Effect of acute antagonist static stretching on upper-body agonist power

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Abstract

There are many studies showing acute static stretching to be detrimental to power generation. However, the majority have focused upon the impact of stretching the agonist musculature. To date, few have examined the potential benefits of acute antagonist static stretching; none have focused on upper-body power. Utilising a repeated-measures design, thirty male participants were randomly assigned to one of two groups whereupon they performed four bench-throw tests; two control (NO-STRETCH) and two experimental (STRETCH), in a counter-balanced manner. Prior to the experimental measures, participants undertook a series of antagonist stretches. Mean *Pmax* (SD) in the NO-STRETCH trials was 862.76 (146W) and 898.50 (144W) respectively. For STRETCH trial 1, *Pmax* = 930.10 (146W) and trial 2, *Pmax* = 953.36 (136W). When compared to the respective NO-STRETCH trials, antagonist static stretching did have a significant effect on *Pmax* for both the initial ($p < .01$, d = 1.33) and the re-stretching procedures ($p < .01$, $d = 1.35$). A significant difference was also found between the STRETCH trials ($p < .01$, $d = .46$). The results have practical implications for those involved in upper-body power activities. Specifically, incorporating upper-body antagonist static stretching into pre-performance routines might offer a simple and effective means of enhancing agonist power.

Key words: Pre-Performance, agonist, stretching protocols, bench-throw, Training, Ergogenic

Introduction

The ability to generate muscular power is important for the successful performance of many athletic and sporting activities.[1, 2, 3] Muscular power can be influenced by chronic adaptations to training which optimises muscular activation patterns; for example, intermuscular (the interaction of muscles that control a movement) and intramuscular (e.g. motor unit recruitment and stretch reflex) adaptations. [3, 4] Acute factors such as pre-performance active warm-up [5] and static stretching can also affect power generation. The current investigation will focus on the latter. Many studies have shown pre-performance static stretching to be detrimental to strength and power-based activities. [6, 7, 8] Several theories for this phenomenon have been offered; for example, it has been hypothesised that acute bouts of static stretching may reduce muscle/tendon stiffness and/or blunt the stretch reflex.[6, 9, 10] This reduces fibre contractibility and neural activity, [6] resulting in diminished power generation capability. Whilst not all research has supported such conclusions, [6, 7] the potential for functional losses, combined with findings that static stretching seldom enhances performance [11] and has minimal impact on injury rates, [12] has led some to advocate its omission from pre-performance routines; particularly when strength and/or power are considered important to optimal performance.[7, 8, 10, 13] However, recommendations for complete abstinence might be premature given that most of the available research has focused on stretching the agonist musculature.

Currently, there is a small body of research that shows acute antagonist static stretching to be beneficial to strength/power-based activities. Miranda, Maia, Paz and Costa [14] examined the impact of such stretching on seated-row performance. The authors also considered whether multiple sets of stretching produced an additive effect. Participants undertook three sets of seated-row exercises with each set preceded by a series of antagonist stretches. Overall, the stretching protocol induced a significant increase in training volume (repetitions × sets × load). In this instance, repeated stretching led to reductions in repetition performance in both the control and experimental conditions; this was attributed to short between-trial rest periods. Regarding power generation, de Souza et al.[15] found three sets of antagonist passive stretching (supine leg flexion) led to significant increases in vertical jump height. Similarly, Sandberg, Wagner, Willardson and Smith [16] also showed three sets of antagonist static stretches (hamstrings, hip flexors and dorsiflexors) to provoke significant increases in jump height. Explaining these findings, during dynamic actions, agonist contraction initiates coactivation of the antagonist musculature.[17] Thought to be a protective mechanism,[18] this intrinsic response is generally considered advantageous as it is believed to stabilise the joint and increase muscle stiffness.[17] However, coactivation also produces resistance as antagonist muscles activate and apply force in the opposite direction to agonist movement. [17, 19, 20] Stretching the antagonist musculature reduces this intrinsic response resulting in enhanced agonist power generation. [15, 16]

