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Journal of Electromyography and Kinesiology 17 (2007) 739–746

Journal of Electromyography and Kinesiology

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Anticipation mechanism in body sway control and effect of muscle fatigue

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Received 30 March 2006; received in revised form 14 August 2006; accepted 25 August 2006

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 ($p < 0.05$).
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Keywords: Anticipation; Body sway; Cross-correlation function; Fatigue; Electromyography

1. 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 (Winter et al., 1998, 2001) and superior levels of the central nervous system (CNS) (Lakie et al., 2003; Loram and Lakie, 2002a; Loram et al., 2005; Morasso et al., 1999; Morasso and Sanguineti, 2002; Morasso and Schieppati, 1999). While standing, the body sways in all directions with variable muscular activity regulated by discrete neural stimuli (Lakie et al., 2003) to maintain the projection of the center of mass (COM) within the limits of the support base. According to the stiffness theory (Winter et al., 1998, 2001, 2003), 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 (1999) 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. (1996) 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

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doi:10.1016/j.jelekin.2006.08.011

dysfunction generally show postural balance difficulties, these authors suggested that sensory information participate in the postural control in addition to the feedback. Other theoretical (Morasso and Sanguineti, 2002) and experimental (Loram and Lakie, 2002b) 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 (Loram et al., 2005; Morasso et al., 1999; Morasso and Sanguineti, 2002; Morasso and Schieppati, 1999; Shumway-Cook and Woollacott, 1995). 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 (Allison and Henry, 2002; Aruin and Latash, 1995; Brown and Frank, 1987; Dietz et al., 2000). The anticipatory control of postural adjustments also have been studied with perturbations applied directly to the subject or to it base of support (Allison and Henry, 2002; Brown and Frank, 1987), 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 of 90 ms (Brown and Frank, 1987), 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. (1999) 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. (2005) 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 (Vuillerme et al., 2001, 2002a,b). Nardone et al. (1997, 1998) observed increased area of the COP and length of the sway path after fatigue. Additionally, Allison and Henry (2002) 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.

2. Materials and methods

2.1. 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.

2.2. Stabilometric data recording

A three-point vertical force platform, developed by the authors (Oliveira, 1996) according to the specifications of the Association Française de Posturologie (Bizzo et al., 1985), 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 TΩ) with a second order anti-aliasing Butterworth 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.

2.3. 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 TΩ 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.

2.4. Software

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

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