Exercise Bouts at Three Different Intensities Fail to Potentiate Concentric Power

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ABSTRACT

Int J Exerc Sci 2(1) : 38-47, 2009. Postactivation potentiation (PAP) has been hypothesized previously to occur during voluntary, concentric actions. We tested the hypothesis that one of at least three different intensities of conditioning exercises would evoke potentiation of power during the concentric, bench press throw (BPT). Twelve men (age = 22.9 ± 2.7 years, bench press 1 repetition maximum (1RM) = 1.20 ± 0.12 kg·kg⁻¹ body weight) completed five isotonic conditioning presses at ~55, 70, and 86% 1RM, in counterbalanced order, and on separate days. Average and peak power of the BPT using a load of 55% 1RM along with surface electromyography (EMG) of the triceps brachii were collected prior to and 4-minutes following each conditioning bout. Both average and peak power and EMG values (mean ± SD), respectively, were evaluated using two-way analyses of variance with repeated measures. Significant main effect decreases (p < 0.05) in average (-18.6 ± 4.9 W) and peak power (-37.4 ± 9.9 W) occurred across the three different intensities evaluated. No main effects or interactions were observed with the EMG data. Contrary to the previously reported hypothesis, we were unable to demonstrate that conditioning exercise, with three different intensities, can evoke potentiation of power using a load equating to that which is optimum for power production.

KEY WORDS: Accelerometer, bench press throw, postactivation potentiation

INTRODUCTION

Postactivation potentiation (PAP) is defined as a transient increase in muscle contractile performance that occurs subsequent to conditioning exercise (21). The concept of PAP dates back to the 1950’s (13) but has become in vogue in the sport science literature within the last decade (11). The theory of PAP is that a conditioning exercise bout saturates the muscle with calcium which enhances phosphorylation of the myosin regulatory light chains (11). Sale (20) hypothesized that for concentric, volitional performance, neither peak force nor peak shortening velocity can be potentiated in response to conditioning exercise. Rather, he suggested that recognition of PAP in humans performing high intensity power actions would occur within the middle of the force-velocity relationship. Specifically, he described that a rightward shift in the middle of the force-velocity relationship would occur due to a transient improvement in rate of force development (RFD) evoked by conditioning
exercise. Such a hypothesis is relevant to concentric power performance given that the central portion of the concentric force-velocity curve corresponds with optimum power production.

Few studies on PAP have observed transient increases in muscular power evoked by conditioning exercise when concentric-dominated actions were used (e.g., squat jumps, bench press throw) (11). One factor that may be contributing to the inability for investigators to support the hypothesis offered by Sale (20) is that less than appropriate loads for the conditioning exercise were utilized. In an effort to reveal an optimum load for conditioning exercise, Brandenburg (4) evaluated the potential of three different loads of bench press repetitions to evoke a transient increase in power for the bench press throw (BPT). Specifically, his participants performed five conditioning repetitions using 100, 75, or 50% of the 5 repetition maximum (5RM), where 100% of 5RM equates to approximately 86% of the 1RM. In each instance, Brandenburg observed no effect on power. However, it has been reported that optimum load for power production for the BPT is ~55%1RM (2) and the load used by Brandenburg was 45%. Given that Sale hypothesized PAP occurs toward the central portion of the force-velocity relationship, it is possible Brandenburg failed to observe a change in power because the 45% 1RM load for the BPT was too low.

Our study therefore examined whether three different intensities of conditioning exercise would evoke a transient increase in power, when optimum load was used for the BPT. Surface electromyography (EMG) was used to explore any changes in muscle recruitment that would be suggestive of a potentiated performance confounded by neuromuscular fatigue.

**METHOD**

**Participants**

Based upon *a priori* power analysis (effect size of 1.2, 1-β of 0.80), we recruited 12 men with no history of upper extremity injuries within the prior 6-months (mean ± SD, age = 22.9 ± 2.7 years, mass = 78.8 ± 9.6 kg, height = 177.3 ± 6.7 cm). For safety consideration and to ensure a minimum training status, every participant had to possess the ability to bench press a load exceeding their body weight. The mean ± SD bench press strength to body mass ratio for this sample was 1.20 ± 0.12 kg·kg⁻¹. All procedures for this experiment were approved by the host university institutional review board and all subject provided written informed consent prior to participation.

