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Luke Garceau
Marquette University, luke.garceau@marquette.edu

Aaron Gray
Marquette University

McKenzie Fauth
Marquette University

Phillip Hanson
Marquette University

Brittni Hsu
Marquette University

See next page for additional authors

ANTAGONIST CONDITIONING CONTRACTIONS IMPAIR AGONIST FUNCTIONING

Luke R. Garceau[1], Aaron Gray[1], McKenzie L. Fauth[1], Phillip Hanson[1], Brittni Hsu[1], Tejin Yoon[1], Chris Szalkowski[1], Brittney Lutsch[1], and William P. Ebben[1,2]

[1]Department of Physical Therapy, Program in Exercise Science, Marquette University, Milwaukee, WI, USA
[2]Department of Health, Exercise Science & Sport Management, University of Wisconsin-Parkside, Kenosha, WI, USA

This study assessed the effect of antagonist conditioning contractions (ACC) on the subsequent force and electromyography of an agonist. Twelve subjects performed isokinetic elbow flexion on a dynamometer in 4 test conditions including a baseline condition without, and 1, 3 and 6 seconds after, isometric triceps extension. Average peak torque (T), peak torque/body weight (T:BW), average power (P), and rate of torque development (RTD) were assessed. Electromyographic data were obtained from elbow extensors and flexors. A repeated measures ANOVA with post hoc analysis demonstrated that T, T:BW, P, and RTD were higher in the baseline, compared to the post ACC conditions ($P \leq 0.05$), and appears to be due to higher brachioradialis activation in the baseline condition in compared to some post ACC conditions ($P \leq 0.05$).

KEY WORDS: reversal of antagonists, successive induction, Golgi tendon organ, superset

INTRODUCTION: The activation of the antagonist in order to potentiate the agonist muscle group has been referred to as the successive induction and is thought to stimulate the Golgi tendon organ (GTO) (Kroll, 1972). The stimulation of the GTO in an antagonist, may inhibit its activation and stimulate the activation in a subsequently contracted agonist. In addition to successive induction, other similar terms for this process include the reversal of antagonists, which is present in the rapid transitions between antagonist and agonist muscle groups during movements such as walking, running, and rowing (Voss et al., 1985). Skilled athletes appear to be able to reduce antagonist co-activation as an adaptation that allows them to produce greater agonist force (Bazzucchi et al., 2008).
In addition to successive induction and the reversal of antagonists, a small body of literature defines these phenomena as antagonist conditioning contractions (ACC). Research examining the role of ACC on agonist force demonstrates higher rates of force development but not higher peak force, and no evidence of increased muscle activation (Gabriel et al., 2001; Grabiner et al., 1994; Kamimura et al., 2009). Variations exist in the magnitude and duration of the antagonist activation which may affect the ergogenic potential of this stimulus. Furthermore, some evidence demonstrates that stimulating a muscle with a maximal or near maximal activation may potentiate rather than inhibit it (Robbins et al., 2005) and efforts to reduce the activation of the antagonist via a fatiguing stimulus resulted in its potentiation and subsequent impairment of agonist functioning (Maynard and Ebben, 2003). Finally, some evidence indicates that the antagonist inhibition may last only 1 second (Chalmers, 2004). Thus, the challenge seems to be to activate the antagonist enough to stimulate the GTO, while not potentiating it, and to take advantage of the antagonist inhibition before it decays. Therefore, the purpose of this study was to assess the duration of ACC on subsequent performance and activation of the agonist.

METHODS: Twelve men (mean ± SD: age = 21.08 ± 1.80 years; height = 183.54 ± 8.88 cm; body mass = 83.39 ± 9.35 kg; frequency of resistance training = 3.25 ± 0.75 days/week) volunteered to serve as subjects for the study. Subjects signed an informed consent form prior to participating in the study which was approved by the Institutional Review Board.
Subjects performed a general and dynamic warm up prior to the study. Subjects then performed a task specific warm up on the dynamometer (System 4, Biodex Inc., Shirley, NY) consisting of 2 sets of 1 repetition of isometric (ISOM) elbow extension at 75 and 90% of

their self perceived maximum ability and 2 sets of 2 repetitions of isokinetic (ISOK) elbow flexion at 75 and 90% of their self perceived maximum ability. Subjects then rested for 5 minutes and performed the test sets.

