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ORIGINAL ARTICLE

Static stretching of the pectoralis major decreases triceps brachii activation during a maximal isometric bench press

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ABSTRACT

BACKGROUND: Static stretching (SS) not only increases the range of motion (ROM) of the stretched muscle but can also enhance the ROM of homonymous and heteronymous contralateral muscles. Whereas prolonged SS can lead to performance impairments of the stretched muscle, deficits in muscle activation have not been investigated with non-stretched muscles that contribute to a task such as a bench press. The purpose of this study was to examine the effect of prolonged SS of the pectoralis major muscle on the synergic activation of the pectoralis major (PM) and triceps brachii (TB) muscle during an isometric bench press action.

METHODS: Fourteen young, healthy, resistance-trained men had their shoulder complex passively stretched (horizontal abduction) with six stretches of 45-sec each, with 15-sec rest between each stretch at an intensity of 70-90% of the point of discomfort. The integrated electromyography (IEMG) activity and the median frequency (Mfreq) of the PM and TB were monitored during a 3-sec of maximal isometric bench press action.

RESULTS: Passive shoulder ROM significantly increased 5.5%. Both PM (32.60%) and TB (12.60%) IEMG decreased from pre- to post-SS. There were no significant differences between pre- and post-SS for RPE and Mfreq.

CONCLUSIONS: Prolonged SS of a muscle (PM) can negatively impact the activation of auxiliary muscle (TB) involved with the same multi-joint action, which can have implications for individuals who are training or competing.

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Key words: Fatigue - Applied kinesiology - Neurosciences.

Bench press is a multi-joint exercise that is commonly used for improving upper body strength, and requires coordination of a number of joints during the upper body movement. Consequently, bench press exercise requires a well-controlled activation from prime mover muscles to produce a correct movement pattern. This movement pattern

is influenced by individual muscle and inter-muscular activation derived from the central nervous system.¹ Electromyographic²⁻⁸ and ultrasound⁹ studies found that the pectoralis major (PM), anterior deltoid (AD), and triceps brachii (TB) muscles contribute to bench press movement with similar intensity levels. Hence, “interference” from factors such as

fatigue or stretching, of a single-joint (or a muscle group), might affect synergistic muscle activation.

It is well documented that prolonged static stretching (SS) can improve flexibility but impair subsequent performance.¹⁰⁻¹² These performance reductions, of stretched muscles, can originate from neurophysiological (*i.e.*, mechanoreceptors of the skin, muscle and joint proprioception), endocrine, cellular (structural changes such as titin, nebulin), or mechanical (*i.e.* stiffness, torque-length characteristics) factors.^{10, 13-18} However, no known scientific literature exists, which have investigated non-local stretching effects on an auxiliary muscle that inserts on a similar joint and works synergistically with the target muscle.

Stretching a target muscle group can improve the range of motion (ROM) of non-local muscles or joints.^{19, 20} In 2015, Behm *et al.* demonstrated that static or dynamic stretching of the shoulders improved ROM of the hip flexors (5.2%), while similar stretching of the hip flexors increased shoulder ROM (8.2%) without force impairments of the non-stretched muscles. Chaouachi *et al.* found that unilateral static or dynamic stretching of the hip flexors increased contralateral hip flexor ROM (5.7-8.4%) with no isokinetic torque impairments in the contralateral non-stretched limb. However, prolonged unilateral static stretching of the plantar flexors²¹ or static stretching of the shoulders²² impaired subsequent maximal concentric jump performance. Thus, while prolonged static stretching can have global ROM effects, the effects on non-local muscle performance are conflicting. Therefore, further studies are necessary to clarify whether prolonged static stretching can adversely affect non-stretched muscle activation and whether these effects are prevalent in a synergistic or auxiliary muscle.

