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A simple test of muscle coactivation estimation using electromyography

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Abstract

In numerous motor tasks, muscles around a joint act coactively to generate opposite torques. A variety of indexes based on electromyography signals have been presented in the literature to quantify muscle coactivation. However, it is not known how to estimate it reliably using such indexes. The goal of this study was to test the reliability of the estimation of muscle coactivation using electromyography. Isometric coactivation was obtained at various muscle activation levels. For this task, any coactivation measurement/index should present the maximal score (100% of coactivation). Two coactivation indexes were applied. In the first, the antagonistic muscle activity (the lower electromyographic signal between two muscles that generate opposite joint torques) is divided by the mean between the agonistic and antagonistic muscle activations. In the second, the ratio between antagonistic and agonistic muscle activation is calculated. Moreover, we computed these indexes considering different electromyographic amplitude normalization procedures. It was found that the first algorithm, with all signals normalized by their respective maximal voluntary coactivation, generates the index closest to the true value (100%), reaching 92 ± 6%. In contrast, the coactivation index value was 82 ± 12% when the second algorithm was applied and the electromyographic signal was not normalized ($P < 0.04$). The new finding of the present study is that muscle coactivation is more reliably estimated if the EMG signals are normalized by their respective maximal voluntary contraction obtained during maximal coactivation prior to dividing the antagonistic muscle activity by the mean between the agonistic and antagonistic muscle activations.

Key words: Muscle coactivation; Electromyogram; Muscle cocontraction; EMG index

Introduction

Muscle coactivation, the simultaneous activation of agonist and antagonist muscle groups around a joint (1,2), is an important and common strategy for the control of voluntary movement in humans and has been experimentally observed during a wide variety of conditions including locomotion (3), isometric and functional activities (4), low and high accurate pointing tasks (2,5), upright standing (6), central nervous system impairment (7,8), and lumbopelvic stabilization (9), among others. In the control of movements, it has been suggested that muscle coactivation modulates the impedance of a joint, mainly stabilizing the joint (2).

In experimental conditions, coactivation is most often estimated by comparing the amplitude of the myoelectric activity of muscles that generate opposite torques during a task. Despite the fact that a muscle can be active more than another and yet generate less force (or vice versa) due to a number of factors, including pennation angle and fiber length, and also considering that caution should be used if the relationship between force and electromyographic (EMG) signal is not established for all the muscles investigated, various indexes of coactivation based solely on the EMG signal have been proposed (10-17). However, it is not known how coactivation based on EMG is reliably estimated with such indexes. The goal of this study was to test the reliability of the estimation of muscle coactivation using EMG.

Subjects and Methods

Subjects

Ten volunteers with no history of neuromuscular disorders (6 men and 4 women; mean ± SD: age = 25 ± 5 years, height = 176 ± 10 cm, and mass = 74 ± 11 kg) participated in the experiment. The study was conducted in accordance

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with the Helsinki Declaration and was approved by the Aalborg University Ethics Committee (VN2003/61). The volunteers received information about the experiment and gave written informed consent to participate.

Indexes of coactivation

Two indexes were selected to represent most of the coactivation indexes (CI) based on the amplitude of the EMG signal employed in the literature (10-17). In the first index (CI_1), the antagonistic muscle activity (the lower EMG amplitude between two muscles that generate opposite joint torques) is divided by the mean between the agonistic (EMG_{AG}) and antagonistic (EMG_{ANT}) muscle activations (10). The second index (CI_2) is obtained by calculating the ratio between antagonistic and agonistic muscle activation (11). The formulas for these indexes are shown below:

$$CI_1 = 2 * \frac{EMG_{ANT}}{EMG_{AG} + EMG_{ANT}} * 100 \quad (1) \qquad CI_2 = \frac{EMG_{ANT}}{EMG_{AG}} * 100 \quad (2)$$

The method for EMG amplitude normalization used when coactivation is calculated varies in the literature. The EMG signals have often been normalized by the maximum voluntary isometric contraction (MVIC) (13,18), the maximum voluntary isometric coactivation value (MVICa) (10), or have not been normalized (19,20). Here we will investigate how these three methods affect the CI.