Subjects performed 4 test sets in random order with 5 minutes of recovery between the sets. Tests sets included a baseline ISOK elbow flexion set without a preceding ACC set, and 3 other test sets of ISOK elbow flexion occurring 1 second, 3 seconds, and 6 seconds after ISOM ACC elbow extension. For each test set, kinetic and muscle activation data were collected using dynamometry and electromyography, respectively.

For the test sets, subjects were positioned in the dynamometer according to manufacturer specifications. The system was calibrated with the system software. The right elbow was positioned goniometrically at a starting position of 10°of elbow flexion. Isometric ACC and ISOK elbow flexion was performed from this starting point. Isokinetic elbow flexion was performed at 60° per second through a range of motion of 120° of elbow flexion. The order of test sets was randomized with 5 minutes of recovery between tests to reduce order and fatigue effects.

Torque curves for each subject were analyzed using manufacturer's software. Data were sampled for the entire range of motion of the ISOK test sets. Peak torque (T), torque to body weight ratio (T:BW), power (P), and rate of torque development (RTD) were calculated as the average of the two repetitions of each ISOK test sets. Rate of torque development was calculated for the first 300 ms of each test exercise and normalized to a second.

Electromyography was used to quantify muscle activity using a telemetered EMG system (Myomonitor IV, DelSys Inc. Boston, MA, USA). The input impedance was 10^{15} Ohms with a common mode rejection ratio of >80 dB. Electroymyographic data from the biceps brachii (BB) and brachioradialis (BR), were used to assess the agonist elbow flexors, consistent with previous work assessing ACC (Holt et al., 1969). Electromyographic data were also recorded from the triceps brachii-long head (TB-Long) and triceps brachii-lateral head (TB-Lateral) in order to assess muscle activation of the antagonist during all test conditions. Data were recorded at a sampling rate of 1024 Hz using rectangular shaped (19.8 mm wide and 35 mm long) bipolar surface electrodes with 1 x 10 mm 99.9% Ag conductors, and an inter-conductor distance of 10 mm. A common reference electrode was placed on the lateral malleolus of the right leg. Skin preparation included shaving hair if necessary, abrasion, and cleaning the surface with alcohol. Elastic tape was applied to secure electrode placement in order to minimize motion artifact and to provide strain relief for the electrode cables. Surface electrodes were connected to an amplifier and streamed continuously through an analog to digital converter (DelSys Inc. Boston, MA, USA) to an IBM-compatible notebook computer.

All data were filtered with a 10-450 Hz band pass filter, saved, and analyzed with the use of software (EMGworks 3.1, DelSys Inc., Boston, MA, USA). Root mean square signal processing was used and data were calculated using a 125 ms moving window. Root mean squared EMG data were analyzed for the muscle burst for ISOM ACC and the ISOK elbow flexion tests. Burst onset and offset was determined as the points at which the RMS EMG values initially exceeded and eventually fell below 150 percent of baseline EMG values for each muscle burst. Data were averaged for the two trials and normalized to a resting value for each muscle assessed.

Data were analyzed with SPSS 16.0 using a repeated measures ANOVA and Bonferroni adjusted pairwise comparisons to identify the specific differences in T, T:BW, P, and RTD and RMS EMG for each muscle assessed between the test conditions. The criterion for significance was set at $P \leq 0.05$. Effect sizes and power were determined η_p^2 and d, respectively.

RESULTS: Statistical analysis revealed a significant main effect for T ($P = 0.001$, $\eta_p^2 = 0.64$, $d = 1.00$), T:BW ($P \leq 0.05$, $\eta_p^2 = 0.34$, $d = 0.92$), P ($P = 0.003$, $\eta_p^2 = 0.34$, $d = 0.92$) and RTD ($P \leq 0.001$, $\eta_p^2 = 0.58$, $d = 1.00$) demonstrating differences between the test conditions for these variables. Table 1 shows the specific differences for these variables based on post hoc analysis. Statistical analysis of RMS EMG data revealed significant main effects for BB ($P = 0.04$, $\eta_p^2 = 0.19$, $d = 0.54$), BR ($P = 0.04$, $\eta_p^2 = 0.23$, $d = 0.68$), TB-long ($P = 0.33$), and

TB-lateral (P = 0.72). Table 2 shows the specific differences for these variables based on post hoc analysis.

Table 1. Peak torque (T), torque to body weight ratio (T:BW), power (P), and rate of torque development (RTD) for isokinetic elbow flexion in baseline and 3 post ACC conditions.

