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EFFECTS OF DIFFERENT ANTAGONIST PROTOCOLS ON REPETITION PERFORMANCE AND MUSCLE ACTIVATION – ORGINAL RESEARCH

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Abstract

Objective: To investigate the acute effects of different antagonist manipulation protocols on maximal repetition performance and muscle activation during seated row (SR) exercise.

Methods: Fifteen men (22.4 ± 1.1 years old, height 175 cm ± 5.5, weight 76.6 kg ± 7, and 12.3 ± 2.1 of body fat percentage) with previous resistance training experience (3.5 ± 1.2 years) performed four experimental protocols: (TP) one set to repetition failure of SR exercise; (AS) Antagonist static stretching for the pectoralis major (PM) followed by one set of SR; (PNFA) Proprioceptive neuromuscular facilitation for PM followed by one set of the SR; (APS) One set of the bench press with a 10 RM loads followed by one set of the SR. The maximal repetitions and the electromyographic (EMG) signal were recorded for the latissimus dorsi (LD), biceps brachii (BB), triceps brachii lateral head (TL), and PM during the SR.

Results: A significant increase in SR repetition performance was noted for the APS (14 ± 1) versus the TP (9 ± 1.2, $P = 0.0001$), PNFA (10 ± 1.5, $P = 0.001$), and AS (12 ± 1.5, $P = 0.004$) protocols. A significant increase in SR repetitions was also noted for the AS versus the TP ($P = 0.001$) and PNFA ($P = 0.002$) protocols. The muscle activation of the BB and LD were significantly higher during the APS and AS versus the PNFA and TP sessions.

Conclusions: These results suggest that either using the APS or AS approaches can facilitate an increase in SR repetition performance versus traditional resistance exercise sets.

Keywords*: paired set, strength, stretching, coactivation, performance*

Introduction

Resistance training (RT) provides an overload to the musculoskeletal system, leading to an increase in muscle strength [1]. In formulating a RT prescription, it is of the utmost importance to understand the interaction among training variables such as the load, volume, number of exercises, number of repetitions per set, exercise order, number of sets per exercise or muscle group, and the rest interval between sets and exercises [2].

Most functional movements and RT exercises involve some activation of the antagonist muscles in conjunction with activation of the agonist muscles [3]. This phenomenon has been described as coactivation or co-contraction and affects the net joint torque and subsequent movement velocity [4]. Greater activation of the antagonists during a movement produces a braking effect for the agonists in the mechanical expression of force and power [5,6]. Prior studies have incorporated pre-stretching or pre-fatiguing of the antagonist musculature to facilitate the action of the agonists during subsequent movements [7,8]. The stretching or pre-loading of the antagonist musculature may promote neural inhibition of these muscle groups, lowering the ratio of agonist/antagonist coactivation [9], and consequently increasing rotary torque for the agonist musculature [10,11].

One method for achieving antagonist pre-loading during RT is to perform a set for the antagonist musculature immediately prior to a set for the agonist musculature. This model of pre-loading has been referred to as "agonist-antagonist paired set training (APS)" [9]. During APS training, agonist and antagonist muscles are trained "back-to-back", with limited or without rest between paired sets [12]. However, there is insufficient evidence to support this hypothesis, since some authors found deleterious effects on force production of the agonists [11] or observed no changes in the electromyographic (EMG) amplitude normalized by percentage of maximal voluntary contraction of antagonist muscles following different manipulation protocols such as pre-loading or static stretching [5,13,14].

Despite the lack of evidences about the potential training effects of antagonist manipulation protocols, multiple studies with varying methodologies have investigated different aspects of manipulating the antagonist musculature on subsequent movement performance; these have included the application of static stretching of the antagonists during warm-up [7,8], comparison between different types of muscle action (eccentric, concentric, isometric) [15,16], and velocities [10,13]. However, few studies have reported

EMG data for the agonist/antagonist musculature during movements preceded by antagonist manipulation [3,7,8,17].

