

Research article

## Muscle activation during low-intensity muscle contractions with varying levels of external limb compression

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### Abstract

The purpose was to investigate muscle activation during low-intensity muscle contractions with various levels of external limb compression to reduce muscle perfusion/outflow. A series of unilateral elbow flexion muscle contractions (30 repetitive contractions followed by 3 sets x 15 contractions) was performed at 20% of 1RM with varying levels of external compression (0 (without compression), 98, 121, and 147 mmHg external compression) around the upper arm. Electromyography (EMG) signals were recorded from surface electrodes placed on the biceps brachii muscle and analyzed for integrated EMG (iEMG). Maximal voluntary isometric contraction (MVC) decreased similarly during the control (0 mmHg) and 98 mmHg external compression bout (~18%); the decline in MVC with 121 and 147 mmHg external compression was significantly greater (~37%). Muscle activation increased progressively throughout the contraction bout with each level of external compression, but iEMG was significantly greater during 147 mmHg external compression. In conclusion, low-intensity muscle contractions performed with external compression of 147 mmHg appears to alter muscle perfusion/outflow leading to increased muscle activation without decrements in work performed during the contraction bout.

**Key words:** Neuromuscular function, EMG, cuff pressure, biceps brachii, ischemia.

### Introduction

Increasing muscle activation is an essential factor to sustain physical activities of daily life (10-30% of maximal work capacity) as evaluated by electromyography and metabolic cost measurements (Astrand and Rodahl, 1970; Sawai et al., 2004). Muscle contractions greater than 20% of maximal voluntary isometric contractions (MVC) result in increased intramuscular pressure and significant vessel compression which impedes arterial inflow to and venous outflow from muscle during exercise and increases the rate of muscle fatigue (Bonde-Petersen et al., 1975; Humphreys and Lind, 1963). On the other hand, volitional restriction of blood flow (external compression of vessels) during low-intensity exercise also leads to significantly greater vessel compression and muscle fatigue compared to contractions with no external compression and the same contractile load (Moritani et al., 1992; Sundberg 1994; Takarada et al., 2000b). MVC and level

of muscle activation are reduced following sustained muscle contractions (Bigland-Ritchie et al., 1986b; Garland et al., 1991) but muscle activation is progressively increased during repetitive low-intensity muscle contractions (Moritani et al., 1992).

Recent studies show that external limb compression during low-intensity resistance exercise training leads to muscle adaptations comparable to those observed during high-intensity resistance exercise training, e.g. muscle hypertrophy, strength gains and increased muscle work capacity (Abe et al., 2005; Burgomaster et al., 2003; Shinohara et al., 1998; Sundberg 1994; Takarada et al., 2000b; 2002; 2004; Yasuda et al., 2005). The mechanism by which external compression potentiates the training effect of low-load resistance training remains obscure but appears to be related, in part, to an increased muscle activation associated with reduced blood flow (Moritani et al., 1992; Sundberg 1994; Takarada et al., 2000a; 2000b). Yet other studies (Burgomaster et al., 2003; Madarame et al., 2008; Reeves et al., 2006) have suggested that altered metabolite levels may be the basis for enhancement observed with external compression. The training effects noted with partial restriction of blood flow appear to be the result of an ideal match between the level of blood flow limitation (external cuff pressure), level of increased muscle activation (increased integrated electromyography (iEMG) signal), and a level of fatigue that does not prevent the completion of the training load (Abe et al., 2005; Takarada et al., 2000a; Yasuda et al., 2008a). Thus it would appear that there may be a relationship between the level of external compression and increased iEMG during muscle contractions. However, the optimal external compression for eliciting an increased neuromuscular response with minimal fatigue is not known. Thus, the purpose of the present study was to ascertain the level of external compression that elicits an increased muscle activation (elevated iEMG) during repetitive muscle contractions.

### Methods

#### Subjects

Ten healthy male students (mean (SE): age 22.3 (1.0) yrs, height 170.5 (2.0) cm, body mass 64.9 (2.5) kg) volunteered for this study. While all subjects were physically active participating in regular aerobic-type exercise (2-3

times per week), none had engaged in resistance exercise training for at least 6 months prior to inclusion in the study. This study was approved by the Ethics Committee for Human Experiments, Tokyo Metropolitan University. All subjects received verbal and written description of the study and presented written informed consent prior to participating in the study.