To summarise, few have investigated the effects of acute antagonist static stretching on power-based activities. None have examined the effect on upper-body peak power generation. Therefore, more research is required to determine the merits of such stretching procedures. The current investigation will assess the impact of an antagonist static stretching protocol on an upper-body peak power test. We will also examine the consequence of a re-stretch protocol.[14] The outcomes could have practical implications for the multitude of sports/activities that demand such actions.[1, 4, 21, 22]

Methods

Methodological Approach

This investigation utilised a counter-balanced design with repeated-measures. Participants were randomly assigned to one of two groups. Those allocated to group one completed the control measures prior to experimental (control – experimental). Group two completed the experimental trials prior to the control (experimental – control). Regardless of group designation, all participants performed four bench-throw trials; two control and two experimental. Because inter-muscular co-ordination, or lack of, can affect an individual's ability to express power, a sample of strength-trained participants was utilised; thus eliminating neurological learning.[23, 24]

Participants

Participants were recruited from a British Rugby Union Academy. From the one-hundred and fifteen members, thirty volunteered to take part (Mage = 17.4yrs ± 0.9 years; years of Academy training = 2.4 ± 1.2 yrs; Mean bench-press one repetition maximum (1RM) = 97.9 ± 11.7 kg). As part of their programme, all had undertaken regular strength training sessions for a minimum of one year and typically three times per week. All were informed of the benefits and risks of the study before being asked to provide written consent. The authors' Institutional Review Committee approved this study. This investigation adhered to the European Union Agency for Fundamental Human Rights with regards to age of research participatory consent for the UK.

Peak Power (Pmax)

Peak power (*Pmax*) is the maximum power produced at a given moment under a specific set of conditions[2] and is considered a valid performance measure. [25] To assesses *Pmax*, the free-weight bench-throw was utilised.[26, 27] The bench-throw is considered to be a more effective measure of power than the traditional bench press as it allows for greater velocity, power and muscular activation.[19] *Pmax* values were obtained using GymAware PowerTool (version 3.2x) linear position transducer. This device employs a wire tether attached to the barbell to measure vertical displacement. The GymAware also incorporates sensors to monitor horizontal displacement; final *Pmax* values are corrected to account for horizontal motion.[28] As an additional check, movements were also monitored visually for excessive deviation from the vertical plane. As recommended by the manufacturer, the tether was attached to the inner right collar of an Olympic barbell. The GymAware unit was situated directly under the attachment position. This configuration provides the most accurate measurements. [29] The Gymaware PowerTool is considered both valid and reliable.[30, 31] Participants performed three repetitions for each trial and from the data collected an average *Pmax* for each trial was calculated.

Load

For upper-body exercises, 30% of 1RM is shown to be the optimal load for determining peak power.[32, 33] Participants' bench-press 1RM was a known quantity as the Academy recorded this at three-month intervals.

Stretching Protocol

The agonist muscle groups during bench press actions are the pectoralis major, triceps brachii [34] and anterior deltoid.[35] The antagonist muscles were therefore considered as the posterior deltoid, supraspinatus, infraspinatus, teres major and minor. [36] To target the antagonist group, two static stretching techniques were performed; these were taken from Nelson and Kokkonen. [37] Stretch one is described as the 'Shoulder Adductor and Extensor Stretch' (performed actively) and stretch 2, the 'Shoulder Adductor, Protractor, and Elevator Stretch' (performed passively); these stretches purport to target the Latissimus Dorsi, Posterior Deltoid, Rhomboid Major and Minor, Infra- and Supraspinatus and the Teres Major and Minor. All stretches were maintained for a period of 30-seconds [38, 39, 40] at a point of moderate discomfort [15, 16] and on both arms. Upon completion of a stretch, participants immediately performed the manoeuvre on the opposite limb. Therefore, for each limb there was a rest period of (approx.) 30-seconds between stretches.[15, 16] In the experimental condition, two sets of static stretches were performed prior to the first *Pmax* trial; this because some researchers [41, 42] have shown a single, 30s action to have limited influence upon muscle function. An additional set (re-stretch) was undertaken prior to the second experimental *Pmax* trial.