Thus, the objective of the present study was to investigate the effect of prolonged SS of the PM muscle on the synergic activation of the pectoralis major and triceps brachii muscle during an isometric bench press action. It was hypothesized that prolonged stretching of the PM would inhibit activation of the PM and TB

due to the reported neural effects of stretching.^{10, 11, 19, 23, 24}

Materials and methods

Based on a statistical power analysis derived from IEMG data from a pilot study (N.=5), a sample size of ten subjects would be necessary to achieve an alpha level of 0.05 and a power (1- β) of 0.80. Therefore, 14 young, healthy, resistance-trained men (age: 23.4 \pm 5.0 years, height: 178.2 \pm 5.8 cm, and total body mass: 81.8 \pm 9.6 kg, biacromial width: 37.4 \pm 2.0 cm) were recruited to participate in the current study. The subjects had, at least, four years of experience with the bench press exercise with no previous surgery or history of injury with residual symptoms (pain) in the upper limbs within the last year. The University Research Ethics Committee approved this study (#03/2014) and all subjects read and signed an approved informed consent document.

Experimental procedures

All subjects were right-arm dominant based on their preferred arm to write. Volunteers attended one laboratory session and refrained from performing upper body exercise other than activities of daily living for at least 48 hours prior to testing. Subjects performed a 5-min cycle warm-up at 70 rpm, and a familiarization session with five submaximal isometric contractions of the bench press with 30-sec rest between trials.

After the warm-up and familiarization, all subjects lay supine on a weight-lifting bench and grasped a barbell at twice the biacromial width, at 90° of elbow joint flexion and shoulder joint abduction. They performed three trials of 5-sec maximal isometric contractions against a locked Smith machine with a rest period of 5-min between trials, before and after a SS protocol. All trials were performed with maximal or near maximal perceived intensity as all subjects reported a rating of perceived exertion between 9-10. They also received verbal encouragement during all trials. All measurements were performed between 9 a.m. and 12 p.m., by the same researcher.

Measures

PASSIVE ROM

Subjects adopted a supine position with knees flexed, and the lumbar spine supported on a bench. The fleximeter (Sanny, Brazil) was placed on the dominant arm, above the elbow, and the hands were placed together to set zero degrees on the fleximeter. Then, each subject performed three trials of the passive ROM for a horizontal abduction movement of the shoulder joint, with a rest period of 10-sec between trials before and after the SS protocol. The maximal passive shoulder ROM value was considered with the fleximeter (sensitivity of 1°).

SURFACE ELECTROMYOGRAPHY (sEMG)

Participants' skin was prepared before placement of the sEMG electrodes. Hair at the site of electrode placement was shaved, abraded, and the skin was cleaned with alcohol. Bipolar passive disposable dual Ag/AgCl snap electrodes were used which were 1 cm in diameter for each circular conductive area with 2-cm center-to-center spacing. These were placed on the dominant limb over the longitudinal axes of the PM at the mid-belly in the direction of the muscle fibers (sternal portion),²⁵ and 4-cm from the lateral to the midline of the TB at 50% of the distance between the posterior crista of the acromion and the olecranon.²⁶ A ground electrode was placed on the right patella. The sEMG signals of the PM and TB were recorded by an EMG acquisition system (EMG630C, EMG system Brasil, São José dos Campos, Brazil) with a sampling rate of 2000 Hz using a commercially designed software program (DATAQ Instruments Hardware Manager, DATAQ Instruments, Inc., OH, USA). The sEMG activity was amplified (bipolar differential amplifier, input impedance 2 MΩ, common mode rejection ratio >100 dB min (60 Hz), gain ×20, noise >5 μV), and analog-to-digital converted (12 bit). EMG signals were collected during maximal isometric contractions against a fixed bench press exercise.

All sEMG data were analyzed with a customized Matlab routine (MathWorks Inc., USA). The digitized sEMG data were band-pass filtered at 20-400 Hz using a fourth-order Butterworth filter with a zero lag. For muscle activation time domain analysis, RMS (150 ms moving window) was calculated for all trials. Then, the first second was removed from sEMG RMS to avoid body adjustments, and the following 3-sec of each trial were integrated (IEMG). For the frequency analysis, the same sEMG data (3-sec) was analyzed by a short-time Fourier transform and the median frequency (MFreq) of the spectrum was analyzed.

