Low-Level Laser Therapy Facilitates Postcontraction Recovery with Ischemic Preconditioning

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ABSTRACT

CHEN, Y.-C., Y.-T. LIN, C.-L. HU, and I.-S. HWANG. Low-Level Laser Therapy Facilitates Postcontraction Recovery with Ischemic Preconditioning. *Med. Sci. Sports Exerc.*, Vol. 55, No. 7, pp. 1326–1333, 2023. **Purpose:** Despite early development of muscle fatigue, ischemic preconditioning is gaining popularity for strength training combined with low-load resistance exercise. This study investigated the effect of low-level laser (LLL) on postcontraction recovery with ischemic preconditioning. **Methods:** Forty healthy adults (22.9 ± 3.5 yr) were allocated into sham (11 men, 9 women) and LLL (11 men, 9 women) groups. With ischemic preconditioning, they were trained with three bouts of intermittent wrist extension of 40% maximal voluntary contraction (MVC). During the recovery period, the LLL group received LLL (wavelength of 808 nm, 60 J) on the working muscle, whereas the sham group received no sham therapy. MVC, force fluctuations, and discharge variables of motor units (MU) for a trapezoidal contraction were compared between groups at baseline (T0), postcontraction (T1), and after-recovery (T2). **Results:** At T2, the LLL group exhibited a higher normalized MVC (T2/T0; 86.22% ± 12.59%) than that of the sham group (71.70% ± 13.56%; $P = 0.001$). The LLL group had smaller normalized force fluctuations (LLL, 94.76% ± 21.95%; sham, 121.37% ± 29.02%; $P = 0.002$) with greater normalized electromyography amplitude (LLL, 94.33% ± 14.69%; sham, 73.57% ± 14.94%; $P < 0.001$) during trapezoidal contraction. In the LLL group, the smaller force fluctuations were associated with lower coefficients of variation of interspike intervals of MUs (LLL, 0.202 ± 0.053; sham, 0.208 ± 0.048; $P = 0.004$) with higher recruitment thresholds (LLL, 11.61 ± 12.68 %MVC; sham, 10.27 ± 12.73 %MVC; $P = 0.003$). **Conclusions:** LLL expedites postcontraction recovery with ischemic preconditioning, manifesting as superior force generation capacity and force precision control for activation of MU with a higher recruitment threshold and lower discharge variability. **Key Words:** PHOTOTHERAPY, HYPOXIA, FORCE FLUCTUATIONS, MOTOR UNIT, ELECTROMYOGRAPHY

Also known as blood flow restriction (BFR), ischemic preconditioning in combination with low-load resistance exercise (20%–40% of the 1-repetition maximum) has recently become a standard to train muscle (1). For instance, for ischemic preconditioning of the upper limb, an inflated pneumatic cuff is applied on the proximal portion with pressure of 40% to 80% of brachial systolic blood pressure for occlusion of venous return (2). Muscle hypertrophy and strength gains trained with BFR contraction are attributable to enhanced mechanical tension and metabolic stress, which increase the release of growth hormones (3) and activation of protein synthesis signaling (4). In addition, ischemic preconditioning alters the muscle activation pattern, increasing both muscle recruitment (5,6) and the discharge rates of active motor units (MU) (7).

Because of the occlusion of venous return, ischemic preconditioning may impair muscle activation by allowing lactate to accumulate, which does not occur in traditional low-load resistance exercise. Lactate accumulation impairs myoelectrical conduction (8) and accentuates inhibitory control from type III and type IV muscle afferents to alpha motoneurons (9). Because of impairment of muscle excitation–contraction coupling and reduction in corticospinal excitability (10), maximal voluntary contraction (MVC) and twitch torques are smaller with BFR than without BFR (11,12). Hence, strength training combined with ischemic preconditioning gives rise to early development of neuromuscular fatigue (10–14). In a previous study, at a task failure of a 45% MVC contraction, a BFR protocol led to greater force fluctuations and higher median frequency of surface electromyography (EMG) than did a non-BFR protocol (13). Early fatigue development with ischemic

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preconditioning could delay the recovery of contractile function and thus reduce the training volume (or resistance load) to expedite muscular adaptation in real practice (15). In practice, quick recovery from postexercise should add to the benefits of BFR resistance exercise training.