Further study is warranted on the practical implications of manipulating the antagonist musculature in different ways for acute enhancement of agonist performance that may in turn positively affect longitudinal training outcomes. Additionally, RT protocols that improve acute performance could be a time efficient alternative for coaches and practitioners aiming to optimize the quality of exercise sessions and outcomes. Therefore, the purpose of this study was to investigate the acute effects of manipulating the antagonist musculature via performance of the bench press, static stretching and proprioceptive neuromuscular facilitation stretching for pectoralis major on subsequent maximal repetition performance and muscle activation for the agonist/antagonist muscles during a wide grip seated row (SR) exercise in trained men.

Methods

Participants

Fifteen recreationally trained men participated as subjects in this study (22.4 ± 1.1 years old, height 175 cm ± 5.5, weight 76.6 kg ± 7, and 12.3 ± 2.1 of body fat percentage). All subjects had previous RT experience (3.5 ± 1.2 years), with a mean frequency of four 60-minute sessions per week, using 1- to 2-minute rest intervals between sets and exercises. All subjects completed the Physical Activity Readiness Questionnaire (PAR-Q) and signed an informed consent before participation in this study according to the Declaration of Helsinki. Subjects were encouraged to report for workout sessions fully hydrated and to be consistent in their food intake throughout the duration of the study; and asked to refrain from any upper-body training in the 48 hours prior to each workout session. The study was approved by the university's ethic committee.

Experimental Protocols

This study used a randomized crossover design during which subjects performed four experimental protocols. The protocols were preceded by two testing sessions during which the 10 repetition maximum (RM) was assessed for the bench press (BP) and SR exercises. The four experimental protocols were then instituted on non consecutive days and 72 hours apart in random order and included: 1) Traditional Protocol (TP) - one set to repetition failure of the SR exercise; 2) Antagonist Stretching (AS) - one set of static stretching (40 s) for the pectoralis major followed by one set of the SR; 3) Antagonist Proprioceptive Neuromuscular Facilitation (PNFA) stretching for the pectoralis major followed by one set of the SR; 4) Antagonist paired set (APS) - one set of the BP to repetition failure followed by one set of the SR. The AS protocol involved one set of 40 seconds of static stretching for the pectoralis major (PM) muscle followed by one set of the SR exercise. The PNFA protocol involved one set of 40 seconds (20 seconds of isometric tension and 20 seconds of passive stretch) of the contract-relax PNF stretching technique for the PM, followed by one set of the SR exercise. No rest interval was allowed between antagonist manipulation and the ensuing SR exercise. Dependent variables included the number of repetitions completed and root mean square (RMS) EMG signal for the latissimus dorsi (LD), biceps brachii (BB), triceps braquii lateral head (TL) and pectoralis major (PM) during the SR.

10 Repetition Maximum Testing

In the week prior to performance of the first randomly selected protocol, 10RM loads were tested and re-tested in two sessions for each subject in the BP and the SR (Life Fitness, IL, USA) exercises (Fig. 1). The 10RM was defined as the maximum weight that could be lifted for 10 consecutive repetitions at a constant velocity of 4 seconds per repetition (2 seconds for the concentric phase and 2 seconds for the eccentric phase) [8]. The execution of the BP and SR were standardized and pauses were not permitted between the concentric and eccentric phases (Fig. 2). A metronome (Metronome Plus, M&M System Germany, version 2.0) was used to help control the lifting

