Electromyographic Changes of Agonist and Antagonist Calf Muscles During Maximum Isometric Induced Fatigue

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Bibliography

Abstract
The purpose of this study was to examine electromyographic changes of the agonist and antagonist muscles during fatigue. Nine healthy, untrained subjects exerted a maximum voluntary heel lifting contraction with their dominant limb. The EMG activity over the soleus and the tibialis anterior muscles was recorded during the contraction. The results showed that the torque output during heel lifting and the soleus EMG activity decreased, whereas the tibialis anterior EMG revealed a small but non-significant decrease. However, the ratio of the tibialis anterior to the soleus EMG increased significantly at the end of the fatigue protocol, a fact that reveals that the decrease rate of the antagonist’s activity was significantly lower than the decrease rate of the agonist activity. It is concluded that during a maximal fatigue protocol, both the agonist and antagonist muscle activity may decline, however, the slower rate of antagonist’s activity decrease relative to the agonist’s activity is a finding that requires further investigation. This finding may reflect a higher level of agonist and antagonist muscle co-activation and probably a relatively higher opposing torque from the antagonist muscles at the end of the fatigue session.

Key words
Isometric contraction · electromyography · co-activation · soleus · tibialis anterior

Electromyography (EMG) has been widely used in the past to detect the responses of the central nervous system during fatigue and non-fatigue activities. Concerning fatigue, it has been confirmed that the alteration of the EMG amplitude of the agonist muscle is dependent on the contraction intensity. Previous studies have shown that during submaximal fatigue the EMG amplitude increases though the torque output remains constant [7,26]. In contrast, during maximal fatigue the torque output and the EMG amplitude decreases [41,42] and this decrease can be either attributed to an inhibition of some motor units (probably the fast ones) [17] and/or a decrease in the motor unit firing rate [29]. Furthermore, it has been observed that this regulation is achieved by increasing the activity of some spinal and supraspinal mechanisms [12,25], which cause a facilitation of the inhibitory reflex pathways [45]. However, the torque output during a sustained maximum contraction is the net result of the torque applied around a joint, both from agonist and antagonist muscles. Studies in the past have shown that the level of co-activation - defined as simultaneous recruitment of two or more muscles [20] - of the agonist and antagonist muscles, is dependent on many factors, such as the type and the velocity of the contraction, the muscle length, the training level or former joint injuries (for review see [23]). Hence, it seems that the central nervous system regulates the level of co-activation of the agonist and antagonist muscles de-
EMG was calculated by summing the rectified EMG activity. The data of the above mentioned studies were concerned with the knee extensor muscles and cannot be generalized to other muscle groups. Currently, there is no available information concerning the level of co-activation of the tibialis anterior (acting as antagonist muscle) during fatigue induced by heel lifting, although this muscle group is potentially chosen for studies involving repeated stretch shortening cycles (e.g. [14, 19]) or for studies which use the method of H-reflex as a tool for estimating the function of some spinal mechanisms (e.g. [31, 32]). Thus, the purpose of this study was to examine the effect of maximal sustained isometric heel lifting on the antagonist activity.

**Materials and Methods**

Seven male and 2 female volunteers (mean age ± SEM: 28.9 ± 1.8 years, body mass: 79.5 ± 4.2 kg, height: 176.0 ± 3.2 cm) participated with informed consent. All subjects were non-athletes with moderate levels of physical activity and did not suffer from any neuromuscular diseases.

Each subject laid comfortably on a medical chair with his/her dominant foot fixed firmly on the foot platform of a CYBEX II isokinetic dynamometer. Non-elastic Velcro straps were applied around the foot in order to fix it on the force platform. The ankle flexion axis was aligned with the dynamometer axis of rotation. The knee was flexed (110°), in order to reduce the contribution of the gastrocnemius muscle to the torque production [11, 18, 36, 38]. Before the fatigue protocol, 3 maximal voluntary contractions (MVC) were performed (4–5 min interval) for the ankle extensors muscle group. The MVC test was used to maximize the performance for the following fatigue protocol, since it is very common that the subjects can exceed their putative MVC if they try again to reach a higher torque value [1]. After the MVC testing, fatigue was induced by a maximal isometric heel lifting. The procedure was terminated when the torque output decreased to 50% of maximum voluntary torque [8, 35]. All subjects were verbally motivated to the development of their maximal voluntary torque during the whole fatigue protocol. Visual feedback of the torque output was provided throughout the experimental session.

The EMG activity was differentially recorded with bipolar surface disk electrodes (Ag/AgCl) of 1 cm diameter. The center-to-center interelectrode distance was set at 2 cm. The surface of the electrodes was covered with electrode gel and fixed with adhesive tape. Before the electrode placement the skin was dry-shaved, rubbed with sand paper and cleaned with alcohol. The EMG electrodes were placed above the soleus and tibialis anterior muscles according to Basmajian and Blumenstein [2]. More specifically, for the soleus muscle the electrodes were applied over the midline of the calf and 2–3 cm lower from the distal margin of the gastrocnemius heads and the Achilles tendon. For the tibialis anterior muscle, the electrodes were centered one or two fingers laterally to the tuberosity of the tibia, were the muscle belly can be observed during dorsal lifting of the foot. The ground electrode was placed on the ankle of the contra-lateral foot. The cross-talk between agonist and antagonist muscles was minimized by validating each electrode placement after the performance of specific manual resistance tests [44], which are consisted of isolated movements specific to each muscle.