Experimental Procedure

All participants completed the control and experimental trials (Figure 1). In a counter-balanced order, participants performed two control measures on one day (trial 1 – rest – trial 2) and two experimental (stretch – trial 1 – rest/re-stretch – trial 2) on another. Control and experimental testing sessions were one week apart. To minimise the influence of residual fatigue from training and competition, testing days occurred when participants were in a rested state. Prior to testing a 5-minute warm up was undertaken; this consisted of upper-body resistance band exercises, upper-body ballistic activities and one set of bench press (4-8 repetitions @ 60-65% of 1RM). Upon completion of the warm-up, participants were either instructed to rest for four-minutes (control) or perform the initial stretching protocol (experimental). The first peak power trials were completed after this period. For the bench-throw, the head, shoulder blades and buttocks remained in contact with a flat weightlifting bench and the feet were in contact with the ground throughout. Participants were instructed to use their preferred grip width. For consistency, the individual's grip-width was recorded and applied to all their trials. The individual lowered the bar until it lightly touched the mid-sternum at nipple level [43] where it remained for one second before the movement was initiated.[44] Next, participants were instructed to extend the arms explosively, until full extension of the elbows occurred, and to project the barbell straight upwards and perpendicular to the ground. Throughout the movement the wrists and elbows were in alignment. Two spotters were placed to catch the bar at the top of its flight and to return it to the participant. A repetition was to be discounted if (a) there was visual evidence of countermovement (if the participant elevated any part of their torso off the bench), (b) if full elbow extension was not achieved or (c) if there was evidence of excessive horizontal displacement. All participants adhered to instructions and as such, no attempts were disregarded. A period of four minutes was given between trial 1 and trial 2.[14] During the control condition, this consisted of passive rest and for the experimental condition, participants performed the static stretching protocol during the final two minutes.[14]

Figure 1 Here

Figures 2 and 3 Here

Data Analysis

Shapiro-Wilks tests showed all data groups to be normally distributed (p >.05). Repeated Measures ANOVA was employed to assess statistical significance between the four trials. Effect sizes were calculated using Cohen's *d* for repeated measures.[45] Effect size magnitudes were based upon; 0.2 = 'small', 0.5 = 'medium' and 0.8 = 'large'. Mean Difference and 90% Confidence Intervals were also calculated. Values are reported as mean (± *SD*). Two-tailed alpha was set a-priori at 0.05.

Results

For the control condition, *Pmax* was 862.76W (*SD* = 146W) for trial 1, and 898.50W (*SD* = 144W) for trial 2. For the experimental condition, trail 1 *Pmax* = 930.10W (*SD* = 146W) and trial 2 (re-stretch), *Pmax* = 953.36W (*SD* = 136W). ANOVA revealed significant differences between the conditions; F (3) = 39.81, p < .01. Pairwise comparisons with Bonferroni correction revealed significant differences between control trail 1 and experimental trail 1; $p <.01$; $d = 1.33$, *Mdiff* = 67.33, *90% CI* = 51.70W - 82.95W. Significant differences were found between control trial 2 and experimental trial 2; $p <.01$; $d = 1.35$, *Mdiff* = 54.86W, *90% CI* = 41.86W - 67.86W. Results also showed significant differences between experimental trail 1 and experimental trail 2; $p <.01$, $d = .46$, *Mdiff* = 23.26W, *90% CI* = -8W - 37.96W. No significant differences were found between the control trials.

Discussion

A large volume of literature exists concerning the effects of statically stretching agonist musculature prior to power dependent activities; a substantial portion deems the practice to be detrimental to performance. To date, few have examined the potential benefits to be derived from acute antagonist static stretching. This investigation aimed to augment this under-researched area by examining the effect of acute antagonist static stretching on upper-body peak power (*Pmax*). We also considered whether a re-stretch protocol produced an additive effect. When compared to the first control trial, two sets of 30s antagonist stretching led to a significant increase in *Pmax*. Effect size was 'large' and the 90% CI shows that a positive effect is very probable. Experimental trial 2 showed a single bout of re-stretching to provoke a significant and 'large' effect when compared to control trial 2. The 90% CI suggests that a positive effect is again, likely. Contrary to Miranda et al. [14], the re-stretch protocol also caused a significant additive effect when compared to experimental trial 1. However, in this instance ES was 'moderate' and the 90% CI between the stretching trials show that there is a slight chance that a negative effect is possible.