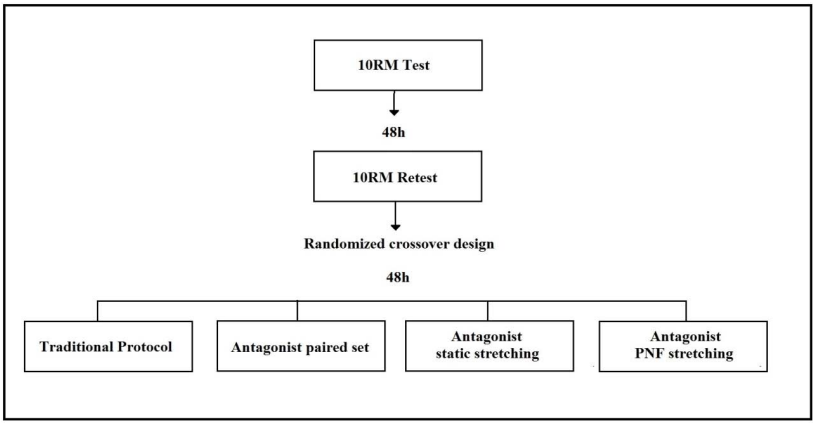

Fig 1. *Summary for experimental protocol trials*

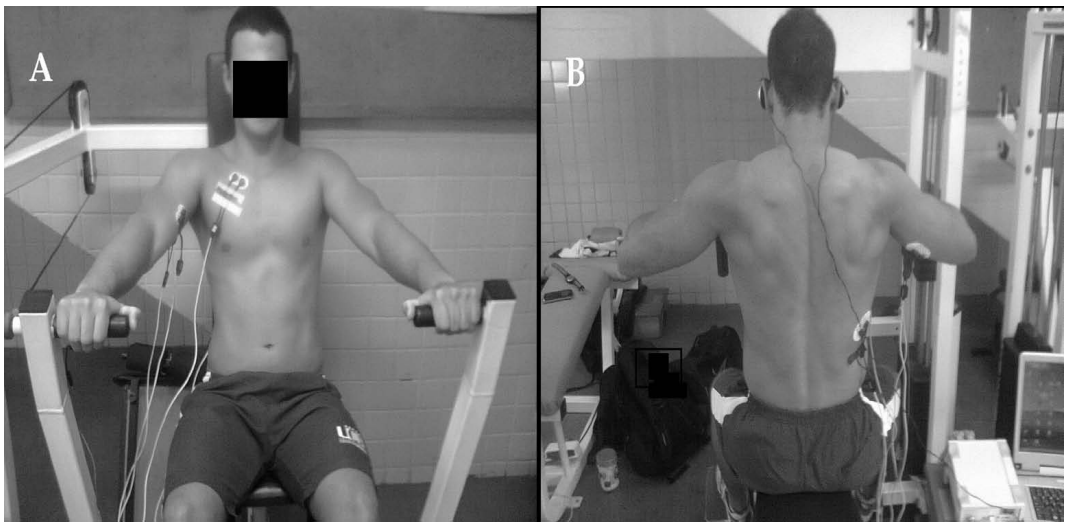

Fig. 2. *Resistance exercises bench press (a) and wide grip seated row (b)*

cadence. However, if subjects slowed their cadence due to fatigue, all completed repetitions were still counted. If a 10RM was not accomplished on the first attempt, the weight was adjusted by 4–10 kg and a minimum 5-minute rest was permitted before the next attempt. Only three trials were allowed per testing session. The test and retest trials were conducted on different days with a minimum of 48 hours between tests.

Stretching Exercises

The static and PNF stretches applied to the PM muscle were consistent with the protocol previously conducted by Franco et al. [20]. Subjects maintained a standing position, preserving the physiological curvature of the spine; the researcher then instituted a passive stretch for the PM via horizontal abduction of the shoulder joints with the elbow joints fully flexed. According to Franco et al. [20], 40 s of static or PNF type stretching induced significant reductions in the force production and activation of the stretched muscles.

Electromyographic acquisition and analysis

The EMG data of LD, BB, PM, and TL muscles were evaluated during the SR exercise. Before the placement of the electrodes, the areas were shaved and cleaned with alcohol until a slight redness was apparent [21]. The PM electrode was placed at the midpoint between the acromion process and the xiphoid process. The LD electrode was placed lateral to the inferior angle of the scapula. The BB electrode was placed on the line between the medial acromion and the cubit fossa. The TL electrode was placed half way between the acromion process and the olecranon process at 2 finger widths below the medial line [22].