Conventionally, low-level laser (LLL) therapy uses radiation of wavelengths from 655 to 950 nm to enhance wound healing and pain control (16). Taking advantage of the photobioenergetic effect, a novel application of LLL is to excite muscle fibers to improve performance and prevent neuromuscular fatigue (17,18). The delivery of near-infrared photons to muscle tissue can increase oxygen uptake, remedy mitochondrial dysfunction, and improve adenosine triphosphate (ATP) utilization (19,20). LLL intervention can also inhibit the activities of reactive oxygen species/reactive nitrogen species series, which interfere with the binding of calcium ions to myofibrils (21) and reduce blood lactate levels caused by fatiguing contraction (22). In practice, strength exercise and endurance training combined with LLL before and during sets of exercise increase the resistance to exhausting physical activities, corroborating the observations of decreases in lactate concentrations (18,23) or oxidative stress markers (24) and relative increases in peak torque/power (24) or EMG amplitude (25) during muscle contraction. However, the facilitation effects of LLL on muscle contractability are contingent upon multiple factors, including the laser parameters (26), research population, and exercise protocol (27).

Although the combination of low-load resistance exercise and ischemic preconditioning is a promising trend for strength training, this approach could cause neuromuscular fatigue similar to that from strenuous exercise. To our knowledge, no previous works have focused on resolving early fatigue with ischemic preconditioning even during low-load resistance exercise. Considering the energetic effects of LLL, this study aimed to investigate the effects and neurophysiological mechanisms of LLL for postcontraction recovery after low-load resistance exercise with ischemic preconditioning. Within this context, it was hypothesized that LLL therapy could accelerate postcontraction recovery, leading to superior force generation and force scaling as compared with those of sham therapy. In addition, the recruitment pattern and discharge modulation of MU would be adapted to the fine-grained force scaling for superior postexercise recovery with LLL therapy.

METHODS

Participants. Forty healthy young adults (22.9 ± 3.5 yr; 22 men, 18 women) with right-hand dominance signed a consent form before they participated in this study. The experiment was approved by a local institutional human research review board (Institutional Review Board of the National Cheng Kung University Hospital, No. B-ER-109-070) and conformed to the standards set forth by the Declaration of Helsinki with the exception of database registration. Subjects neither performed any vigorous activity nor consumed caffeine for 12 h before the experiment.

Experimental protocol and setup. The participants were randomly allocated into gender-balanced sham (age, 22.5 ± 3.7 yr; 11 men, 9 women; height, 166.4 ± 7.2 cm; weight, 62.2 ± 9.7 kg; body mass index, 22.4 ± 2.6 $kg \cdot m^{-2}$) and LLL (age, 23.5 ± 3.4 yr; 11 men, 9 women; height, 166.0 ± 7.0 cm; weight, 62.3 ± 9.9 kg; body mass index, 22.5 ± 2.4 $kg \cdot m^{-2}$) groups. The amounts of physical activity in the sham and LLL groups were 4.2 ± 2.9 and 4.8 ± 3.4 $h \cdot wk^{-1}$, respectively. On the day of the visit, resting blood pressure was first prescreened. The participants sat on a chair and relaxed for about 3 min before their blood pressure was measured with an electric sphygmomanometer (HEM-7121; OMRON Inc., Kyoto, Japan). Then the wrist extensors of the dominant hands of the participants were trained with a low-load resistance exercise under the hypoxic condition (Fig. 1A). They were seated with the dominant elbow constrained on a testing platform in the position of slight elbow flexion (~30°) with the wrist pronated. A baseline measure was performed to determine force generation capacity and force scaling ability with MVC and trapezoidal isometric contraction of wrist extension, respectively. The MVC of the wrist extensor was first determined from the peak values of three 3-s maximum contraction trials separated by 1-min rest periods.

