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The Effects of an Ankle Destabilization Device on Muscular Activity while Walking

Abstract

Chronic Ankle Instability sprain causes are unclear and many factors or mechanisms may contribute to recurrence of this injury. The aim of the study was to investigate how an ankle destabilization device affects the EMG patterns of the ankle muscles during ankle stabilization against inversion. The left foot was equipped with a mechanical device mounted under the heel of the shoe. This mechanical device induces subtalar joint destabilization necessitating the control of ankle muscles. Surface electrodes were placed over the tibialis anterior, the peroneus longus, the peroneus brevis, the gastrocnemius lateral, and the gastrocnemius medial. Nine healthy subjects (mean age 37 ± 12 yr; mean mass 68 ± 17 kg; mean height 1.73 ± 0.7 m) were instructed to walk normally along a tape fixed on the floor. The ankle destabilization device altered the walking pattern of all subjects. More specifically, the walking pattern is disturbed resulting in higher amplitude of the EMG activity of the peroneal muscles and the Tibialis Anterior and anticipatory reactions in the peroneal muscles. The results suggest that the ankle destabilization device could be beneficial for rehabilitation programs especially during the training of walking. Using this material may help to a specific reinforcement of muscles involved in anti-inversion ankle movement.

Key words
Electromyography · orthosis

Introduction

Lateral ankle sprain (LAS) account for fifteen to forty-five percent of injuries encountered in athletes especially when sports activities required jumps, jump reception, or abrupt change in direction like soccer [16], volleyball [22], basketball, or handball. The recurrence rate of such injury is estimated at eighty per cent [24]. Moreover, ten to twenty percent of persons with LAS may later develop chronic lateral ankle instability [10]. Two main hypotheses explain the mechanisms which may contribute to Chronic Ankle Instability (CAI).

Firstly, CAI may be related to neuromuscular causes herein called neuromuscular instability. The main cause of CAI would be a reduced ankle proprioception [6]. Proprioception arises from various specialized receptors in muscles, tendons, skin, and articular structures [17]. The lateral ligament and the joint capsule of the talocrural and subtalar joints but also muscles and tendons acting at the ankle, are strongly innervated by sensory receptors. When tension in lateral ligaments excessively increases, mechanoreceptors inform the Central Nervous System (CNS) and a motor response is generated to slow down or reverse the direction of the movement. When the ankle is twisted, some of muscular

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and joint receptors are damaged and proprioceptive signals may be altered. Several studies have addressed this issue with various experimental designs such as single limb balance test [24], reaction to sudden ankle inversion [15], angle replication [9], or movement detection [20,21]. Although, alteration of the proprioceptive message could be compensated by various facilitation mechanisms occurring at the spinal or higher centers level [7] Konradsen (2002) found a measurable deficit in ankle proprioception for subjects with chronic ankle instability.

Secondly, CAI may be related to mechanical causes herein called mechanical instability. CAI could yield changes in mechanical strength of the damaged lateral ankle ligaments and/or ankle evertor muscles [25]. Certainly past research in the area of ankle stability suggests the importance of evertor muscles strength [1]. Also, Konradsen et al. and Wilkerson et al. have demonstrated [13,26] that muscular weakness of the muscles which evert the ankle could contribute to the recurrence of the injury following lateral ankle sprain. Moreover, in a recent study, Yildiz et al. [28] investigated eccentric evertor/concentric invertor strength ratio. They conclude that rehabilitation should include eccentric muscle strengthening particulary for the evertor muscles. Consequently, it could be advantageous to specifically train the muscles acting in the stabilization of the ankle against inversion during rehabilitation and prevention programs.

Most of the rehabilitation programs include proprioceptive exercises (see [18] for a review), like (i) single limb stance training, (ii) wooble-board training allowing uniplanar movement at the ankle, and (iii) ankle disk training allowing multiplanar movement at the ankle. It is known that these exercises induced significant improvement in joint position sense, postural sway, and muscle reaction time [5]. However, few of them use muscular activity characteristics around the ankle to assess the improvement of their training program. Peroneal muscles are the first muscles to contract to a sudden ankle inversion. As underlined by Hertel [10], increased magnitude of peroneal muscles activity in response to inversion stress may be a good indicator of the restored functional stability. Some authors [10] argued that rehabilitation programs should address the restoration of the control of volitional contractions of the muscle acting at the ankle. In consequence, it should be significant to improve neuromuscular control by using a device that induces a normal pattern of movements and a specific pattern of muscular activity related to dynamic condition (such as walking, for example).