Protocol

Coactivation around a joint that does not move and with no external moments implies that the net joint moment is equal to zero. Such task can be performed at any level of muscle activation, as long as the joint does not move (full coactivation). Thus, the volunteers were instructed to activate simultaneously elbow extensor and flexor muscles in order to achieve full coactivation at various levels of muscle activation. In order to allow the volunteers to maintain a targeted level of muscle activation, established for that task, the EMG linear envelope from the medial belly of the biceps brachii muscle was shown in real time on an oscilloscope. The volunteers practiced the task prior to data collection by performing 3 to 4 times 2 s of full coactivation at various muscle activation levels. The data for 1 volunteer was not included in the study because she was unable to perform the task. The subjects rested for 1 min between two practicing trials. A full coactivation was then carried out for 4 s, at different biceps muscle activation levels (25, 50, and 75%) of the biceps muscle EMG activity achieved during a maximal effort muscle activation (100%) keeping a full coactivation. At the beginning of the session, the volunteers performed a coactivation at a maximum effort so the EMG activity from the medial head of the biceps brachii muscle could be used as the reference for the biofeedback and the peak EMG from the biceps and triceps brachii muscles could be used for EMG normalization. For a second EMG normalization procedure, the peak EMG from a maximal isometric voluntary contraction for elbow flexors and elbow extensors was used.

Setup

The volunteers were comfortably seated with the arm supported at 90° abduction and 90° elbow flexion. A pair of surface electrodes (Medicotest 72001-k, Denmark) was placed in the direction of the muscle fibers (2-cm apart) on shaved, cleaned skin. On the biceps brachii muscle, the electrodes were placed on the medial and lateral head, on the lead-line between the acromion and the fossa cubit at 1/3 from the fossa cubit. On the triceps brachii muscle, the electrodes were placed on the lateral and medial head - 1 cm lateral to the lead-line just on the midpoint between the acromion and the olecranon process. The EMG signals were bandpass filtered (2nd order, 20 to 500 Hz), amplified 1000 times (CounterPoint MK2, Dantec, Denmark) and sampled at 1024 Hz.

Data analysis and statistics

The digital EMG signal was band-pass filtered (2nd order, zero-phase-lag Butterworth, 20 to 400 Hz), full wave rectified, and smoothed (low-pass, 4th order, zero-phase-lag Butterworth filter with a 3-Hz cutoff frequency). From the 4 s of coactivation, a 1-s interval was extracted in which the squared difference between the acquired EMG and the targeted EMG intensity was the lowest. This procedure allowed us to select the 1-s window where the subject's EMG activation was closest to the target displayed on the oscilloscope. The difference between the target versus the actual EMG activation level was calculated as percent error.

Data are reported as means ± SD. Three-way repeated analyses of variance (ANOVA) were used to examine the effects of CI (CI_1 and CI_2), normalization procedures [non-normalization (NN), normalization by the MVIC (NMVIC), and normalization by the MVICa (NMVICa)], and muscle activation level (25, 50, 75, 100%) on the coactivation index. When ANOVA was found to be significant, the Student-Newman-Keuls *post hoc* test was used for multiple comparisons. The level of significance was set at $P < 0.05$.

Results

Figure 1 shows representative plots of the EMG time series for different target levels and the respective error for one subject. The means ± SD for the 10 subjects for the 25, 50, 75, and 100% levels were 3 ± 3, 3 ± 1, 5 ± 3, and 10 ± 6%, respectively. These low values suggest very consistent patterns of activation if we consider that the subjects had as feedback the EMG values of their muscles, which are well known to be very variable. Based on the variability of the results, we suggest that the subjects were familiarized with the task and that they performed it as they were asked

