall rate can be related to the rate of the muscular decreasing force [7,10,17,25]. Therefore, the protocol of isometric plantar flexion was sufficient to induce localized muscle fatigue in most of the subjects.

The local muscle fatigue caused a significant increase in the delay of COP displacement in relation to the muscular activity. This result agrees with other recent studies, including the pilot study of Allison and Henry [1]. These authors observed a significant increase after fatigue of the latency for the trunk muscle activation anticipatory to shoulder flexion movement, although it was not observed in every trial. Vuillerme et al. [40] also observed increased latency between EMG of semitendinosus muscle and the onset of arm raising movement, suggesting a functional adaptation. In this sense, as a localized fatigue may be due to lactic acid accumulation and a consequent pH reduction [24], a possible cause for the increased delay is the need of a longer and more intense neural stimulation after fatigue to provide the effective muscular contraction [24]. Thus, the effects of muscle action over COP displacement are delayed, and the body sway tends to be increased with fatigue as confirmed by the increased area and velocity of COP displacement.

Conclusion

The present study indicates that anticipation mechanism plays a relevant role in the body sway control with an average latency around 1 s between myoelectric activity of gastrocnemius muscle and movements of COP, which is greater than that observed for other controlled movements of the body. Fatigue causes a secondary effect which increases this latency.

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