The purpose of this study was to evaluate the effect of an ankle destabilization device on the ankle muscular activity observed while walking. It was expected that this device would 1) lead to a specific activation pattern improving functional stability at the ankle and 2) increase evertor muscle strength.

Methods

Subjects characteristics

Nine healthy (mean age 37 ± 12 yr; mean mass 68 ± 17 kg; mean height 1.73 ± 0.7 m) volunteers without any history of ankle sprain participated in the study. Subjects were excluded if they reported any musculoskeletal systemic disease. Prior to the experiment they were informed about the goal of the study and the general procedure. All subjects provided written informed consent according to University protocol.

Apparatus

The electromyographic activity (EMG) was recorded from the left leg with a portable ME3000 P8 device (Mega® Electronics, Finland). To minimize skin impedance, the skin was shaved and cleaned with an ether-alcohol-acetone solution and then rubbed with sandpaper. Surface electrodes (Skinact® Ag-AgCl electrode type F55) were placed 3 cm apart and oriented longitudinally over the Tibialis Anterior (TA), the Peroneus Longus (PL), the Peroneus Brevis (PB), the Gastrocnemius Lateral (GL), and the Gastrocnemius Medial (GM). The positions of the electrodes were defined according to the recommendations of Cram et al. [4]. The measured EMG signal was low-pass filtering (8 – 500 Hz), amplified 375 times at the source, sampled at 1000 Hz (12 bits A/D conversion), and stored on a memory card (type Flash memory, PCMCIA standard 32 MB).

Ankle destabilization mechanism

For all subjects, only the left foot was equipped with the ankle destabilization device. As shown in Fig. **1**, the device used in the present experiment consisted of an articular device (Fig. **1 a**) fixated under the heel of a shoe (Fig. **1 b**) inducing specifically subtalar joint destabilization during touchdown. Destabilization axis has a mean inclination of 42° in the saggital plane and deviates by 23° toward the medial side. Ankle movements' amplitudes are controlled by two laterals screws. For this study, inversion and eversion amplitudes were set to 5 and 20 deg, respectively. With this orthosis, subjects are forced to walk with an extra ankle inversion and plantar flexion. A switch fixed on the articular device allows us to record precisely heel-strike occurrence and to synchronize mechanical events with EMG signal.

Procedure

Muscle activity

Before testing, the maximal muscle activity of each muscle was recorded during a maximal isometric voluntary contraction (MVC) test. Measurements were done during resistive ankle plantarflexion, dorsiflexion, and pronation movements with the subjects in a sitting position. After a muscular warm-up, all subjects performed 3 MVC trials. The highest EMG value (in µV) attained was kept as the MVC reference value. The mean muscular activity was expressed as the percentage of the MVC reference value. Subjects had a practice period to familiarize themselves with the destabilization device while walking. Then, subjects were instructed to walk normally along a tape fixed on the floor. The walking distance was fixed at 6 meters. The subjects were asked to perform 5 strides (stance and swing phases). This task was executed both normally (NC) and with the ankle destabilization mechanism (ADM) applied to the left leg. Subjects performed 10 trials per condition and the order of presentation of the condition was randomized.

Parameters

For each stride, all EMG signals were smoothed with a low-pass filter of 20 ms, integrated and finally normalized as a percentage

Fig. **1 a** and **b** **a** Lateral view of the device used in the study, the dotted line represents the destabilization axis. **b** Illustration of the subtalar joint destabilization induced by the device. When the subject steps (left), the shoe rotates around destabilization axis, eliciting an inversion and plantarflexion movement of the ankle (right).

of the IEMG obtained for the MVC. These values were defined as the Muscle Percentage Activation (MPA). Temporal parameters were calculated only for anti-inversion muscles, that is for PL and PB. EMG onsets were determined automatically for each step. Muscular activation starts when IEMG value exceeds 3% of the basal level during 5 ms at least. Muscle Onset Time (MOT) is defined as the time between the moment of the touchdown of the foot wearing the ankle device and the onset of the EMG response. MPA parameter was submitted to a 2 conditions (NC vs. ADM) × 5 muscles (TA vs. PL vs. PB vs. GL vs. GM) analysis of variance (ANOVA) with repeated measures on both factors. MOT parameter was submitted to a 2 conditions (NC vs. ADM) × 2 muscles (PL vs. PB) ANOVA. Post-hoc analyses (orthogonal planned comparison) were performed whenever necessary.