The EMG signal was preamplified (×1000) and filtered (bandpass 10 Hz to 3 kHz) with an EMG mainframe (Columbia 9010, input impedance: 200 MΩ, CMRR: >94 dB). The signal was further digitalized using an A/D card (Data Translation DT2801a, 12 bit) with a sample rate of 1 kHz.

The data acquisition for the torque and EMG measurements was repeated every 10 seconds during the fatigue protocol and each acquisition lasted for 500 msec. The EMG signal was fully rectified and the mean EMG value (EMG) for each acquisition was calculated. The EMG was calculated by summing the rectified EMG recordings over the time interval of 500 ms and then dividing the summation by the number of recordings. All torque, EMG values and ratios were normalized relatively to the first measurement during the fatigue protocol.

Results are presented as mean values and standard error of mean (SEM). The analyzed dependent variables were the soleus and tibialis anterior EMG, the ratio of the tibialis anterior to the soleus EMG and the ratios of the soleus and tibialis anterior EMG to the produced torque. One-way analysis of variance (ANOVA) for repeated measurements was applied for each dependent variable to calculate the mean differences when the maximal torque output decreased to 90, 80, 70, 60 and 50% of the initial MVC. The Scheffé post-hoc test for multiple comparisons was performed when the ANOVA indicated an overall p value less than 0.05.

**Results**

All subjects showed a 50% decrease in the torque output within 1.6 to 3 min. During the fatigue protocol the soleus EMG activity declined gradually reaching at the last seconds of the contraction the 65.6 ± 5.8% of the initial value of the fatigue protocol (Fig. 1).

![Fig. 1 Means ± 1 SEM (n = 9) alteration (%) of the soleus EMG during the development of fatigue, as the MVC decreases from 100% to 50%. The EMG values are normalized relatively to the initial measurement at the beginning of the sustained contraction. The asterisks on the top indicate significant difference between the mean values that the short vertical lines designate (p < 0.05).](image-url)
The statistical analysis revealed a significant decline of the soleus EMG activity compared to the initial value after the torque fell to 80% of MVC or lower (F = 14.6, p < 0.05). At the end of the protocol the tibialis anterior activity decreased to the 88.1 ± 6.7% of the initial value (Fig. 2). Nevertheless this difference was not significant (F = 1.34, p > 0.05). On the other hand, the ratio of the tibialis anterior EMG to the soleus EMG increased significantly (F = 2.99, p < 0.05) when the MVC reached 50% (Fig. 3). This fact indicates that the rate of decrease of the soleus EMG was greater than for the tibialis anterior.

The ratio of the soleus EMG to the produced torque showed a significant increase (130.0 ± 11.9%) at the end of the fatigue protocol (F = 5.57, p < 0.05) (Fig. 4). On the other hand, the ratio of the tibialis anterior EMG to the torque output during the maximal heel lifting was affected by the effects of fatigue (F = 11.9, p < 0.05) and more specifically, the ratio remained unaltered at the initial phases of the fatigue protocol but increased after the torque dropped to 60% of MVC (Fig. 5).

Discussion

The findings of the present study indicate that during a sustained maximal voluntary heel lifting, the decline of the MVC, which indicates the appearance of fatigue, is accompanied by a significant reduction of the soleus EMG and not significant of the tibialis anterior EMG. Additionally, when calculating the ratio of the tibialis anterior to the soleus EMG during fatigue, it was shown that this ratio was increased at the end of the fatigue protocol.
The decrease of the torque output and the agonist EMG is well documented and is in agreement with previous studies [41,42]. Peripheral factors such as insufficient function of the neuromuscular junction [10], or lacking of cross-bridge excitation coupling [9], might be involved. Besides, there are also central factors that may be involved. It has been suggested that the reduction in EMG might be a result of inhibition of some motor units (probably fast ones) [22] or decrease in the firing rate of the motor units [30]. There is experimental evidence that fatigue affects the discharge rate of afferents, such as la afferents from the muscle spindles [27], Ib afferents from the Golgi tendon organs [21] and small diameter afferents (III and IV group) [13]. Hence, these afferents might be involved supplying the central nervous system with feedback about the metabolic and contractile status of the muscle. The further regulation of the discharge rate of the motoneurones can be achieved through the involvement of spinal and supraspinal mechanisms. For example, the mechanism of the recurrent inhibition is at least partly responsible for the increase or decrease of EMG during submaximal or maximal fatigue, respectively [25,26]. Other mechanisms such as presynaptic inhibition, reciprocal inhibition and autogenic inhibition may also be involved, though this has not been studied yet.