Condition	T(N)*	T:BW(N)*	P(w)**	RTD(N·sec^{-1})*
Baseline (No ACC)	65.30 ± 12.26	81.06 ± 11.22	46.12 ± 11.72	154.60 ± 22.98
1 sec post ACC	60.06 ± 10.39	73.88 ± 9.19	39.30 ± 8.73	130.49 ± 17.14
3 sec post ACC	60.18 ± 10.19	73.67 ± 8.55	40.37 ± 8.66	124.14 ± 24.34
6 sec post ACC	59.61 ± 10.33	73.39 ± 9.67	38.38 ± 9.29	132.73 ± 21.17

*Baseline condition is significantly different than all ACC conditions ($p \leq 0.01$).
**Baseline condition is significantly different than all ACC conditions ($p \leq 0.05$).

Table 2. Muscle activation (millivolts) expressed as RMS EMG normalized to resting values for biceps brachii (BB), brachioradialis (BR), triceps brachii-long head (TB-long) and triceps brachii-lateral head (TB-Lateral) in the baseline and 3 post ACC conditions.

	Antagonist		Agonist	
Condition	BB*	BR**	TB-Long	TB-Lateral
Baseline (No ACC)	0.803 ± 0.24	0.525 ± 0.11	0.032 ± 0.01	0.050 ± 0.02
1 sec post ACC	0.816 ± 0.26	0.497 ± 0.09	0.031 ± 0.01	0.051 ± 0.02
3 sec post ACC	0.785 ± 0.24	0.517 ± 0.11	0.033 ± 0.02	0.051 ± 0.02
6 sec post ACC	0.837 ± 0.28	0.530 ± 0.12	0.033 ± 0.01	0.052 ± 0.03

*3 second post ACC is significantly different than 6 seconds post ACC conditions ($p \leq 0.05$).
**Baseline condition is significantly different than 1 second post ACC; 1 second post ACC is significantly different than 6 seconds post ACC ($p \leq 0.05$).

DISCUSSION: This study demonstrates that MVIC ACC impaired subsequent agonist performance for all variables assessed, regardless of the duration of time after the ACC. This impairment appears to be due to higher levels of BR activation in the baseline condition compared to some of the conditions, following the ACC. Despite using brief and maximal ACC based on previous recommendations (Grabiner et al., 1994; Holt et al., 1969; Kamimura et al., 2009), no evidence of the inhibition of the antagonist was found. These results raise questions about the effectiveness activating the antagonist in order to augment performance in a subsequently activated agonist.

Results of the present study differ from previous research examining the effect of ACC which demonstrated increased rate of force development (Gabriel et al., 2001; Grabiner et al., 1994; Kamimura et al., 2009), but not force (Grabiner et al., 1994; Kamimura et al., 2009) or work (Grabiner et al., 1994).

Most studies assessing ACC failed to find any increase in EMG of the agonist (Gabriel et al., 2001; Holt et al., 1969; Kamimura et al., 2009) demonstrating that either EMG was unable to detect, or another mechanism was responsible for, the increase rate of force development demonstrated in these studies. In contrast, results of the present study show some differences in muscle activation of the agonist. However, these data demonstrate that the agonist is impaired, even when the agonist is activated only one second after the ACC. The present study also shows that there is no antagonist inhibition as assessed by EMG, regardless of time elapsed after the ACC. This finding is in contrast to the belief that the inhibition may only last one second (Chalmers, 2004), since in the present study, no antagonist inhibition was present at 1, 3 or 6 seconds post ACC. Thus, it is possible that any potential inhibition may last less that 1 second. If so, this limitation reduces the practical benefit of ACC for resistance training though does not mitigate the functional benefit of reversal of antagonists during a variety of functional movement that quickly transition between antagonist and agonist such as walking or running (Voss et al., 1985) or the potential for chronic adaptation in skilled performers (Bazzucchi et al., 2008).

CONCLUSION: This study demonstrated that maximal short term ACC do not enhance, and appear to impair, kinetic performance and activation of prime movers in some conditions. No evidence of inhibition of the antagonist was found. These findings provide evidence that the use of ACC to enhance agonist performance may not be effective. The activation of the antagonist shortly before the activation of an agonist muscle group during resistance training may have not be beneficial and may possibly be detrimental. Thus, agonist/antagonist, push pull, and compound set resistance training strategies should be avoided in cases where maximum force development is desired.

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