Results

A representative example of individual data is shown in Fig. **2**.

Effects of the ankle destabilization mechanism on muscular activation

For the MPA parameter (Fig. **3**), the ANOVA showed a main effect of condition ($F[1.8] = 11.42$; $p < 0.01$). Muscular activation was higher when subjects walked with the ankle destabilization device (11.7 vs. 8.4% for the normal and destabilization conditions, respectively). For all variables, the power of the main effect of orthosis was 0.96, using what is suggested as a "large effect". Moreover comparison of means showed a significant interaction of condition × muscle ($F[4.32] = 17.73$, $p < 0.001$). When subjects were wearing the ankle destabilization device, muscular activation was higher for TA (16.1 vs. 8.3%), PB (16.7 vs. 14.6%), and PL (16.6 vs. 10.6%), and lower for GM (5.4 vs. 8%) and GL (5.6 vs. 7.5%).

Effects of the ankle destabilization mechanism on muscle onset time

For the MOT parameter, the ANOVA showed a main effect of Condition ($F[1.8] = 52.73$, $p < 0.001$) but no effect of Muscle nor any interaction Condition × Muscle ($p > 0.05$). On average, MOT occurred 144 ms after the heel-strike while subjects walked normally and 77 ms before heel-strike while subjects walked with the ankle destabilization device. Hence, as illustrated in Fig. **4**, ankle destabilization device induced anticipatory muscular events compared to normal condition.

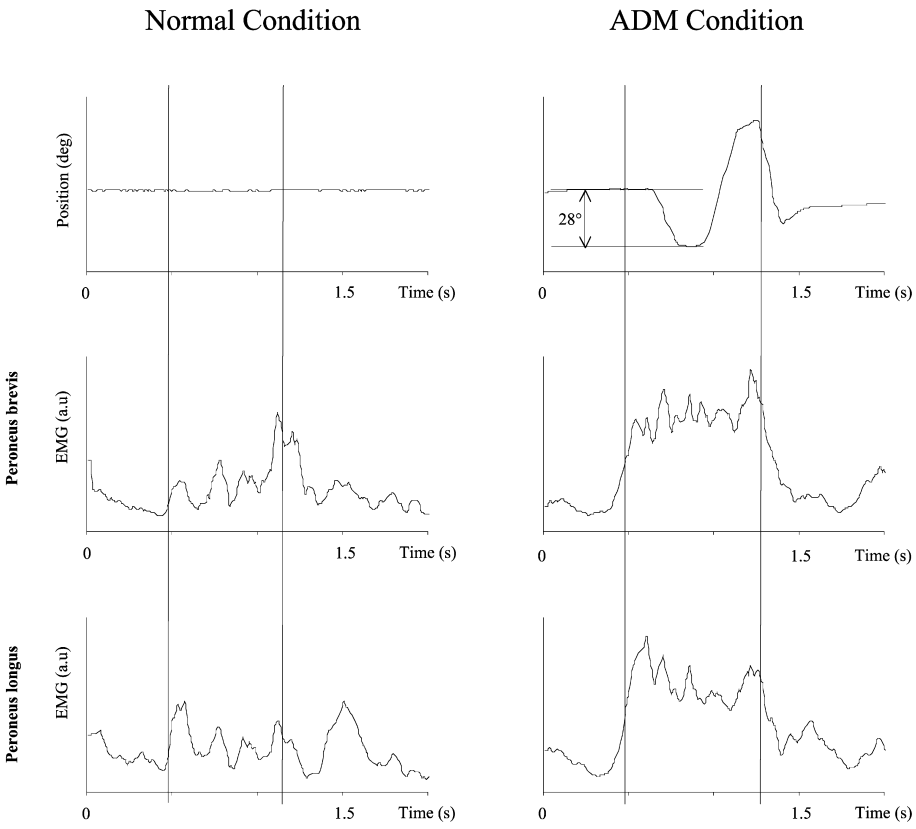

Fig. **2** Mean biomechanical influence of the device on the ankle joint and peroneal muscles. Note that in ADM condition, when touchdown (first vertical line), the device induced an extra inversion of 28° associated with a larger muscular activity of the peroneals.