After 3 min of rest following the MVC test, the participants performed a trapezoidal isometric task to maintain a static target force at 40% MVC. During the trapezoidal isometric task, the participants carefully exerted the isometric force of wrist extension to couple a ramp-up–hold–ramp-down target signal displayed on a computer screen (Fig. 1A). The target signal was a 3-s latent period and a 4-s ramp-up, phase to 40% MVC, 30 s of the static level for the 40% MVC isometric force task, a 4-s ramp-down, phase to rest, and 3 s of latency at the end. Each contraction trial lasted 44 s. The time window of interest was the 9th to 35th seconds of the contraction trial because of the relatively stable force output in this window. The isometric strength at 40% MVC corresponded to the testing time points (T0, T1, T2) so as to analogize resistance exercise at a low intensity of 40% of 1-repetition maximum reported for BFR training. The protocol of trapezoidal contraction made it convenient for the subsequent EMG decomposition using a previous proof of algorithm (28,29). Each participant completed three trials of the trapezoidal contraction interleaved with 3-min rest periods for assessment of force scaling capacity at the baseline and in the postcontraction and after-recovery stages.

Figure 1B presents the timeline of the whole experimental procedure. At the baseline (T0), postcontraction (T1), and after-recovery (T2) points, the MVC and protocol of the trapezoidal isometric contraction, separated by at least 3 min, were assessed. After the baseline measure at T0, a custom-made polyester pneumatic cuff (Limited Edition, H^+ Cuff, Santa Barbara, CA) was applied on the muscle belly of the biceps brachii to occlude venous return in the upper limb for 3 min. Restriction pressure was set at 60% of systolic blood pressure. The ischemic preconditioning, known also as blood flow restriction (BFR), allowed the subsequent force task of the