**Assessment of bench press 1RM**

On the first visit to our laboratory, participants performed a warmup consisting of 1 set of 10 repetitions at 50% of their self-projected 1RM followed shortly by 1 set of 4 repetitions at 80% of the self-projected 1RM (4). Upon completion of this warmup, 1RM bench press testing was conducted on a Smith machine (Samson Equipment, Inc., Las Cruces, NM) that limited motion to the vertical plane. Participants lay supine on the bench, both feet in contact with the ground, and were positioned with the bar 5 cm superior and parallel to the nipple line. Mechanical restraints were placed at the starting point, 2 cm anterior to the thorax, and spotters were instructed to help prevent the
The bench press load was increased progressively after each successful lift until the actual 1RM was attained. A bench press lift was deemed complete if full elbow extension was achieved. A period of 4-min separated warmup sets and each 1RM attempt.

**Familiarization**
The initial visit to our laboratory was also used to familiarize participants with the BPT, thereby reducing the potential of a learning effect biasing subsequent trials. For each BPT, body positioning, pressing motion, mechanical restraints, and spotters were the same as with the 1RM test; however, participants were instructed to press and throw the bar as fast and high as possible. Participants were encouraged to press beyond the release of the bar to avoid deceleration. The load set for the BPT was ~55% 1RM, an intensity reported previously as optimum of generating concentric power (2). A 30-sec rest period was provided between each BPT (4). Trials were repeated until the participants felt comfortable with the execution of the technique (4).

**Protocol**
The interventions consisted of 5 full ROM, isotonic bench press repetitions on the Smith machine using one of three prescribed loads: 55% 1RM, 70% 1RM, or 86% 1RM, performed on three separate visits to the laboratory. Fifty-five percent was selected as it equaled the load used in the BPT. Eight-six percent was selected as it represented the 5RM, ensuring fatigue to a point, in theory, where a 6th repetition was not possible. Seventy percent was selected as an approximate intensity half the distance between 55 and 86% 1RM. Participants were instructed to perform the 5 isotonic presses as quickly as possible (1). Interventions were counterbalanced between participants to avoid an order-effect and a minimum of 2-days separated each visit (N.B., visits actually occurred within 3 to 4-days of each other). Participants were instructed to refrain from exercises involving use of the pectoralis major and triceps brachii muscles, 2-days prior to each testing session.

Each of the three interventions began with a 5-min bout of low intensity, leg cycling followed by a 4-min recovery period. Three baseline BPTs at 55% 1RM were then performed followed by 2-min recovery before commencing the isotonic exercise intervention. A 4-min recovery period followed the intervention after which post-testing of 3 BPTs commenced (4). A programmable timer (Chronomix Pro Time, cc8152HB, Sunnyvale, CA) was used to help manage the protocol and collection of the power and EMG data.

**Data Collection and Processing**
Power of the bar was measured using a piezoelectric linear accelerometer (Pasco Scientific I-6558., Roseville, CA) interfaced with a computer (Data studio software, Pasco science workshop 750, Roseville, CA). Calibration was performed by simply dropping the accelerometer to measure gravitational acceleration (i.e., -9.8 m·s⁻²). The accelerometer was taped to the bar in the same position for all trials. Data collection began 5-seconds before the BPT and lasted for 10-seconds. Power was calculated using the function: [1] Power (W) = kg * (x + 9.8) * integral (x); where x = acceleration with respect to time and kg is
the mass for each participant’s 55% 1RM. Change in power over time for each of the three trails was averaged and the following two-pass Butterworth filter (23) and cutoff frequency was used to fit the data: 

\[ X'(nT) = a_0X(nT) + a_1X(nT-T) + a_2X(nT-2T) + b_1X'(nT-T) + b_2X'(nT-2T) \]

and 

\[ \text{Cutoff Frequency} = (1.4845 + 0.1523 F_s^{1/2})^2 \]

where \( F_s \) is the sampling frequency (1000 Hz).

Surface EMG (BIOPAC Systems Inc., Santa Barbara, CA) was recorded from the triceps brachii of the dominant arm. Raw signals were collected with surface (bipolar) self-adhesive Ag/AgCl pre-gelled disc electrodes over the motor point of the lateral triceps head (15). Preparation included hair removal and skin abrading followed with cleansing with isopropyl alcohol. Interelectrode impedance was verified at < 5000 \( \Omega \). EMG signals were sampled at 1000 Hz, amplified (x 2500), filtered (high pass, 30 Hz; and low pass, 500 Hz), and full-wave rectified. EMG sampling occurred concurrently with the accelerometer (i.e., 10-seconds of data). The BIOPAC software package was also used to perform a Fast Fourier transformation (linear magnitude, hamming window, pad with zeros) on the respective EMG data to determine their spectral content resolution (signal power vs. frequency). The mean and median power frequency of the spectral density function was calculated according to procedures described by the software’s manufacturer.