### Protocol

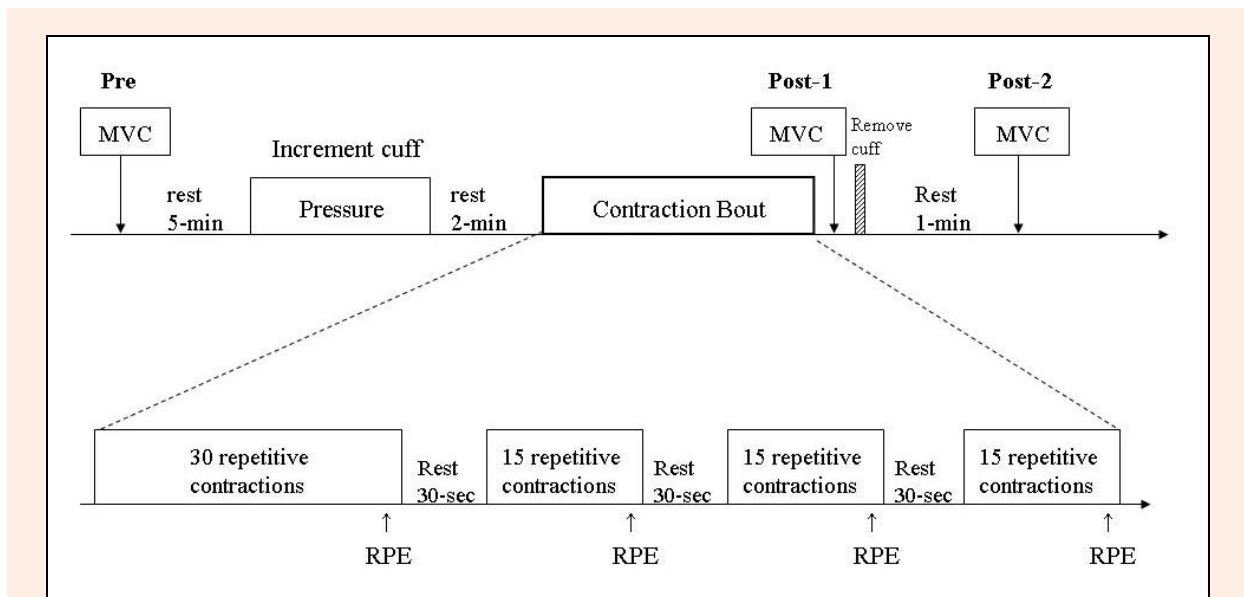
One week prior to experiments subjects completed a study orientation which included measurement of blood pressure, familiarization with the muscle contraction paradigm and external limb compression and performed a one-repetition maximum (1RM) biceps curl test. During the orientation session, the subjects sat in a chair with testing arm placed on a table at heart level and blood pressure was measured three times after 5-min rest. The systolic and diastolic blood pressures were determined by the mean of the three measurements. A generalized experimental time line is shown in Figure 1.

To begin each experiment, a maximal voluntary isometric contraction (MVC) biceps curl was determined with the elbow at 90°. Following a 5-min rest, the external compression cuff was inflated to the desired level of pressure and a 2-min rest period was given. Once the cuff was inflated it remained inflated for the entire contraction bout including rest periods between contractions. Then subjects performed one of four bouts of unilateral elbow flexion (biceps curls) muscle contractions at varying levels of external compression. Immediately following the contraction bout, MVC was determined (Post-1) and the pressure cuff was quickly removed. MVC was again determined 1-min after cuff removal (Post-2). Subjects performed one experiment (one contraction bout and one level of external compression) each day with 3-4 days rest between trials. Order of contraction bout and level of external compression was randomized.