The EMG data were captured through passive bipolar surface electrodes (Kendal Medi Trace 200, Tyco Healthcare, Pointe-Claire, Canada) with recording diameter = 1 mm and distance between electrode center = 1 cm. The surface electrodes were placed over the muscles bellies. The electrodes were connected to an analog to digital converter of 16 bits (EMG System of Brazil, Sao Jose dos Campos, SP, Brazil) and acquired with the assistance of proprietary software (EMGlab, EMG System of Brazil, Sao Jose dos Campos, SP, Brazil). The EMG signals were amplified by 1.000 with a common mode rejection ratio of 100dB. The signal was sampled at 1000 Hz and 4th order Butterworth filter was applied in forward and reverse direction. The reference electrode was placed on the clavicle bone. A permanent marker was used to mark the location of the electrodes during the first testing session for consistent electrode placement during subsequent sessions [21]. The impedance between electrode pairs was less than 5 kΩ using a 25-Hz signal through the electrodes [21]. All these procedures were performed by the same investigator.

The criterion used for normalization of the EMG activity was the MVIC. Three MVICs were performed against a fixed resistance in the following positions as proposed by Kendall et al. [23]. The isometric action was maintained for 10 seconds with 20 second rest intervals between the three actions for each muscle. For the MVICs, analyses was conducted within a window of 4 seconds between the second and sixth seconds of contraction. The highest RMS value of the three MVICs was used for normalization [24]. The mean amplitude of the RMS was performed using the custom-written software Matlab 5.02c (MathworksTM, Natick, USA). The averaging window for RMS was 100 ms and all reported values are the mean RMS over a predetermined sampling window from the onset to the end of each contraction. EMG data was collected for the entire (concentric and eccentric phases) SR set for each protocol. EMG data was expressed as percentage relative to the largest RMS value of the EMG signal obtained for the MVIC (100%) [18,19].

Statistical analysis

The 10-RM test–retest reliability was calculated through the intraclass correlation coefficient (ICC = $(MS_b - MS_w)/[MS_b + (k-1)MS_w)]$), where MS_b = mean-square between, MS_w = means-square within, and k = average group size. The normality and homoscedasticity of the data was analyzed via the Shapiro-Wilk test and Bartlett test of Sphericity ($P = 0.167$); subsequently, all variables presented normal distribution and homoscedasticity. A one-way ANOVA with repeated-measures was used to assess differences in repetition performance between experimental protocols and muscle activation during the SR exercise. Significant main effects were further assessed using Bonferroni post hoc test. A probability value of $P < 0.05$ was used to establish the significance of all comparisons. Statistical analysis was performed with the SPSS software version 20.0 (Chicago, IL, USA).

Results

The 10RM loads for BP and SR exercise were 85 ± 10.1 kg and 70.2 ± 12,3 kg, respectively. The ICCs for the 10RM tests were as follows: SR = 0.95 and BP = 0.92. The total repetitions completed for the SR under the TP, AS, PNFA, and APS protocols are presented in Figure 3. Significant increases on repetition performance for SR exercise were noted for APS versus the TP ($P = 0.0001$), PNFA ($P = 0.001$) and AS ($P = 0.004$) conditions. Furthermore, a higher number of SR repetitions were also found for AS versus the TP ($P = 0.001$) and PNFA ($P = 0.002$), respectively. No significant differences were noted between the PNFA and TP.