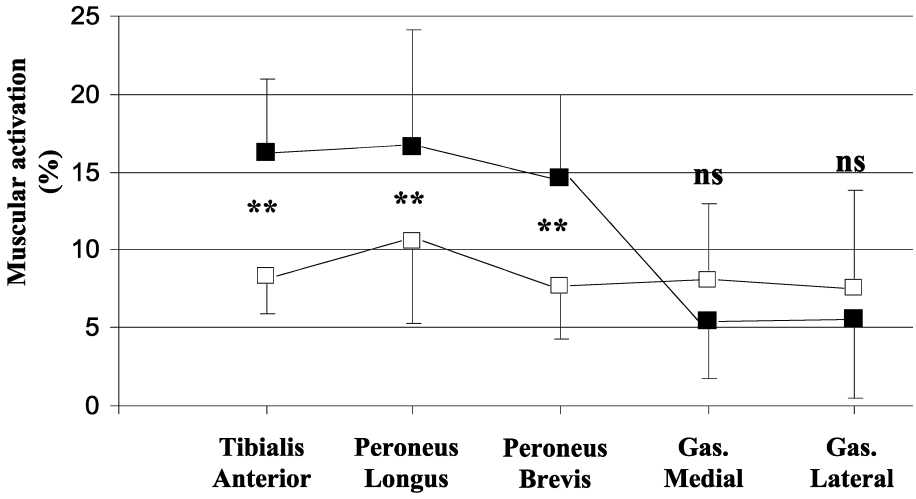

Fig. **3** Mean muscular activation and standard deviation for the five muscles and the two conditions (□ normal condition vs. ■ orthose). Note that with orthose muscular activation increased especially for anti-invertor muscles (** $p < 0.01$).

Discussion

Lateral or inversion ankle sprain has been identified as common musculoskeletal injuries. The incidence of re-injuries remains relatively high, between 10–30%, despite the initial treatment taken by the injured subject [19]. The aim of the study was to increase muscular activity while walking with a new ankle destabilization device. It is believed that increased muscular activity could improve ankle stabilization against inversion. One main result showed that the ankle destabilization device increased activation of the tibialis anterior and peroneal muscles. One functionality of these muscles is to stabilize the ankle and protect the ankle against inversion movement. This illustrates the muscular specificity of such a device. These particular points were illustrated in Fig. **5 a**.

The low muscular activity, expressed by the MAP parameter, observed for the medial and lateral gastrocnemius can also be explained by the ankle destabilization device. Indeed, the ankle destabilization device does not give sufficient support to the foot during the midstance and the propulsive phases of the walking cycle. Moreover, with the ankle destabilization device, subjects are forced to walk with an extra plantar flexion.

The second main result concerns temporal modifications of the muscular activation when subjects wore the ankle destabilization device. Since peroneal muscles are directly involved in the