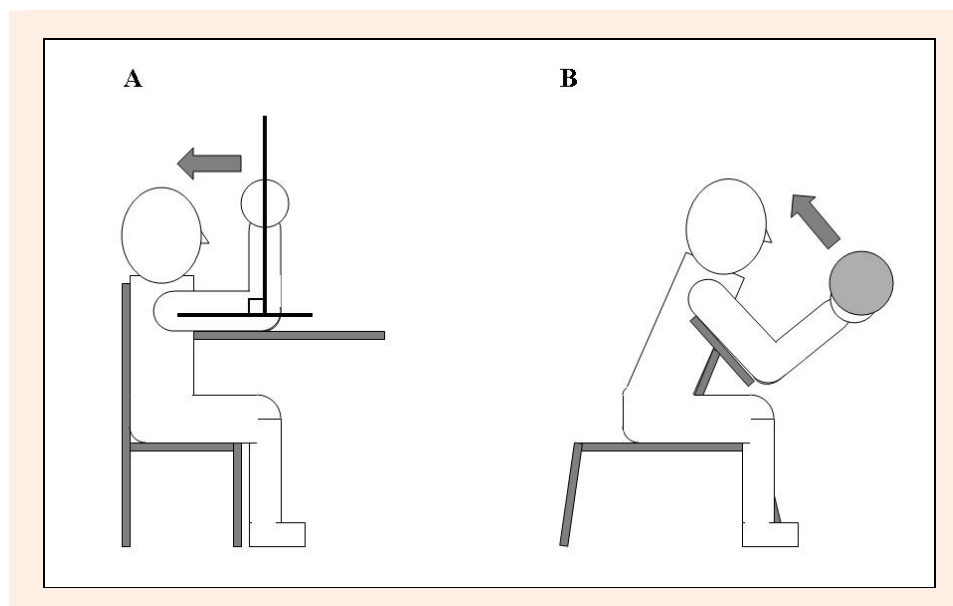
### Measurements

**One-repetition maximum (1RM) biceps curl:** 1RM testing was conducted as described previously (Abe et al., 2000). Subjects performed 5-6 biceps curls with a moderate load as a warm-up and to familiarize subjects with the biceps curl exercise. After warming up, the load was set at ~80% of the predicted 1RM. Following each successful lift, the load was increased by ~5% until the subject failed to lift the load through the entire range of motion. A test was considered valid if the subject used proper form and completed the entire lift in a controlled manner without assistance. On average, five trials were required to complete a 1RM test. Approximately 2-3 min of rest was allotted between each attempt to ensure recovery. Contraction bout was monitored individually by qualified staff and the participants were instructed and coached to perform each protocol with constant motion.

**Maximal voluntary isometric contraction (MVC):** MVC was measured (Biodex System 3 dynamometer; Sakai Medical Instrument, Tokyo, Japan) and used as an indicator of force-generating capacity to evaluate the impact of repeated muscle contractions on contractile function. Fatigue is defined here as a decrease in MVC. The subjects were comfortably seated on a chair and the arm was positioned on firm and stable table at chest level with an elbow joint angle of 90° (0° at full extension, Figure 2A). The upper arm was maintained in the horizontal plane (at 90°) while the hand grasped the Biodex lever in the pronated position. The elbow flexion force was measured with a transducer, while a diagonal strap was secured over the elbow to maintain a stationary position during the MVC. Subjects performed a total of four MVC's; 2 at Pre separated by a 60 sec rest interval, and one each at Post-1 and Post-2. Subjects were instructed to attain the maximal contraction and maintain it for 5 seconds. The recorded value for MVC was taken as the highest and most stable 3-sec of the 5-sec contraction. Pre



**Figure 1.** Experimental timeline. Pre is resting data collected 5 min prior to the start of the experiment, Post-1 is immediately following the contraction bout, and Post-2 is 1-min following removal of the cuff. MVC=maximal voluntary isometric contraction, RPE=ratings of perceived exertion.



**Figure 2.** Illustrations showing the MVC measurement (A) and the contraction bout (B). (A): The upper arm was maintained in the horizontal plane (at  $90^\circ$ ) while the hand grasped the Biodex lever in the pronated position. (B): Elbow flexion contractions were performed with a dumbbell at a contraction intensity of 20% of the 1RM.

MVC was selected as the highest of the two measurements collected.

**Contraction bout:** During the contraction bout, subjects sat on the arm curl bench, with the arm positioned in front of the body supporting the shoulder flexion with the angle of near  $45^\circ$  (Figure 2B). Elbow range of joint motion during muscle contractions was  $0$  to  $150^\circ$  ( $0^\circ$  being full extension). Elbow flexion contractions were performed with a dumbbell at a contraction intensity of 20% of the 1RM. Contraction duration was 2.4 sec with a 1.2 sec concentric: 1.2 sec eccentric contraction cycle controlled by a metronome (50 beats per min). The contraction bout (30 repetitive muscle contractions followed by 3 sets of 15 repetitive contractions with 30s between sets; Figure 1) was determined by reference to previous studies (Abe et al., 2005; Sato et al., 2005; Yasuda et al., 2005). All experiments were carried out in the same arm of a particular subject, but use of dominant or non-dominant arm was randomized between subjects. Prior to the first MVC, each subject performed a standardized warm-up that included 5 light stretches on the upper arm (2 sec duration each) followed by 3 individual, low-intensity ( $<20\%$  MVC) isometric contractions (2 sec duration; 10 sec rest interval).