Significant increase on LD activity was noted for APS versus the TP ($P = 0.0001$) and PNFA ($P = 0.002$) protocols; significantly greater LD activation was also found for AS compared to TP ($P = 0.001$) and PNFA

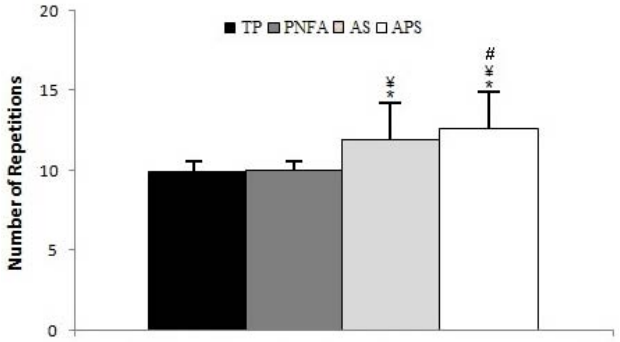

Fig. 3. *Mean ± SD repetitions for the SR exercise under antagonist manipulation protocols; SR: seated row; TP: traditional protocol; PNFA: antagonist neuromuscular proprioceptive facilitation; AS: antagonist stretching; APS: antagonist paired set; *Significant difference versus TP; ¥ Significant difference versus PNFA; # Significant difference versus AS.*

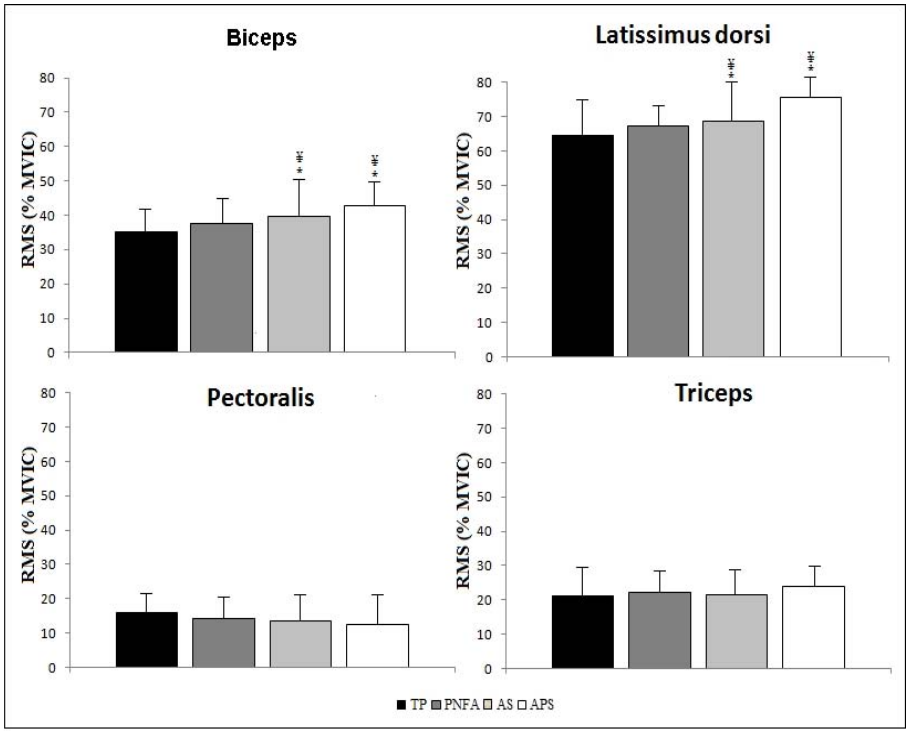

Fig. 4. *Normalized values for the SR exercise under the TP, PNFA, AS, and APS protocols; RMS values for biceps brachii, latissimus dorsi, pectoralis major and triceps lateral head muscles were normalized to the MVIC; TP: traditional protocol; PNFA: antagonist neuromuscular proprioceptive facilitation; AS: antagonist stretching; APS: antagonist paired set; MVIC: maximal voluntary isometric contraction; *Significant difference versus TP; ¥ Significant difference versus PNFA.*

($P = 0.003$). Similarly, BB muscle activation was higher for APS when compared to TP ($P = 0.001$) and PNFA ($P = 0.003$) protocols; significantly greater activation was also observed for AS versus TP ($P = 0.001$) and PNFA ($P = 0.002$). However, no significant differences in PM and TL activation were noted between all protocols (Fig. 4).