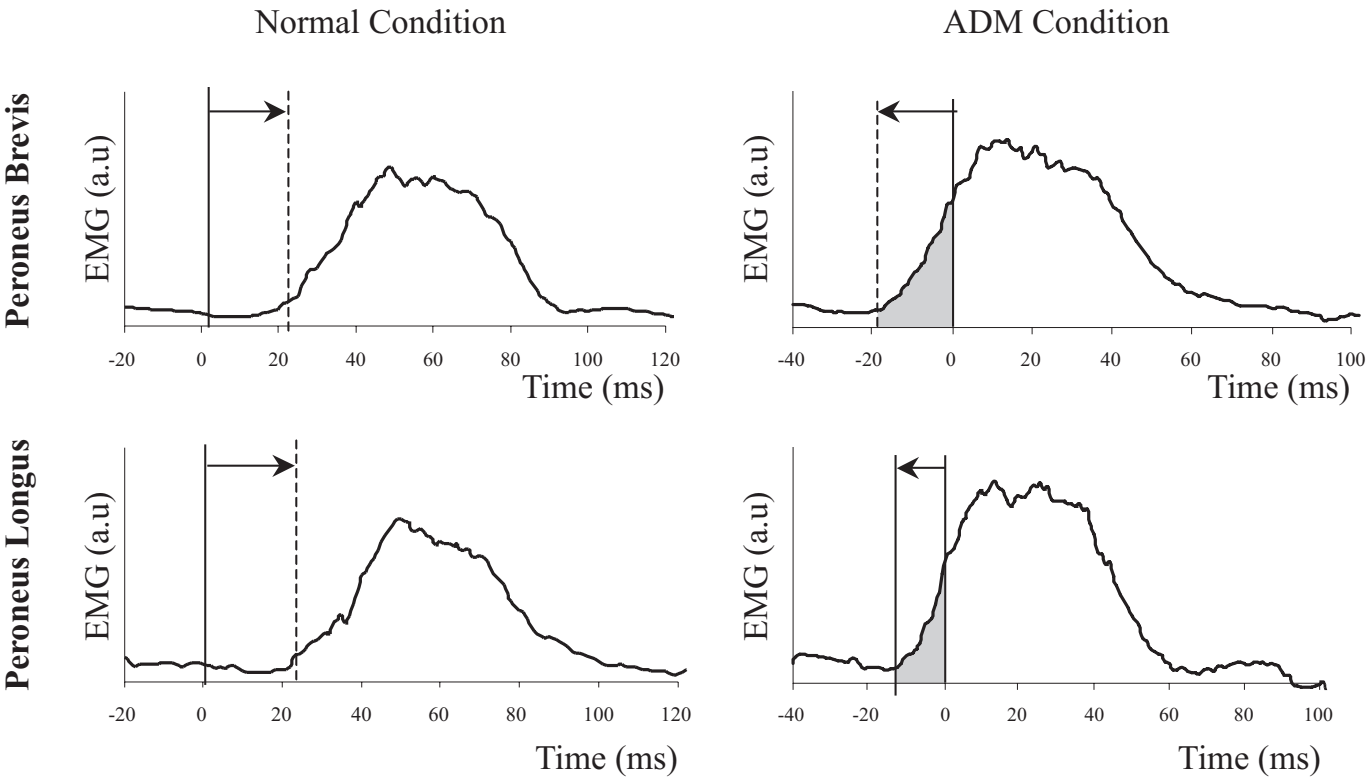

Fig. **4** Illustration of two typical peroneal EMG profils for one subjects (S2). Vertical solid lines represent foot contact while vertical dashed line represent EMG onset. With destabilization device, EMG onset occurred before heel-strike.

inversion movement, the measurement of Peroneal Reaction Time (PRT) is generally used for the assessment of proprioceptive capabilities. PRT was considered as a reflex reaction to sudden inversion initiated by the inversion movement and followed by a reaction pattern mediated by spinal or cortical motor centers [14]. Benesch et al. [2] reported a PRT equal to 64 ms for peroneal muscles and concluded that PRT may be used as a clinical test parameter to differentiate the diagnosis of chronic ankle instability. During walking the peroneal EMG response to sudden inversion was formed by an early medium latency signal with a connected small eversion torque output and a long latency EMG signal resulting large eversion torque response [12]. In order to counteract the inversion rotation the speed and the magnitude of the recruitment of the long latency eversion torque response could be optimized. One possibility is that the spindle receptors within the peroneal muscles were recruited early as the muscles came under tension. As a result, a more powerful response achieved in a shorter time could be produced by the peroneal muscles to help protect the twisting ankle. However, in a recent study, Grüneberg et al. [8] investigated the reflex responses of the lower leg muscle when landing on a surface that would not induce ankle inversion. The authors clearly showed that the response latencies (estimated at 44 and 90 ms for the short and long latency response, respectively) are too late to counteract the inversion movement itself (estimated at 42 ms) and that anticipation is more efficient. Moreover, our results showed that with the device both peroneal muscles were activated before heel strike. In other words, the present results may not depend on reflexes being more optimal but on muscular anticipation being more efficient.

This pro-active muscular defense reaction to ankle inversion was similar to the dynamic defense mechanism mentioned by Konradsen [12,14]. These anticipatory mechanisms were certainly caused by the large instability induced by the device. Muscular contraction of peroneal muscles may add a substantial torque output allowing the stabilization of the rearfoot during midstance phase of the walking cycle. This anticipatory activity may also help to stabilize the center of mass and absorb the impact forces and moments of force created when the left foot touched the ground.

In conclusion, the results showed potential benefits of the ankle destabilization device for rehabilitation programs under more natural condition. Using this device led to a specific muscle strength training of the peroneal and calf muscles. Moreover, a pro-active muscular response was established prior to the heel-down when subjects wore the device. A limitation of the present study is that muscular activity while walking with or without the ankle destabilization device was only assessed in healthy young subjects. It may be interesting to assess the effects of such a device with a population suffering from CAI. It could be hypothesized that using this device could lead to a decrease in the frequency of recurrent ankle sprains. Future study will attempt to assess injury incidence with and without orthosis and will further examine muscles responses for shoe-integrated orthoses during more dynamic functional condition such as jumping and running.