**External limb compression:** A method for providing external compression around a limb has been previously reported and is termed *KAATSU* (Abe et al., 2005). A specially designed elastic cuff belt (30 mm wide) for the arm (Kaatsu Master, Sato Sports Plaza, Tokyo, Japan) was placed around the most proximal portion of the testing arm. The subjects performed the contraction bout at four levels of external compression calculated as a percentage of their systolic blood pressure; 0% (cuff around arm without compression), 80% ( $98 \pm 3$  mmHg), 100% ( $121 \pm 4$  mmHg), and 120% ( $147 \pm 4$  mmHg).

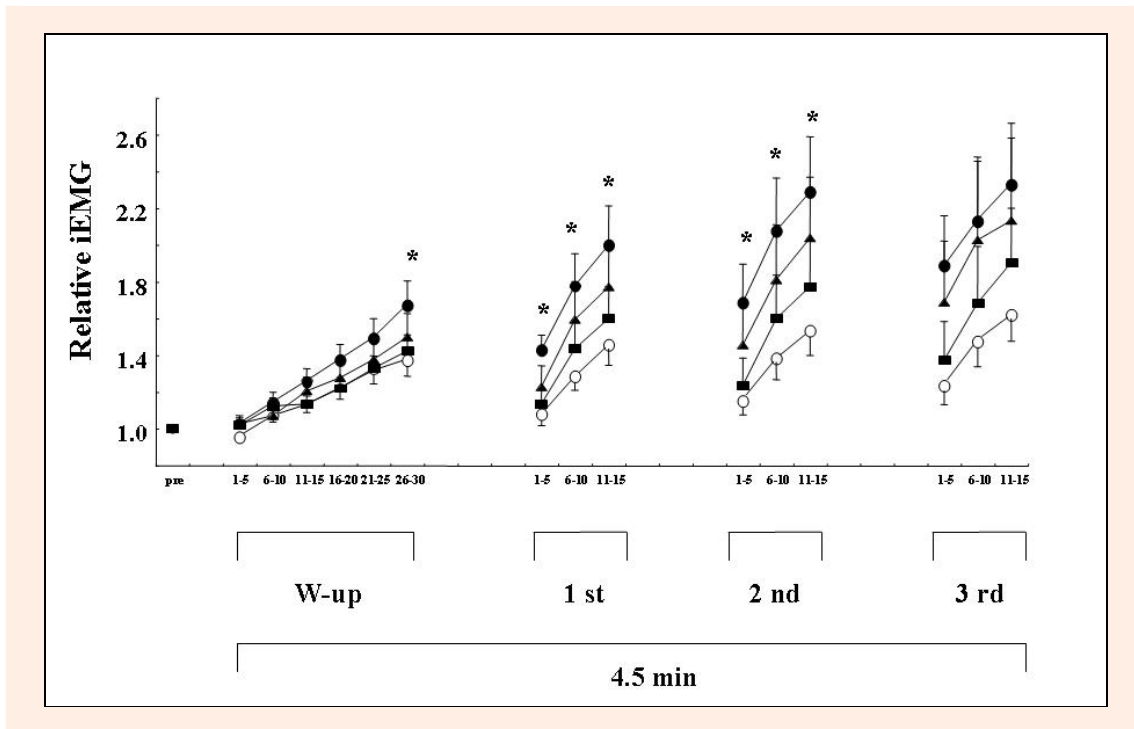
**Electromyography (EMG):** EMG signals were recorded from surface electrodes (sEMG). The skin was shaved, abraded with skin preparation gel (Skinpure,

Nihon Kohden, Japan) and cleaned with alcohol wipes. During all experiments skin impedance was less than 2 k $\Omega$ . The ground electrode was positioned on the lateral epicondyle. Bipolar electrodes (Vitrode F, Ag/AgCl, 1-cm diameter, Nihon Kohden, Tokyo, Japan) were placed over the belly of muscle with a constant inter-electrode distance of 20 mm. The electrodes were connected to a pre-amplifier at a differential amplifier having a bandwidth of 0 Hz to 500 kHz (AB 6216, Nihon Kohden, Tokyo, Japan). EMG signals were collected continuously from the biceps brachii and a sampling rate of 1024 Hz using a 12-bit analog-to-digital converter (Macintosh, Power PC 750, Apple, Japan). To determine integrated EMG (iEMG), signals were fully rectified and integrated (Power Lab Chart 4 software, ADInstruments, Japan). sEMG was collected during each 5-sec MVC and analyzed (iEMG) for the same 3-sec period corresponding to the period used to select MVC (above). During the contraction bout sEMG was recorded continuously and each contraction was analyzed individually (iEMG). The iEMG of 5 successive contractions was averaged to represent a single data point for statistical analysis (Figure 3).