**Measurement Reliability**

For each dependent variable, averaged intraclass correlation coefficients (ICC) were performed. Univariate analyses of variance (ANOVA) revealed no significant (\( p > 0.05 \)) differences between trials for any of the dependent variables. A high reliability was observed between trials at baseline for each variable (Table 1). As such, the average of the three trials were calculated on all power and EMG data and utilized as the dependent variables for all inferential statistics.

**Table 1. Internal Consistency Reliability of BPT Power and Triceps Brachii Surface EMG**

| Variable           | Averaged ICCs |
|--------------------|---------------|
| Average Power      | 0.93          |
| Peak Power         | 0.80          |
| Mean EMG           | 0.98          |
| Median Frequency   | 0.98          |

**Statistical Analysis**

Descriptive statistics on all dependent measures are reported as mean ± SD. Normality was assessed with Kolmogorov-Smirnov tests and homogeneity of variance was assessed using Levene’s test (all data were \( p > 0.05 \)). Separate 2 X 3 analyses of variance (ANOVA) with repeated measures were used to test for differences in power (both average and peak) and EMG (both mean and median frequency). Levels for each variable were as follows: time (pre- and post-testing) and interventions (55, 70, and 86% 1RM). Interaction was examined using multiple \( t \) tests with Holm’s sequential Bonferroni approach. Level of significance was set at \( p < 0.05 \).

**RESULTS**

**Evaluation of Power during the BPT in Response to Conditioning Exercise of Three Different Intensities**

Summary statistics on filtered data for power relative to time were calculated. For
Figure 1. Bench press throw power (mean ± SD) using a 55% 1RM pre (closed circles) and post (open squares) conditioning exercise bouts of 55, 70, and 86% 1RM.
each intervention both pre and post conditioning exercise, power steadily increased during the BPT and peaked at approximately 0.60 sec (Figure 1). ANOVA for average power indicated a main effect for the time variable (pre versus post BPT) \((F = 19.55, p < 0.01, \eta^2 = 0.64)\) with summary statistics indicating a decreased average power evoked by the conditioning exercise across the three different intensities (Figure 2). No main effect for average power was observed between the three conditioning exercise interventions (55, 70, and 86% 1RM) \((F = 0.19, p = 0.83, \eta^2 = 0.02)\). There was also no significant interaction \((F = 0.89, p = 0.42, \eta^2 = 0.08)\).

ANOVA for peak power indicated a main effect for the time variable \((F = 26.94, p < 0.01, \eta^2 = 0.71)\) with summary statistics indicating a decreased peak power evoked by the conditioning exercise across the three different intensities (Figure 2). No main effect for peak power was observed between the three conditioning exercise interventions \((F = 0.39, p = 0.68, \eta^2 = 0.03)\). There was also no significant interaction \((F = 0.49, p = 0.62, \eta^2 = 0.04)\).

**EMG Results**

ANOVA for average EMG (Figure 3) revealed no main effects for either the pre-post variable \((F = 1.07, p = 0.32, \eta^2 = 0.09)\) or among the three conditioning exercise interventions \((F = 0.58, p = 0.58, \eta^2 = 0.10)\). There was also no interaction \((F = 0.16, p = 0.86, \eta^2 = 0.03)\). Median frequency EMG for each trial is also reported in Figure 3. ANOVA for median EMG revealed no significant main effect for either the pre-post differences \((F = 2.39, p = 0.11, \eta^2 = 0.21)\) or among the three conditioning exercise interventions \((F = 0.15, p = 0.86, \eta^2 = 0.04)\). There was also no significant interaction \((F = 0.29, p = 0.75, \eta^2 = 0.06)\).
DISCUSSION