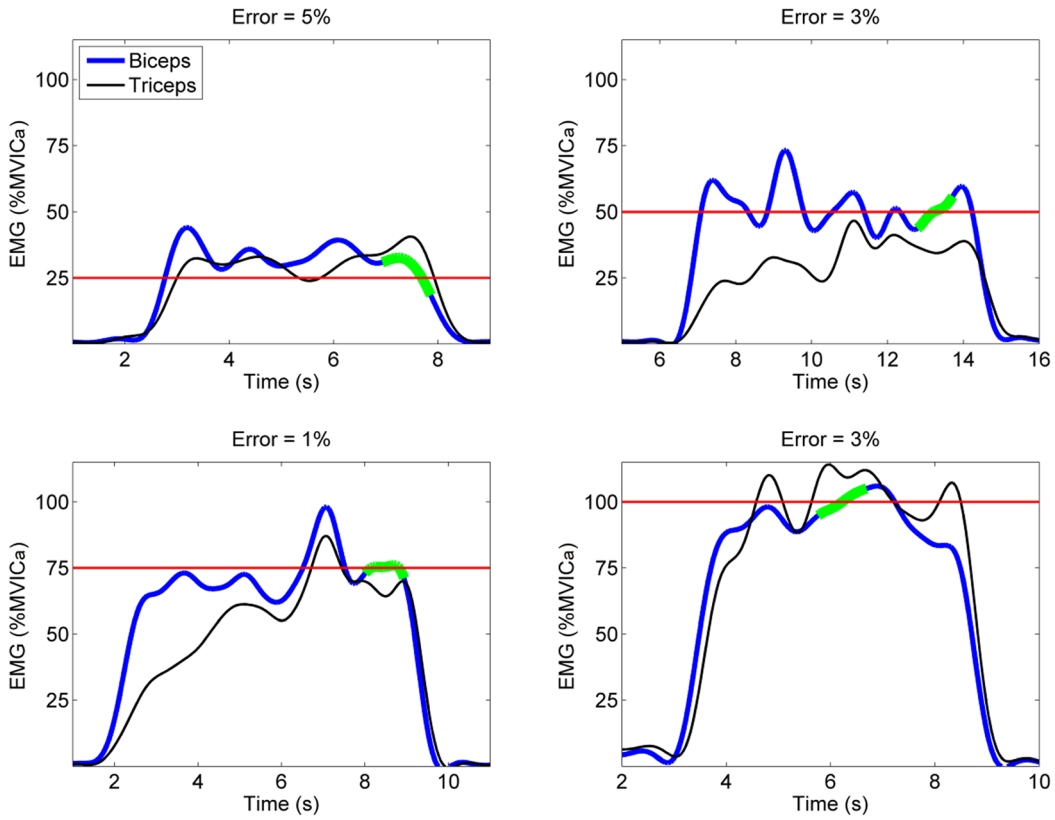

Figure 1. Determination of how far (% error) the actual electromyographic (EMG) signal is from the desired one for different target levels (25, 50, 75, and 100% of muscle activation, horizontal line) for one subject during the task. The thicker line for biceps muscle EMG represents the 1-s selected interval. MVICa = maximal voluntary isometric coactivation.

to. Although the variability throughout the 4-s contractions was quite large, as shown in Figure 1, it is important to emphasize the fact that the volunteers were requested to maintain the contraction at the target for 2 s only in order to avoid fatigue (as instructed in the familiarization training). Moreover, the fact that a 1-s window was selected makes the behavior during the other 3 s less important for the present investigation.

The mean values and standard deviation of the two coactivation indexes considering the different normalization methods applied are shown in Figure 2.

ANOVA for the factors algorithm (CI_1 and CI_2), normalization procedures (NN, NMVIC, NMVICa), and muscle activation levels (25, 50, 75, and 100% MVIC) revealed a main effect of algorithm ($F(1,9) = 137$, $P < 0.001$) and a main effect of normalization procedure ($F(2,18) = 3.9$, $P = 0.04$). The *post hoc* analyses revealed that the CI calculated by applying Equation 1 (CI_1) had a higher value than that calculated by applying Equation 2 (CI_2) for all normalization procedures, with respective pooled means ± SD values of 87 ± 10 and 78 ± 15% ($P < 0.03$). Moreover, the CI was higher when the EMG signal was normalized by the NMVICa compared to the CI obtained when the EMG was not normalized for both indexes (NMVICa > NN; $P < 0.04$); pooled means ± SD respectively equal to 89 ± 6 and 77 ± 12%. No interactions among factors were found by ANOVA.