Overall, these results suggest that acute antagonist static stretching protocols can constrain the mechanisms that oppose agonist peak power.

To explain this effect, previous researchers make reference to two primary mechanisms. The first relates to mechanical adaptations. Specifically, static stretching causes a decrease in the viscoelastic properties of the musculotendinous unit causing increased muscle compliance. In addition, elongation of the sarcomeres places the muscles in a less favourable position on the length-tension relationship. Such responses are believed to reduce antagonist muscle stiffness. [8, 14, 15, 16] Another theory is that acute static stretching reduces neural drive, leading to decreased motor neurone excitability and/or stretch reflex sensitivity. [8, 14, 15, 16] When applied to the antagonist muscles, these mechanisms are thought to reduce antagonist co-activation. [14 15, 16] Whilst both explanations are plausible, to date, neither have been confirmed [14, 16] and as such, cannot be advanced with certainty. Regarding the re-stretch effect, this is likely attributable to a dose-response relationship; that is, additional stretching intensifies the hypothesised mechanical and/or neural responses. [11, 46]

This study does have limitations. First, some advocate the use of linear bearing equipment (e.g. Smith Machine) for the assessment of peak power [47, 48] This is primarily based on the notion that such apparatus eliminates horizontal displacement, thus providing more accurate P*max* values [47, 48] However, the GymAware PowerTool accounts for horizontal displacement when calculating Pmax and trials that exhibit excessive horizontal movement are highlighted as invalid. In addition, visual observations were made throughout the trials and we assert that horizontal displacement was not a significant issue. There are other arguments to support the use of free-weight actions. It is suggested that free-weight barbell motions to allow for greater muscle activation and so, power generation.[49, 50, 51] Also, prior experience with the mode of exercise should be considered when selecting power assessment protocols.[48] All the participants in the current study were familiar with the procedures utilised. Given such arguments, we consider the use of free-weight *Pmax* assessment to be valid.

Second, the stretching protocol was six-minutes in duration; this could be deemed impractical by some. Future research could consider assessing stretches in isolation to determine the influence of each individually. If it transpires that a particular stretch accounts for a major proportion of the effect, this could reduce time requirements for those wishing to implement such procedures. Conversely, given the observed dose-response relationship, there is a possibility that additional stretches might elicit a greater effect. If so, this might offer further benefits for those unconstrained by time restrictions. This is an area for further study. Third, we did not consider the levels of the individual's flexibility. de Souza et al.[15] suggest that base levels of flexibility could potentially alter the neuromuscular responses to antagonist stretching. Specifically, it is theorised that highly flexible individuals have greater stretch tolerance and muscle elongation potential. [52] According to de Souza [15], this could influence neuromuscular responses to the stretching procedures. It should be noted that this assertion is not fully supported. [53] Fourth, like others researching this phenomenon, we have merely shown that acute antagonist static stretching enhances the performance on an isolated power test. However, as Stockbugger and Haennel [54] point out, such field tests, although widely utilised, are nevertheless limited with respect to range of movement and the musculature involved. They do not account for the multi-joint nature of most sporting activities, nor the proprioception or kinesthetics necessary for optimal sports performance. Therefore, further exploration into whether such effects transfer into improvements in actual sports performance [2, 4] is required.

In conclusion, this is a relatively new area of research and as such, many questions remain. Nonetheless, the current results, along with those from previous endeavours, have important implications for those undertaking physical actions that require peak power. From a practical perspective, these results suggest that agonist peak power expression is increased through acute antagonist static stretching. It would also appear that a dose-response exists; thus, performance might be improved further if such stretching protocols are applied between actions. We do recognise that more research is needed in this area before the practical implications are qualified. Research could also consider whether long-term application produces adaptations in the physiological mechanisms responsible for power production.[55, 56] Based on these outcomes, abstaining from pre-performance static stretching completely should not be advised. Rather, coaches and athletes should experiment with antagonist stretching to assess whether it can indeed be used to enhance power activities.

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