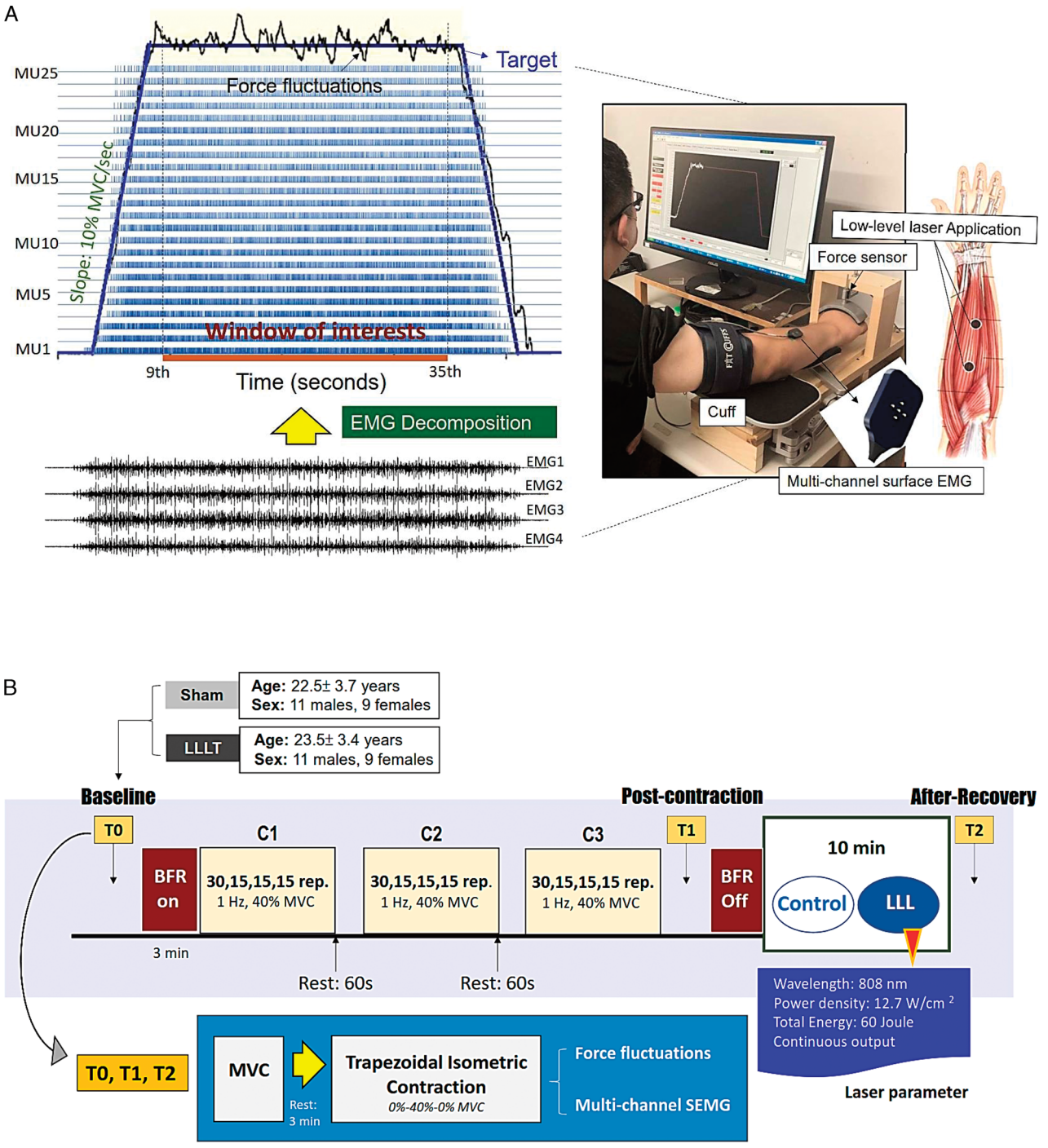

FIGURE 1—A, System setup, example data of the trapezoidal isometric force task, and application of LLL on the wrist extensor for postcontraction recovery. B, Flowchart of experimental process of a force task with ischemic preconditioning for the sham and LLL groups. After BFR, the training force task was three bouts (C1, C2, and C3) of 30–15–15–15 intermittent isometric wrist extension at 40% MVC, interleaved with 60-s rest intervals. At baseline (T0), postcontraction (T1), and after-recovery (T2), the force generation capacity and force scaling ability were assessed with an MVC test and a trapezoidal isometric force task (0%–40%–0% MVC), respectively. When the BFR cuff was removed after T1, the sham group was allowed 10 min for passive recovery, in contrast to the LLL group, who received 60-J LLL (wavelength, 808 nm) on the working muscle.

isometric wrist extension to be completed under the hypoxic condition. Then the participants performed three bouts of isometric wrist extension separated by 1-min rest. Each bout consisted of repeated isometric contraction of wrist extensors of 40% MVC at 1 Hz (30–15–15–15 repetitions, interleaved with 10-s rest intervals). When three contraction bouts were finished, postcontraction measures at T1, including the MVC test and designated trapezoidal contraction, were performed

to assess muscle contractibility. After that, the occlusion cuff was removed, followed by a 10-min recovery stage of relaxation. During the recovery stage, the LLL group received LLL of 60 J for a total of 10 min (the mode continuous) on the muscle belly of the extensor digitorum (22,30). LLL therapy (wavelength, 808 nm; output, 100 mW; Laser 750; Electro-Medical Supplies Ltd., Oxon, UK) was used for the photobiomodulation effect (30). Two sites of laser application were defined by palpation (Fig. 1A). Each site was treated for 5 min, for a total dose of 30 J per site (30). The probe (spot size, 0.785 cm^2) was held stationary in vertical contact with the skin at light pressure. The sham group received sham low-level therapy. At the after-recovery (T2), the MVC tests and trapezoidal contraction protocols of the two groups were assessed.

The force outputs of wrist extension during the MVC and the trapezoidal isometric contraction were measured with a force transducer (Model: MB-100; Interface Inc., Scottsdale, AZ) and sampled at 1 kHz with an analog-to-digital converter with 16-bit resolution (DAQCard-6024E; National Instruments Inc., Austin, TX). Synchronized with the force signals, a multielectrode EMG system that consisted of a sensor array (Bagnoli sEMG system; Delsys Inc., Natick, MA) was used to record the muscle activity of the extensor digitorum. The five pin electrodes (0.5-mm diameter) in the sensor array were placed at the center and at the corners of a 5 × 5-mm square. The EMG sensor array was placed on a line from the lateral epicondyle of the elbow to the second metacarpal, 50–70 mm from the lateral epicondyle. Pairwise differentiation of the five pin electrodes yielded four channels of sEMG, which were amplified (gain, 1000) and band-pass filtered (cutoff frequencies of 20 and 450 Hz) to remove any movement artifact. The conditioned sEMG signals were digitized at a rate of 20 kHz (28,29).