Ratings of perceived exertion (RPE) were measured using the Borg scale (Borg, 1973) after the final repetition of each set of contractions in experiment. Arm circumference was measured (tape measure) at Pre and Post-2 at a position 60% distal between the acromial process and the lateral epicondyle of the humerus. Arm circumference was always measured by the same experienced examiner. Coefficients of variation (SD/mean) for the arm circumferences were approximately 0.09. Heart rate was measured throughout the contraction bout (heart rate monitor CE0537, Polar Electro OY, Finland).

### Statistical analyses

Results are expressed as means  $\pm$  standard error (SE) for all variables. Statistical analyses were performed by multivariate analysis of variance with a compression level  $x$



**Figure 3.** Mean integrated EMG during muscle contractions ( $n = 10$ ). One plot was five muscle activations during low-intensity muscle contractions. Each iEMG value was normalized to those five actions before muscle contractions. Data are means  $\pm$  SE.  $\circ$ -control, unimpaired blood flow;  $\blacksquare$ -cuff pressure at 98 (3) mmHg;  $\blacktriangle$ -cuff pressure at 121 (4) mmHg,  $\bullet$ -cuff pressure at 147 (4) mmHg. \* = different from control,  $p < 0.05$ .

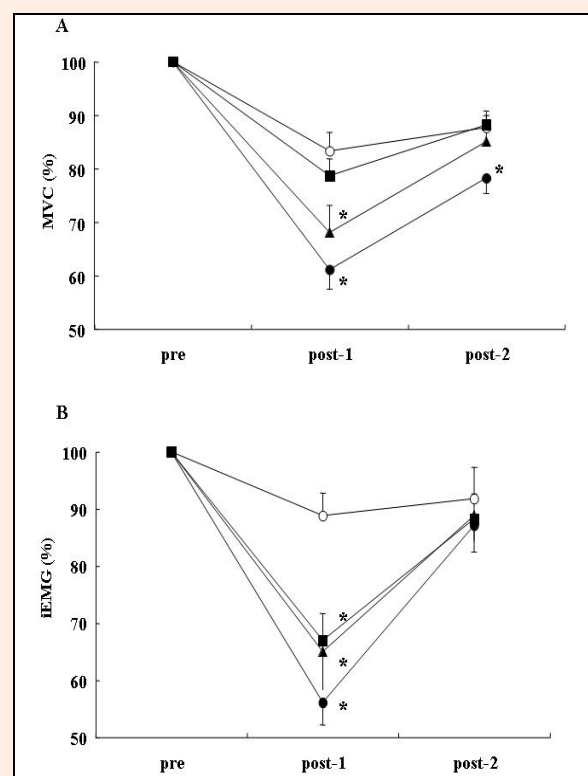
time multivariate. Post hoc tests were performed by simple main effects where appropriate. *A priori* alpha levels were set at 0.05 and corrected for multiple comparisons using the Scheffe Technique.