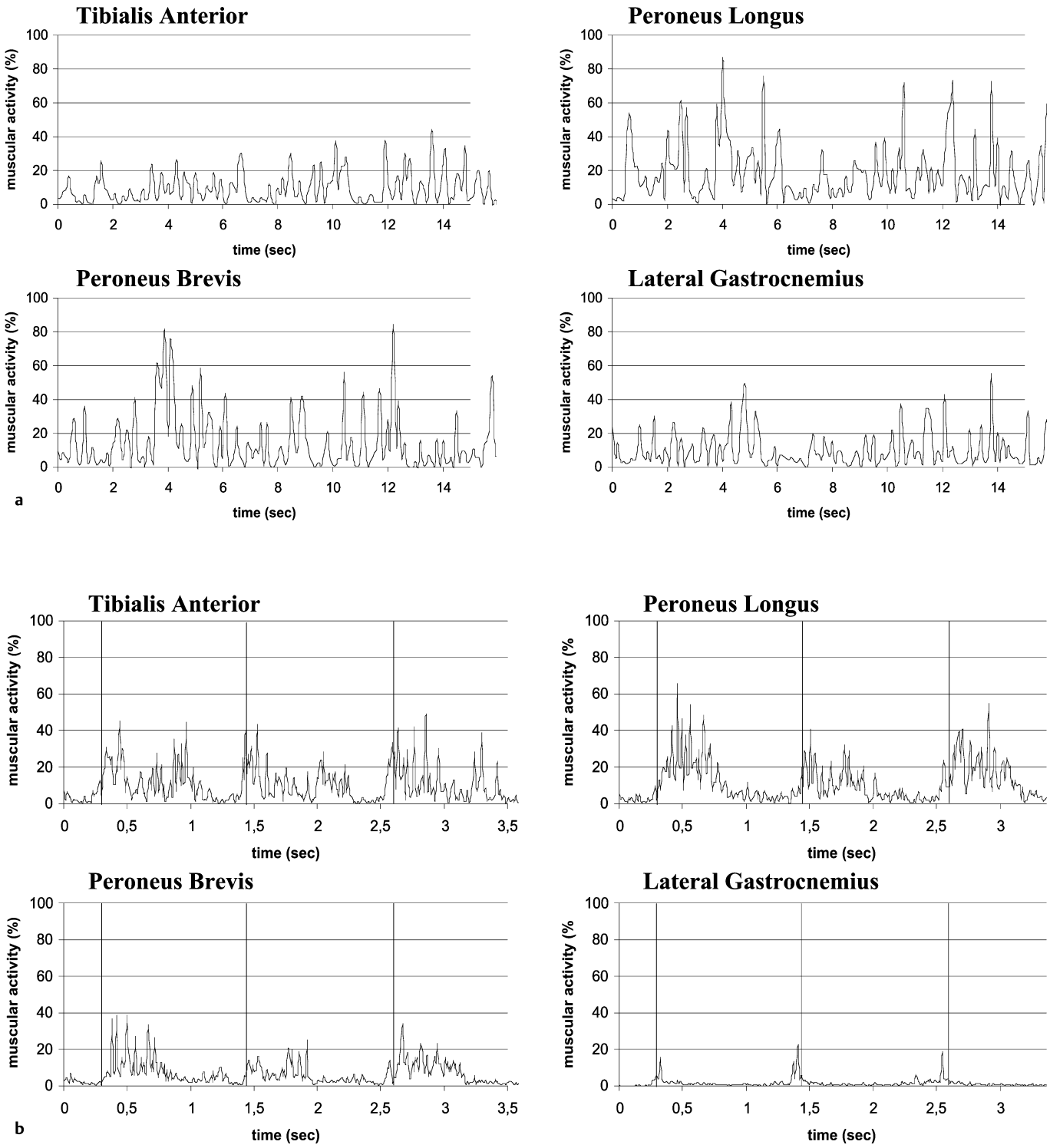

Fig. **5 a** and **b** Rectified EMGs recording for S6 during **a** 15 seconds of balance board exercise and **b** walking with destabilizing device. Vertical solid lines represent foot contact. Note the specificity of the muscular activation induced by the device.

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