Discussion

In this study, we verified the accuracy of CI based on EMG recordings. Our results showed that none of the investigated CI was able to accurately estimate the level of coactivation, which was theoretically known. The present data shows that muscle coactivation is more reliably estimated if the EMG signals are normalized by their respective maximal voluntary contraction obtained during maximal coactivation, prior to dividing the antagonistic muscle activity by the mean between the agonistic and antagonistic muscle activations. The inability of surface EMG electrodes to accurately record all motor units equally, the fact that not all muscles involved are recorded, and data processing limitations certainly contributed to coactivation values

different from the theoretical expected ones. Furthermore, several muscles act at a joint and it seems to be crucial to consider the contribution of all muscles involved, as well as possible nonlinearities to reliably calculate coactivation. Note, however, that these very same limitations are present in the great majority of studies that employ EMG signals to estimate muscle coactivation (e.g., 2, 10-12, 3, 13, 14, 7, 15, 8, 16, 17, 6, 5, 4, 9). Our rationale is that the low accuracy of the CI we found, which certainly resulted from the far from perfect methods employed here (and elsewhere), exposes the limitation of such CI.

When muscles that produce torque in opposite directions are simultaneously activated, they limit the net moment generated at that joint. If the resultant contraction is an active maintenance of a static joint positioning, the muscles surrounding the joint are in full coactivation at any point from the minimum to the maximum muscle activation. However, any estimation of coactivation at that point must show that the net torque around that joint is zero, which is represented by the highest score when coactivation is measured.

In the present study, there was no joint movement or resultant external torques, implying that the volunteers had their muscles around the elbow in full coactivation, meaning zero net torque. In that case, any elbow flexor muscle is an antagonist to an elbow extensor and vice versa. In order to have a reference, the higher and the lower EMG intensity levels were considered for agonistic and antagonistic muscle activation, respectively. In theory, a CI at 100% would be expected in all conditions studied in the present investigation. It was shown that a CI that accounts for the antagonist torque generated at the joint and also for the additional agonist torque required to compensate for it, as proposed in CI_1, is more reliable because the index value reaches values closer to the maximum score. Moreover, there was no significant difference in the CI calculated for different EMG levels, indicating that neither CI is affected by the muscle activation level.

In order to minimize inter-subject EMG differences, the EMG signal amplitude was normalized. Various EMG amplitude normalization methods have been used in the literature when a CI is calculated (10,12,16,17). In the present study, it was shown that the EMG normalization procedure also influences the CI outcome. When the EMG amplitude was normalized by the peak EMG signal obtained during a maximal full coactivation, the CI approached the expected values for that motor task better than when the EMG amplitude was not normalized. Possibly, this is because this normalization procedure is related to the targeted task. That is, full coactivation at various muscle activation levels.

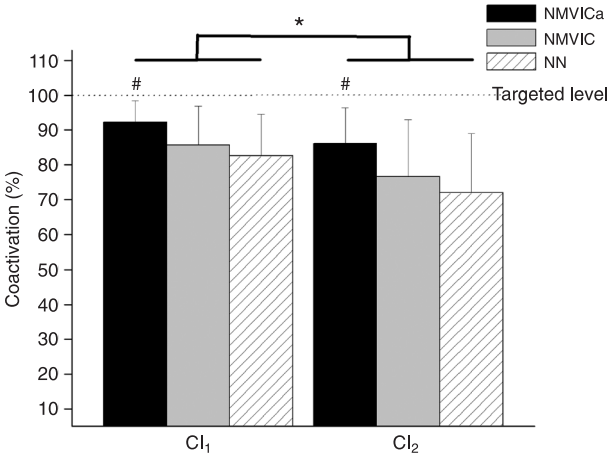

Figure 2. Coactivation indexes (CI_1 and CI_2) based on electromyographic (EMG) signals (data pooled from 25, 50, 75, and 100% of muscle activation levels) non-normalized (NN), normalized by the maximal voluntary isometric contraction (NMVIC), or normalized by the maximal voluntary isometric coactivation (NMVICa). Data are reported as means ± SD. *CI_1 significantly higher than CI_2 for all normalization procedures ($P < 0.001$). #Coactivation index normalized by NMVICa significantly higher than the non-normalized coactivation index for both indexes ($P < 0.04$; Student-Newman-Keuls test).

The present results were derived from a controlled task designed to validate the different methods to quantify coactivation. The task was always performed in a static condition and at the same position (90° elbow flexion). Since muscle force is affected by its length and velocity (21) and during dynamic tasks the length and velocity of the muscles vary differently, these two factors might limit even more the use of CI based on the EMG signals.

Furthermore, the low accuracy of the CI suggests that coactivation indexes should be interpreted with caution and any methodological difference in the calculation should be considered before a comparison of different studies is performed.

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