RATING OF PERCEIVED EXERTION (RPE)

RPE (CR-10 scale) was assessed after bench press condition (pre- and post-SS). Standard instructions and anchoring procedures were explained during the familiarization session. Subjects were asked to use any number on the scale to rate their overall effort for each condition. A rating of 0 was associated with no effort and a rating of 10 was associated with maximal effort and the most stressful exercise ever performed. Subjects were shown the scale 30-min after each condition and asked: "*How was your workout?*"²⁷

Intervention

STATIC STRETCHING (SS) PROTOCOL

Since it has been recommended that less than 60-sec of static stretching is less likely to induce subsequent performance impairments,¹⁰⁻¹² a more prolonged SS protocol of six stretches of 45-sec each, with 15 sec of rest between each stretch was incorporated. Based on prior research²⁸ that induced impairments, the intensity of each SS was 70-90% of the point of discomfort (POD), where 0 means "no stretch discomfort at all" and 100 "the maximum imaginable stretch discomfort." The participants were seated on a chair, with the trunk and waist secured by three straps to prevent extraneous movements, hands behind the head, and arms raised above the shoulder

joint. Then, a researcher passively retracted the scapula and the shoulders to the maximal ROM of the horizontal abduction position. This scapular retraction primarily stretched the PM with no change in length of the TB. The SS protocol was applied and controlled (POD) by the same strength and conditioning researcher.

Statistical analysis

The normality and homogeneity of variances within the data were confirmed with the Shapiro-Wilk and Levene's tests, respectively. To compare the effects of the SS protocol on the shoulder joint passive ROM and RPE, we used a paired *Student's t-test* before and after the SS protocol. To test whether the SS protocol resulted in muscle activity differences (IEMG and MFreq), a Paired Student's *t-test* was used between pre- and post-intervention to PM and TB, separately. Cohen's formula for effect size (*d*) was calculated, and the results were based on the following criteria: trivial (<0.2), small (0.2-0.6), moderate (0.6-1.2), large (1.2-2.0), and very large (>2.0) effects. Interrater reliability was assessed for the researcher who positioned and evaluated all sEMG for all muscles and conditions. Reliability was operationalized using the following criteria: <0.40 poor; 0.40-0.75 satisfactory; ≥0.75 excellent.²⁹ The ICCs ranged between 0.97 and 0.99 (excellent) for all dependent variables. An alpha of 5% was used to determine statistical significance.

Results

Passive ROM significantly increased from pre- to post-static-stretching (mean±SD: 119.80±8.0° to 125.80±8.0°, respectively; $P=0.003$; $d=0.84$; $\Delta\%=5.50$).

Both PM ($P<0.001$, $d=2.02$, $\Delta\%=32.60$) and TB ($P=0.048$; $d=0.28$; $\Delta\%=12.60$) IEMG decreased from pre- to post-SS (Figure 1). There were no significant MFreq differences between pre- and post-SS for both muscles (PM: $P=0.137$; $d=0.26$; $\Delta\%=3.80$, and TB: $P=0.282$; $d=0.22$; $\Delta\%=3.14$). There were no significant RPE differences between pre- and post-SS (8 ± 1 and 9 ± 1 , respectively, $P>0.05$).

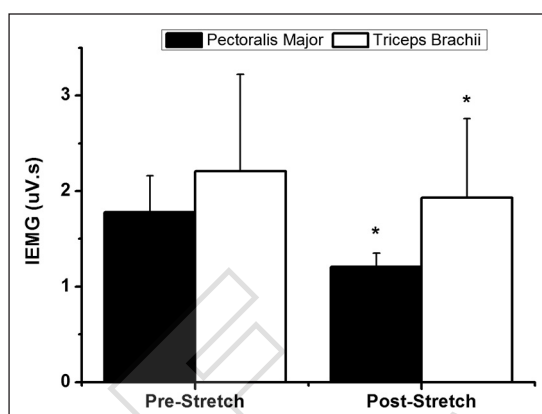


Figure 1. — Mean and standard deviation of the IEMG during the maximal isometric bench press. *Significant difference between pre- and post-SS protocol, $P<0.05$.

Discussion

In agreement with the bulk of the scientific literature,¹⁰⁻¹² prolonged SS of the PM resulted in muscle activation (IEMG) deficits. However, the more important finding of the present study was that prolonged SS of the PM induced decrements in TB IEMG activity. Similarly, non-local increases in ROM have been documented with contralateral lower limbs²⁰ as well as the upper and lower body.¹⁹ This is the first study to demonstrate the non-local effects of prolonged SS on an auxiliary muscle when performing a multi-joint task also involving the stretched muscle.