Discussion

The current study is the first to our knowledge, to examine multiple antagonist pre-activation protocols through two resistance exercises and the application of different stretching techniques. The key finding from the current study was the significant increase in the number of SR exercise repetitions completed for the APS protocol versus all other protocols; and also the AS protocol versus the TP and PNFA protocols. The increase in repetition performance for the APS and AS protocols was consistent with previous studies that involved manipulation of the antagonist musculature as a pre-activation stimulus to facilitate greater performance in the agonist musculature [8,10,12,15,25]. Perhaps surprisingly, no significant increase in repetition performance was evident for the SR exercise following the contract-relax PNFA protocol versus the TP protocol. The muscle activation data from the current study indicated a significant augmentation in agonist activation (BB and LD) following the APS and AS protocols versus the TP and PNFA protocols, respectively. However no significant differences in antagonist activation (PM and TL) was evident between all protocols.

During the APS protocol, we noted a significant increase in SR repetitions versus all other protocols. These results contrasted with those reported by Robbins et al. [17] in which no differences in repetition performance (with 4 RM loads) were noted between an APS protocol (bench pull and bench press) versus TP (three straight sets of bench pull followed by three straight sets of bench press) adopting 2-minute rest interval between exercises in the APS protocol. In the current study, a significant increase in agonist activation (LD and BB) was observed in the APS protocol versus the TP and PNFA protocols. However, Robbins et al. [17] found no significant differences in the EMG activity of the PM, LD, trapezius and anterior deltoid when comparing the APS protocol versus the TP. However, a lighter load with greater repetitions (10RM) was instituted in the current study; and without a rest interval between the BP and SR exercises. This APS protocol in the current study may have induced greater fatigue in the antagonist muscles (PM and TL), which probably contributed to the significantly greater SR repetitions and agonist activation (LL and BB).

A significant increase in SR repetition performance was also noted for the AS protocol versus the TP and PNFA protocols. Additionally, LD and BB activation were significantly higher during the AS protocol versus the TP and PNFA protocols. Recently, Sandberg et al. [7] reported significantly greater isokinetic knee extensor torque and vertical jump performance following static stretching for the antagonist musculature; the hamstrings were stretched prior to the isokinetic knee extensor test and the hip flexors (single-joint) and dorsi-flexors were stretched prior to the vertical jump test. These authors theorized, that static stretching disrupted the length-tension relationship of the hamstrings, leading to a reduction in braking forces which allowed an improvement on quadriceps torque production [7]. Sharman, Cresswell and Riek [26] stated that during a dynamic muscle action, the agonist is neurally inhibited by its own Golgi tendon organs and by the muscle spindles of its stretched antagonist. In the current study, the AS protocol may have elicited a similar disruption in the length-tension relationship of the PM muscle, and facilitated significantly greater SR repetitions.

Surprisingly, the PNFA protocol did not facilitate significantly greater SR repetitions like the AS and APS protocols. Since the PNFA protocol included 40 seconds, equally divided between contract and relax phases; the 20 second duration of the relax phase may have been insufficient to disrupt the braking effect of the PM muscles as did the AS protocol which involved 40 seconds of progressive static stretching of the PM. It was previously acknowledged that during the stretching protocols (AS and PNFA), no stretching exercises were applied to the TL muscles because the PM is the primary antagonist during the SR exercise. When considering the potential confounding effects of different orders and durations of stretching multiple antagonists (PM and TL) it was decided to test the effects of stretching the PM. According to Sharman et al. [26], PNF stretching may elicit autogenic inhibition and a reduction in excitability of contracting or stretched muscles. Franco et al. [20] reported a reduction in muscle endurance (maximum repetitions performed at 85% of 1-RM) during a BP exercise following a low dose of PNF stretching (one set of 20 seconds), consisting of a single stretch for the PM.