On three separate days, we evaluated the extent to which a conditioning bout of heavy isotonic exercise, using loads of 55, 70, and 86% 1RM, respectively, might affect BPT performance (i.e., both average and peak power). The load used for the BPT was set at 55% 1RM due to its association with optimum concentric power for this movement (2). Use of such a load during the BPT should have enhanced our likelihood of observing PAP, in accordance with the hypothesis asserted by Sale (20), given this load is approximately midway to the extremes of the force-velocity relationship. Contrary to this hypothesis, no potentiation of power was observed in response to any of the three interventions (Figures 1 and 2). Moreover, no difference in EMG was observed (Figure 3). Fatigue commonly manifests itself by an increase in EMG amplitude and/or decreased median frequency (12). The absence of change in mean and median frequency EMG in our study suggests that the 4-minute recovery period was sufficient to avoid neuromuscular fatigue from confounding any underlying potentiation. This data perhaps would be more compelling had we evaluated EMG of the pectoralis major. We acknowledge the unavailability pectoralis major EMG data as a limitation. Moreover, we did not measure direct indices of PAP (e.g., twitch potentiation) and cannot conclude an absence of PAP per se. Our data does enable us to reject the hypothesis that conditioning with heavy exercise evokes a recognizable transient improvement in concentric power during voluntary activity.

The magnitude of potentiated power performance subsequent to conditioning exercise is reportedly small, on the order of 2 to 4% improvement (18). With such a small, but potentially beneficial performance improvement, we took several steps to ensure our measures were sensitive and reliable. We used a calibrated accelerometer sampling at a rate of 1000 Hz. Error bars depicted in Figure 1 illustrate the sensitivity of our power data (every 0.25 sec shown). High inter-trial reliability of the data was also observed (Table 1). As such, the lack of potentiated power observed in the present study is unlikely the result of inadequate equipment.

Twitch potentiation following preparatory contractions has been reported to last from 5-min (17) and up to 10-min (3). Using physically-active men as participants, one group (7) observed potentiation of vertical jump performance after 5-minutes recovery from conditioning with heavy exercise. As such, the insignificant potentiation of BPT performance in the present study was unlikely a consequence of using too long of a recovery period.

Another area of concern for our results may be the training status of our sample. Specifically, we used resistance-trained men with bench press 1RM values equivalent to ~120% of body weight. Some investigators (5, 14, 19, 22) have asserted that power-trained athletes are better able to evoke PAP from conditioning with heavy exercise. The results of such research is equivocal and the validity of some findings have been questioned (11). In theory, power-trained athletes either possess and/or express more type II muscle fibers and thus are able to phosphorylate myosin ATP more rapidly (8). Our sample
included some power-trained individuals (i.e., 4 military cadets and 1 competitive Muai Thai fighter); however, we acknowledge that the training status of our sample may have prohibited us from observing a transient increase in power.

A plausible alternative explanation for our inability to observe a transient increase in power may be that factors governing potentiation of the contractile mechanisms in skeletal muscle, as evaluated in animal models, do not translate to a potentiation of voluntary actions, when a stretch component is omitted (i.e., concentric-only power). The lack of PAP during a concentric BPT in the present study is finding consistent with Brandenburg (4) who used a similar protocol. Furthermore, PAP has not been observed in lower body, concentric power tests, including squat jumps (7, 10) and concentric knee extensions (6, 22). These investigations varied in protocol but reported no potentiation collectively with concentric-only actions.

Conversely, potentiation of voluntary actions has been observed in similar movements that include a stretch component. For instance, Baker (1) used a similar accelerometer device to one used in the present investigation but evaluated rebounding as opposed to concentric-only BPT. He reported potentiation subsequent to 6-repetitions using a load of 65% 1RM. Hilfiker et al. (10) examined both counter-movement jumping and squat jumping (i.e., a concentric-dominated action) in response to a short bout of heavy load squats. These investigators observed a transient increase in power evoked by heavy squats only in counter-movement jumping conditions. Fatigue may have biased their results, however, because squat jumps were always performed second in their protocol. A transient potentiation of stretch-shortening actions among these studies was observed and it may be that conditioning exercise augments the myotactic stretch reflex (8) and/or alters mechanical properties, which augments passive tension during the eccentric phase of the action (9, 16). Thus, further research on the ability of conditioning exercise to potentiate power during strength-shortening actions is warranted.

An equally compelling question is why a transient increase in power is not observed in studies that use a SSC action for their criterion power performance. For instance, Robbins and Docherty (19) reported that a 7-second isometric conditioning contraction did not evoke a transient improvement in countermovement jumping power. A plausible explanation for the lack of power enhancement may be that the conditioning exercise was isometric and not isotonic or eccentric. Research on the ability of isotonic or eccentric conditioning exercise to evoke a transient increase in power when the criterion power performance involves a SSC action is warranted.