Prior stretching studies induced non-local ROM improvements, with no non-local SS-induced performance impairments in force^{19, 20} or muscle activation.^{19, 30} Prolonged SS of the stretched muscle can inhibit sEMG,²³ H-reflex activity (down-regulation of motoneuronal excitability)²⁴ and muscle spindle afferent output.³¹ Muscle afferents (group III and IV) can inhibit corticospinal pathways,³² diminishing central drive to the stretched muscle and potentially to the non-stretched muscles.³² The TB sEMG deficits in the present study might be attributed to the closer proximity of spinal segmental inhibitory reflex circuitry with the PM and TB muscles, and the possible common drive between muscles (intermuscular coordination) to produce a synchronized bench press technique. Lateral and medial pectoral nerves

(innervate PM), as well as the radial nerve (innervate TB), emanate from the brachial plexus, which have trunks that all intersect between C5-8 and T1.³³ The closer proximity of the ipsilateral PM and TB spinal neurons may make them more susceptible to the neighboring inhibitory interneurons compared to the greater spinal distances of the contralateral or upper and lower body sensory and motor neurons examined in previous studies.

A similar anatomical rationale can be postulated if the SS-induced inhibition was supraspinal-mediated as the cortical sensory and motor neurons and associated inhibitory interneurons for the ipsilateral PM and TB would have a closer proximity than for contralateral homonymous or upper versus lower body muscles. Gandevia³⁴ provided extensive evidence that muscle afferents can impair cortical excitability. Muscle spindle,³⁵ skin and subcutaneous afferents³⁶ contribute to long loop reflexes that can influence cortical activation of the affected and non-local muscles.³⁷ Accommodation effects of SS can reduce the discharge frequency from Ia afferents of intrafusal stretch receptors, which can contribute up to 30% of the motoneuron excitation³⁴ adversely affecting muscle activation.

Additionally, the lack of changes in the median frequency from the sEMG data may be related to the total time used to calculate the frequency spectrum (3-sec), and consequently, the median frequency may have been altered throughout the sEMG data. Another important consideration is the non-linearity of the force-EMG relationship, which is particularly prominent at the high force portion of the EMG-force relationship.³⁸ Thus, the extent of EMG activation deficits cannot be directly inferred as potential force deficits. Force measures were not evaluated in this study, however, the similarity of the RPE values in both conditions (pre- and post-SS) might be considered as an indirect way of measuring physical effort.

The inhibition of TB sEMG activity with SS of the PM in the present study has implications for individuals who are training or competing. Prolonged SS-protocol of a muscle contributing to a functional multi-joint task can impair

other assistive muscles and thus should not be undertaken. As there was no prior aerobic warm-up or subsequent sport or task specific dynamic activity following SS, the reported decreased EMG activity might not be as substantial or evident if a full warm-up was instituted.¹¹

We recognize that this study has some limitations. We did not control for skinfold thickness of the sEMG detection area, that is considered to be a low-pass filter, and there may have been some inherent differences in the musculotendinous tightness between subjects. We also used a healthy, non-athletic population, and our results are not generalizable to other conditions, populations, or athletes. The blood flow during the stretching activity was not measured, and it may have a deleterious effect on the subsequent activity. However, during intermittent activities, this effect may be considered irrelevant. Finally, we did not control the maximal isometric force during all trials, however, the main comparison was between conditions, and there is no linear relationship between sEMG and force at high levels of intensity.

Conclusions

The present findings suggest that prolonged SS of one muscle (PM) can negatively impact the activation of another muscle (TB) involved with the same multi-joint action. The non-local EMG depression in the present study in combination with prior literature demonstrating contralateral and upper *versus* lower body effects emphasizes that prolonged stretching of a muscle can have diffuse effects throughout the body. Whereas prior non-local effects of SS research demonstrated increased ROM with no significant muscle activation, force impairments or high perception of effort, the present findings suggest that SS-induced muscle activation impairments could be more prevalent with a muscle in closer anatomical proximity or function.

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