Although, in the current study the PNF stretch volume was not sufficient to significantly increase repetition performance in the agonists during the SR exercise and concomitantly induce a reduction on antagonist activation (PM). In contrast to the current study, Paz et al. [8] found a higher number of repetitions completed in SR exercise (with 10RM loads) following 40 seconds of PNF stretching for the PM muscles when compared to a SR set without pre-stretching exercise. On the other hand, Paz et al. [8] adopted 6 seconds of an isometric action followed by a 4 second relaxation phase repeated four times and totalizing 40 seconds.

This type of PNF protocol might elicit an acute improvement in agonist repetition performance versus the type of PNF protocol adopted in the current study 20 seconds of isometric action followed by 20 seconds of relaxation).

Regardless of the antagonist pre-activation; in the current study, no differences were observed on PM and TL activation during all protocols. Another possibility might be that the surface EMG was not sufficiently sensitive to detect potential decreases in the timing of antagonist activity that may have facilitated greater performance of the agonists for the APS and AS protocols, respectively. The triphasic pattern of muscle activity has been suggested as a mechanism to explain the enhanced acute performance of the agonist musculature following pre-activation of antagonist musculature [9]. This triphasic pattern is characterized by an initial large burst of agonist activity, followed by a shorter "braking" burst of antagonist activity, and finally a second burst of agonist activity during rapid or ballistic actions [12]. According to Baker and Newton [25], a pre-activation resistance exercise for the antagonist musculature could shorten the activation time of the braking burst and also may facilitate a longer burst of agonist activation. Maso et al. [6] found that the progressively RT increases the activation of the primary motor cortex which is associated with a decrease in antagonist muscles activation during motor tasks. The authors indicated that these adaptations could be associated with a specific encoding of antagonist muscles activation through cortical oscillations. In addition, Lévénes et al. [27] observed that excitatory drive to the motor neuron pool of the antagonist muscle is increased during fatigue of the agonist muscle, and the different behavior of the Hoffman-reflex and cervicomedullary motor evoked potentials during the fatiguing action in the antagonist muscle, suggests that the level of coactivation is likely under the control of supraspinal rather than spinal mechanisms.

The findings of the current study should be interpreted with caution because antagonist pre-activation protocols were applied for only a single set of a resistance exercise (SR) for upper body muscles. Whereas, a traditional RT session is composed of multiple sets and exercises for different muscle groups. Therefore, the current study contributes additional information to prompt further study on the mechanism that promoted greater agonist performance via antagonist manipulation. The hypothesis that theorized the improvement on agonist performance due to a reduction in antagonist activation did not appear to be a key mechanism accounting for the improvement in repetitions performance. Other mechanical and metabolic mechanisms such as elastic energy storage, fatigue, and alterations in the acute sensitivity of muscle specific proprioceptors (Golgi tendon organs and muscle spindles) have been proposed by previous researchers [3,6,7,9,16]. Short-term and longitudinal studies are necessary to elucidate whether individuals performing antagonist pre-activation protocols can achieve greater gains in strength versus a traditional training model.

Conclusions

The results of the current study suggested that antagonist pre-activation through either resistance exercise or static stretching may increase acute repetition maximum performance in the agonist musculature. Exercise models performed using a reciprocal antagonist/agonist protocol, as in the current study, may also be less time-consuming and could be useful in clinical practice as well as for sports performance training. The antagonist pre-activation protocols (APS and AS) also elicited significantly higher muscle activity for the agonist muscles (LD and BB) versus the protocol without antagonist manipulation (TP). Nevertheless, there is justification for practitioners and coaches to experiment with antagonist manipulation to improve acute repetition performance and potentially longitudinal training outcomes.

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Authors' contribution
A – Study Design
B – Data Collection
C – Statistical Analysis
D – Data Interpretation
E – Manuscript Preparation
F – Literature Search
G – Funds Collection