It has been hypothesized that conditioning exercises can evoke a transient, rightward shift of the concentric, force-velocity relationship such that shortening actions at moderate force and velocity, or optimum power, would experience PAP (20). We investigated this hypothesis using three different isotonic exercise intensities and were unable to observe potentiation of voluntary, concentric BPT performance. We submit that mechanisms mediating
PAP, which have been observed in animal models, may not translate to a transient improvement in voluntary concentric power. Without an active stretch component in the criterion performance (e.g., countermovement actions), one cannot obtain the purported benefits from the stretch-shortening cycle (e.g., myotatic reflex and/or passive force enhancement). Such mediating factors and their ability to translate to observable power enhancement during voluntary actions are worthy of exploration.

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REFERENCES

1. Baker D. Acute effect of alternating heavy and light resistance on power output during upper-body complex power training. J Strength Cond Res 17: 493-497, 2003.

2. Baker D, Nance S, and Moore M. The load that maximizes the average mechanical power output during explosive bench press throws in highly trained athletes. J Strength Cond Res 15: 20-24, 2001.

3. Baudry S, and Duchateau J. Postactivation potentiation in human muscle is not related to the type of maximal conditioning contraction. Muscle Nerve 30: 328-336, 2004.

4. Brandenburg JP. The acute effects of prior dynamic resistance exercise using different loads on subsequent upper-body explosive performance in resistance-trained men. J Strength Cond Res 19: 427-432, 2005.

5. Chiu LZF, Fry AC, Weiss LW, Schilling BK, Brown LE, and Smith SL. Postactivation potentiation response in athletic and recreationally trained individuals. J Strength Cond Res 17: 671-677, 2003.

6. Gossen ER, and Sale DG. Effect of postactivation potentiation on dynamic knee extension performance. Eur J Appl Physiol 83: 524-530, 2000.

7. Gourgoulis V, Aggeloussis N, Kasimatis P, Mavromatis G, and Garas A. Effect of a submaximal half-squats warm-up program on vertical jumping ability. J Strength Cond Res 17: 342-344, 2003.

8. Gullich A, and Schmidtbleicher D. MVC-induced short-term potentiation of explosive force. New Stud Athletics 11: 67-81, 1996.

9. Herzog W, and Leonard TR. Force enhancement following stretching of skeletal muscle: a new mechanism. J of Experimental Biology 205: 1275-1283, 2002.

10. Hilfiker R, Hubner K, Lorenz T, and Matri B. Effects of drop jumps added to the warm-up of elite sport athletes with a high capacity for explosive force development. J Strength Cond Res 21: 550-555, 2007.

11. Hodgson M, Docherty D, and Robbins D. Postactivation potentiation: underlying physiology and implications for motor performance. Sports Med 35: 585-595, 2005.

12. Housh TJ, Housh DJ, and deVries HA. Applied Exercise & Sport Physiology. Holcomb Hathaway, Publishers, 2006, p. pg. 239.

13. Hufschmidt HJ. Wird durch muskelvibration eine eigenreflexreihe erzeugt? Plagers Archiv European Journal of Physiology 267: 508-516, 1958.

14. Jensen RL, and Ebben WP. Kinetic analysis of complex training rest interval effect on vertical jump performance. J Strength Cond Res 17: 345-349, 2003.

15. Kendall F, McCreary EK, Provance P, Rodgers M, and Romani W. Muscles, testing and function: With posture and pain. Lippincott Williams & Wilkins, 2005.

16. Morgan DL, and Proske U. Can all residual force enhancement be explained by sarcomere non-uniformities? J Physiol 578: 613-615, 2007.
17. O'Leary DD, Hope K, and Sale DG. Posttetanic potentiation of human dorsiflexors. J Appl Physiol 83: 2131-2138, 1997.

18. Robbins DW. Postactivation potentiation and its practical applicability: A brief review. J Strength Cond Res 19: 453-458, 2005.

19. Robbins DW, and Docherty D. Effect of loading on enhancement of power performance over three consecutive trials. J Strength Cond Res 19: 898-902, 2005.

20. Sale DG. Postactivation potentiation: role in human performance. Sport Sci Rev 30: 138-143, 2002.

21. Sale DG. Postactivation potentiation: role in performance. J Sports Med 38: 386-387, 2004.

22. Smith CJ, and Fry AC. Effects of a ten-second maximum voluntary contraction on regulatory myosin light-chain phosphorylation and dynamic performance measures. J Strength Cond Res 21: 73-76, 2007.

23. Winter DA. Biomechanics And Motor Control of Human Movement. John Wiley & Sons Inc, 2004.