The alteration of the ratio of soleus EMG to the produced torque in this study is in agreement with previous studies concerning either isometric [35,42], dynamic [15] or isokinetic contractions [43]. The increased ratio reveals that during fatigue more muscle activity is required for a certain amount of torque production. This is also observed during submaximal fatigue, since the EMG increases while the torque remains constant [7,26]. Therefore it seems that when fatigue is present and the torque output decreases from the initial maximum, the central nervous system is increasing the motor unit recruitment per unit of torque production, independent of the intensity of the contraction. This would indicate that the recruited motor units during fatigue are less effective concerning the producing torque. Nevertheless, the decreased muscle conduction velocity which is observed during fatigue also has an effect on the EMG increase [3].

Concerning the level of co-activation of the tibialis anterior acting as an antagonist during heel lifting, the results of the current study support the notion that fatigue induced by a sustained isometric heel lifting has similar effects on the antagonistic muscle activity, compared with a fatigue intermittent protocol of maximal intensity applied to the knee extensors [35]. More specifically, in the current study the antagonist EMG had a tendency to decrease, but not significantly. However, the ratio of the antagonist to the agonist EMG increased at the end of the fatigue protocol. It has been reported that this is not the case for the knee extensors, although it should not be neglected that the fatigue protocol used in this case included intermittent maximal contractions (3 sec contraction with 50% duty cycle) [35]. The observed increase in the ratio of the antagonist to the agonist EMG in combination with the fact that both the agonist and antagonist muscle EMG decreased, indicates that the decline rate of the antagonist EMG was lower when compared to the decline rate of the agonist EMG. This can be assumed to be a situation of increased antagonist muscle co-activation, although the antagonist EMG in absolute values did not change significantly or, to be more precise, decreased slightly. Additionally, the increased ratio of the tibialis anterior EMG to the net produced torque during the heel lifting, indirectly supports the idea of increased antagonist co-activation relative to the agonist torque production, a finding that is in agreement with data for the knee extensors [35]. The increased ratio of the antagonist to the agonist EMG during the contraction would indicate an increased opposing torque of the antagonist muscles relative to the produced torque from the agonists at the end of the fatigue protocol. This can be true only if the EMG - torque relationship for the soleus and tibialis anterior muscle is linear during fatigue, as it is during non-fatigue state [28]. The results presented in this study cannot prove or reject this hypothesis and therefore, caution is warranted in this interpretation.

It could be argued that the registered activity in the tibialis anterior is in fact cross-talk obviously from the triceps surae muscle. According to De Luca and Merletti [6] as much as 16.6% of the tibialis anterior EMG can be recorded from elsewhere in the leg. To eliminate this possibility, before the fatigue protocol, special care was taken to check any potential of cross-talk, using simple manual resistance tests for tibialis anterior and triceps surae muscles [44]. These tests showed a negligible amount of cross-talk (<5%) [39,44], a result that could be attributed to the selected inter-electrode distance (2 cm). According to studies in a muscle model, using this inter-electrode distance, the detection depth for the 95% of the signal amplitude is 18 mm [44]. Additionally, there was no noticeable adipose tissue over the tibialis anterior muscle, a fact that is limiting the chance of cross-talk [39,40].

Previous studies [34,35] have explained their results concerning co-activation of the agonist and antagonist muscles during fatigue based on the common drive theory [5], which supports the idea that both agonist and antagonist activation are controlled by a common neuronal drive. Accordingly, the simultaneous activation of the agonist and antagonist increases the stiffness of the joint but decreases the net torque production. The movement generation is achieved by reciprocal activation of the agonist and antagonist muscles by the central nervous system [37]. Hence, it seems that the central nervous system is shifting between reciprocal activation (less stiffness vs. more net torque) and co-activation (more stiffness vs. less net torque) depending on the requirements of the movement [5]. Concerning fatigue and the results of the current study, the common decrease of the agonist and antagonist activity can be supported by the common drive theory. However, the ratio of the antagonist to the agonist EMG activity was not constant, and revealed that the agonist EMG decreased more at the end of the fatigue protocol compared to the antagonist EMG decrease. Although it has been argued that the voluntarily induced co-activation of the antagonist and agonist muscles is accompanied by reduced levels of reciprocal inhibition [31], this argument is difficult to be generalized to the case of co-activation induced by fatigue.

The functional significance of co-activation of the antagonist and agonist muscles is increase of stiffness around the joint by exerting an opposing torque [33]. This in turn contributes to the stabilization and protection of the joints [16], a property that is definitely important for the ankle joint.

In conclusion, our findings indicate that although fatigue induced by a maximal isometric heel lifting resulted in a significant reduction in agonist (soleus) and non-significant in the an-
tagonist (tibialis anterior) EMG, the rate of this decrease for the soleus muscle was lower compared to the rate of the tibialis anterior. This fact needs to be considered, since it might reflect a relatively higher co-activation pattern and thus a higher opposing torque from the antagonist muscle at the end of the fatigue session.

References

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