## Results

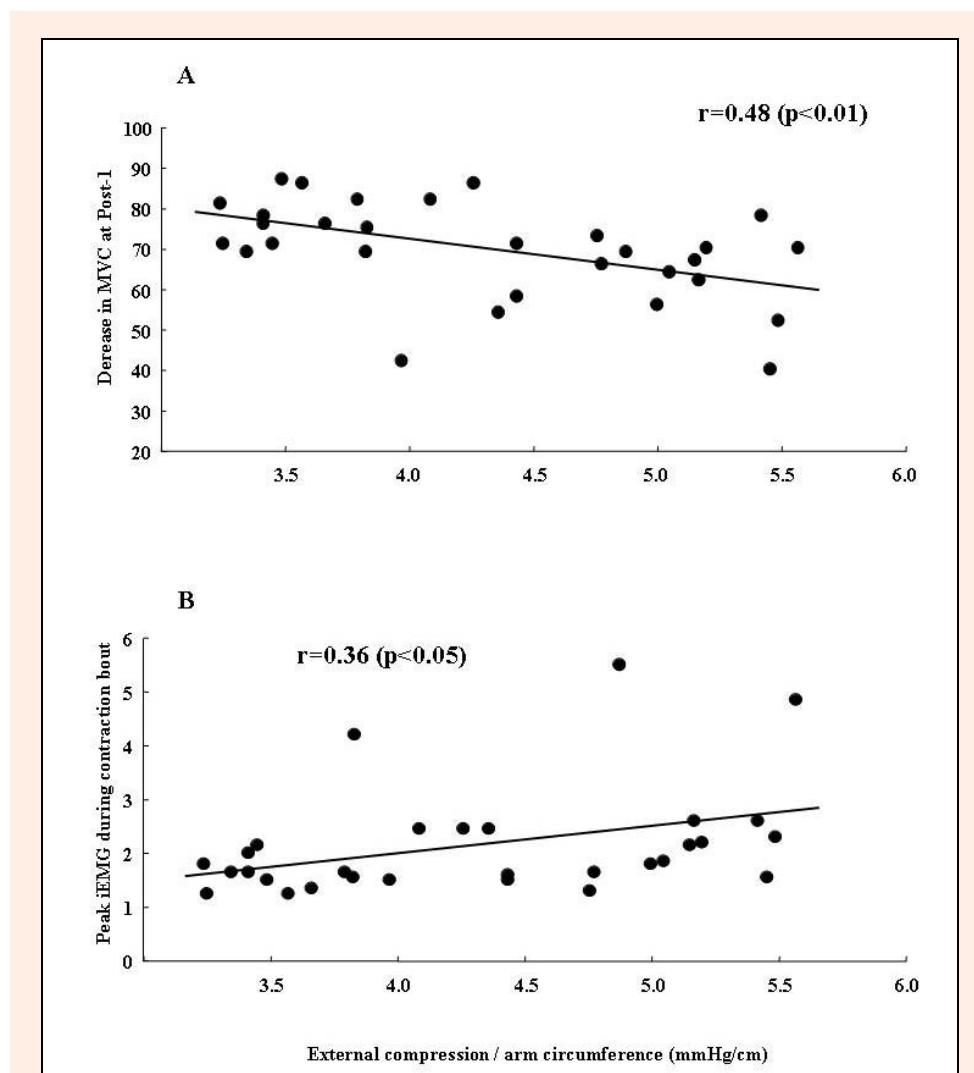
Resting systolic and diastolic blood pressure of subjects were  $121 \pm 3$  mmHg and  $68 \pm 2$  mmHg. None of the subjects had high systolic ( $\geq 140$  mmHg) or diastolic ( $\geq 85$  mmHg) blood pressure.

There were no significant differences among the four trials in pre-bout MVC ( $59 \pm 3$ ,  $61 \pm 3$ ,  $64 \pm 4$ , and  $62 \pm 4$  Nm, respectively). There was a significant decrease in post-1 MVC with 121 and 147 mmHg (32 and 39%, respectively) compared with control (17%). The decline in MVC was associated with significant decreases in iEMG; the changes with 147 mmHg external compression being greater than the others (Figure 4).

During the contraction bout, all subjects maintained and completed all prescribed contractions. iEMG increased progressively during the contraction bout in all four groups and was greater than control with 147 mmHg external compression from the end of 30 repetitive contractions to the end of the 2<sup>nd</sup> set of 15 contractions; otherwise there were no differences among the groups (Figure 3). By one minute after cuff removal MVC had recovered to greater than 85% of initial values in control, 98, and 121 mmHg external compression with no differences in MVC or iEMG among them. However, MVC remained depressed following 147 mmHg external compression (78% of control value; Figure 4) at 1-min post-contraction bout.



**Figure 4.** Maximal voluntary isometric contraction (MVC; Panel A) and integrated EMG (iEMG; Panel B) at MVC in experiment ( $n = 10$ ). Data are means  $\pm$  SE.  $\circ$ -control, unimpaired blood flow;  $\blacksquare$ -cuff pressure at 98 (3) mmHg;  $\blacktriangle$ -cuff pressure at 121 (4) mmHg,  $\bullet$ -cuff pressure at 147 (4) mmHg. \* = different from control,  $p < 0.05$ .



**Figure 5.** Relationships between external compression/arm circumference and decrease in MVC at Post-1 (A) or peak iEMG during contraction bout (B).

There was a significant correlation between external compression and decrease in MVC at post-1 ( $r = 0.54$ ,  $p < 0.01$ ), but not in peak iEMG during contraction bout ( $r = 0.32$ ,  $p > 0.05$ ). When corrected for arm circumference (external compression/arm circumference) external compression was significantly correlated with the decreased in MVC at Post-1 ( $r = 0.48$ ,  $p < 0.01$ ; Figure 5A) and peak iEMG during contraction bout ( $r = 0.36$ ,  $p <$

$0.05$ ; Figure 5B).

During the contraction bouts, heart rate increased progressively in all four groups but there were no significant differences among the four trials (Table 1). RPE during, and arm circumference following, the contraction bout were significantly greater than control compared and the trend was greater with 147 mmHg than with other compressions (Table 1 and 2).

**Table 1.** Heart rate and ratings of perceived exertion during multiple contractions, 30 repetitive plus 3 sets of 15 repetitions of biceps curls performed at 20% of 1-RM ( $n = 10$ ). Values are mean ( $\pm$  SE).

	Pre	C30	S1-R15	S2-R15	S3-R15
<b>Heart rate (BPM)</b>					
Control	75 (3)	89 (3)	89 (3)	90 (3)	91 (3)
98 mmHg	76 (4)	91 (4)	92 (4)	93 (4)	93 (4)
121 mmHg	75 (3)	92 (4)	93 (5)	93 (4)	93 (5)
147 mmHg	74 (2)	93 (3)	93 (3)	98 (3)	99 (4)
<b>Rating of Perceived Exertion</b>					
Control	-	12 (1)	12 (1)	13 (1)	13 (1)
98 mmHg	-	13 (1)	14 (1)	15 (1) *	16 (1) *
121 mmHg	-	14 (1) *	15 (0) *	15 (1) *	16 (1) *
147 mmHg	-	14 (1) *	15 (1) *	16 (1) *	17 (1) *

Pre = pre-contraction bout, C30 = 3<sup>0</sup> contraction, S1-R15 = 15<sup>th</sup> repetition of the 1<sup>st</sup> set, S2-R15 = 15<sup>th</sup> repetition of the 2<sup>nd</sup> set, and S3-R15 = 15<sup>th</sup> repetition of the 3<sup>rd</sup> set. Low, Mid, and High = blood flow restriction; cuff pressure at 98 (3), 121 (4), and 147 (4) mmHg. \* different from control ( $p < 0.05$ ).

**Table 2.** Arm circumference before and after multiple contractions, muscle contractions of biceps curls at 20% of 1-RM (n = 10). Values are mean ( $\pm$  SE).

	Pre	Post-2	differences
<b>Circumference (cm)</b>			
Control	28.5 (.8)	28.9 (.8)	.4
98 mmHg	28.5 (.9)	29.2 (.9)	.7 *
121 mmHg	28.4 (.8)	29.2 (.8)	.8 *
147 mmHg	28.4 (.8)	29.4 (.8)	1.0 *

Pre = pre-contraction bout, Post-1 = immediately following the contraction bout. Low, Mid, and High = blood flow restriction; cuff pressure at 98 (3), 121 (4), and 147 (4) mmHg. \* different from control ( $p < 0.05$ ).

## Discussion

The progressive nature of changes in neuromuscular function and fatigue response between external compression and control indicate a relative progression in “apparent” perceived effort despite the same external load. The external compression employed here resulted in progressive changes in the apparent, relative intensity of the contractions as judged by the increased iEMG and level of decline and recovery of MVC (Figure 4). Stated another way, the external compression appears to alter the energy demand-energy supply relationship during the contractions giving the appearances of a greater metabolic demand at the same external load. Compared to control, each contraction bout accomplished the same total work output under external compression but with an increased level of muscle activation.

The basis for the altered energy demand-energy supply relationship and increased activation observed is likely related to changes in energy supply (availability of oxygen, glucose, free fatty acids, etc) following external compression of blood vessels. Recently, most of low-intensity resistance exercise/training studies utilizing external compression have used an elastic cuff under 160 mmHg external compression in arm (Burgomaster et al., 2003; Reeves et al., 2006; Takarada et al., 2000b; Yasuda et al., 2005). At 160 mmHg external compression there appears to be complete occlusion of venous outflow and partial restriction of arterial inflow in arm (Sato et al., 2005). Previous studies (Iida et al., 2007; Takarada et al., 2000b) reported that moderate external compression (100-250 mmHg for thigh; 50-100 mmHg for upper arm) by an elastic designed cuff does not produce complete occlusion of arterial inflow, but arterial blood flow was decreased with increasing levels of external compression. By comparison, an external compression of 300 mmHg completely occludes venous outflow and arterial inflow as indicated by the absence of a pulse wave in the finger of the compressed arm (Yasuda et al., 2008a). Observable pulse waves have been reported with an external compression of 160 mmHg (Yasuda et al., 2008a). Hence, in the present study, an external compression pressure of less than 150 mmHg would theoretically have little or no impact on arterial inflow and energy supply.

However, the greater neural activation by increases in iEMG (Figure 3) may have taken place to compensate for the deficit in force development secondary to changes in energy supply (Bigland-Ritchie et al., 1986a; Moritani et al., 1986). Furthermore, muscle contractile output during low-intensity contractions (20% MVC) with complete

occlusion of blood flow was maintained by greater neural activation (Moritani et al., 1992). Metabolic changes following reduced blood flow and/or venous occlusion may play a role in increased muscle activation. Following external compression there are significant changes in venous blood oxygen saturation and partial pressure of oxygen and carbon dioxide and accumulation of lactate and hydrogen ions (Takarada et al., 2000a; preliminary data of Yasuda et al., 2008b). These changes could potentially stimulate muscle activation (Bigland-Ritchie et al., 1986a; Moritani et al., 1986) or alter sensory feedback and motor unit activation (Leonard et al., 1994). The lack of difference in iEMG between control and 98 and 121 mmHg external compression pressure leads to the suggestion that blood flow and energy supply and venous occlusion were not significantly impaired enough to induce a change in muscle activation.

The observed effect of 147 mmHg external compression on iEMG and reduction and recovery of MVC is similar to that observed with 160 mmHg external compression using the same elbow contraction protocol (Yasuda et al., 2008a). Comparing these studies reveals similar changes in iEMG during contraction bout and depression and recovery of MVC between 147 mmHg and 160 mmHg external compression. An external compression of 300 mmHg resulted in similar changes in iEMG, but in contrast, there were severe reductions in MVC and total work was significantly reduced as subjects were unable to complete the contraction bout (Yasuda et al., 2008a). These severe decrements appear to be related to the complete occlusion of blood flow. Taken together, it appears that optimal level of external compression for altering blood flow and energy supply and increasing muscle activation without affecting total work performed is between 147 and 160 mmHg (Yasuda et al., 2008a).

Various levels of external compression were selected based on the corresponding systolic pressure of each subject. Since systolic blood pressure is independent of arm circumference, we speculated that a large arm circumference would result in a lesser neuromuscular and fatigue response compared to smaller circumference at the same external compression. There was a significant, but limited relationship ( $r^2 \sim 0.4$ , Figure 5) between external compression corrected for arm circumference and iEMG and level of fatigue during and after the contraction bout (Figure 5), but the magnitude of the effect was not as great as we predicted. This may be related to the fact that arm circumference in our subject population was fairly homogenous. Therefore, it appears that the neuromuscular and fatigue response to a given external compression is affected by arm circumference; an effect that would be observable to a greater extent with a greater variation in arm circumference.

## Conclusion

In consideration of this evidence, it is suggested that increased muscle activation associated with external compression during low-intensity muscle contractions is caused by a mismatch in energy demand-energy supply. The optimal external compression pressure for increased activation (iEMG) without exaggerated fatigue (inability

to complete the prescribed contraction bout) appears to be between 147 and 160 mmHg for upper arm (present data and Yasuda et al., 2008a). Low-intensity muscle contractions with optimal external compression may be the most effective in inducing adaptive responses in muscle when the level of external compression results in increased iEMG without changing the total work output.

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### Key points

- Low-intensity muscle contractions with external compression are maintained by greater neural activation.
- It appears there is optimal external compression pressure for increased muscle activation without exaggerated fatigue.
- External compression per arm circumference was related to the neuromuscular response and fatigue.

### AUTHORS BIOGRAPHY



#### Tomohiro YASUDA

#### Employment

The University of Tokyo, Department of Ischemic Circulatory Physiology, Japan

#### Degree

MSc

#### Research interest

Exercise physiology, sport science.

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**William F. BRECHUE****Employment**

Director of the Center for Physical Development Excellence and an Associate Professor, Department of Physical Education, United States Military Academy, NY.

**Degree**

PhD

**Research interest**

Neuromuscular and cardiovascular physiology and metabolic regulation including physiological factors that dictate athletic performance and response to training and exercise prescription.

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**Taku FUJITA****Employment**

Tokyo Metropolitan University, Department of Exercise and Sport Science, Japan

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MSc

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**Yoshiaki SATO****Employment**

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**Takashi ABE****Employment**

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**Degree**

PhD

**Research interest**

Muscle physiology and sports performance.

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**✉ Tomohiro Yasuda**

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