Data analysis and signal processing. The force signal was low-pass filtered (cutoff frequency, 6 Hz) to high-frequency noises, centering on effects of visuomotor processes on force output in the 0- to 4-Hz band (31). The force generation capacity was indexed with MVC force at T0, T1, and T2. Based on the baseline measurement of MVC (MVC_{T0}), we normalized MVCs in the postcontraction and after-recovery stages with MVC_{T0} to obtain $MVC_{T1/T0}$ and $MVC_{T2/T0}$. The force scaling capacity during the trapezoidal isometric contraction was defined as the root mean square (RMS) of force fluctuations within the window of interest. The force fluctuations were force outputs after removal of the linear trend. Based on the RMS of force fluctuations of the baseline (FF-RMS_{T0}), we normalized force fluctuation sizes in the postcontraction and after-recovery with FF-RMS_{T0} to obtain FF-$RMS_{T1/T0}$ and FF-$RMS_{T2/T0}$. The FF-RMS and normalized FF-RMS of force fluctuations of the three experimental trials at T0, T1, and T2 were averaged.

Four EMG signals were collected from the pin electrodes. After conditioning with a band-pass filter (bandwidth, 20–400 Hz), the RMS of the four EMG channels in the window of interest was averaged to represent mean EMG amplitude for trapezoidal isometric contraction at T0, T1, and T2. The EMG amplitude at T1 and T2 was normalized with respect to that of T0 to obtain EMG-$RMS_{T1/T0}$ and EMG-$RMS_{T2/T0}$. Then the EMG signals were processed with a decomposition procedure. With an artificial intelligence–based computation algorithm (28,29), the action potential "templates" of as many MU action potential trains as possible were extracted in EMG Works version 4.3 (Delsys Inc.). Only MU values with accuracy rates of decomposition higher than 90% were reported with the Decomposition–Synthesis–Decomposition–Compare test (29). The EMG decomposition process resulted in discharge events of decomposed MU with values of 0 or 1 (Fig. 1A). In conjunction with the force signal in the ramp-up phase, we determined the recruitment threshold for each MU in terms of %MVC (Rec_{TH}). The mean interspike interval (M-ISI) for each MU was determined by averaging the time intervals of single MU spike trains within the window of interest. The experimentally observed ISI variability of an individual MU was represented with the coefficients of variation (CV) of interspike intervals of MUs in the window of interest. All signal processing was completed in MATLAB 2018 (MathWorks Inc., Natick, MA)

Statistical analysis. On an individual basis, Hotelling's T^2 test and a *post-hoc* independent *t*-test were used to examine group effects on MVC_{T0}, $MVC_{T0/T1}$, and $MVC_{T2/T0}$ for different measurement periods. Likewise, Hotelling's T^2 test and a *post-hoc* independent *t*-test were used to contrast group differences in force fluctuation size (FF-RMS_{T0}, FF-$RMS_{T0/T1}$, FF-$RMS_{T2/T0}$) and mean EMG amplitude (EMG-RMS_{T0}, EMG-$RMS_{T0/T1}$, EMG-$RMS_{T2/T0}$) at T0, T1, and T2. With respect to MU variables (Rec_{TH}, M-ISI, and ISI-CV), a permutation *t*-test was used to examine differences in the variables for all MUs in the sham and LLL groups at T0, T1, and T2. The permutation *t*-test was used because it was difficult to individually identify whether the exact MU values were featured across the experimental trials and different time points. Data were analyzed in SPSS version 22.0 and MATLAB 2018 (MathWorks Inc.). Data reported in the text and tables without specific notations are presented as mean ± SD.

RESULTS

In terms of MVC forces, Table 1 compares force generation capacity between the sham and LLL groups at baseline (T0), postcontraction (T1), and after-recovery (T2). The results of Hotelling's T^2 test showed a significant group difference in the MVC measure (Wilk $\Lambda = 0.707$, $P = 0.005$). *Post-hoc* analysis revealed that the normalized MVC ($MVC_{T2/T0}$) of the LLL group (86.22% ± 12.59%) was significantly greater than that of the sham group (71.70% ± 13.56%) at T2 ($t_{38} = -3.551$, $P = 0.001$). In contrast, baseline MVC (MVC_{T0}) and normalized MVC ($MVC_{T1/T0}$) at T1 did not differ between groups ($P > 0.05$).

In terms of RMS value, Table 2 contrasts the size of force fluctuations between the sham and LLL groups at baseline (T0), postcontraction (T1), and after-recovery (T2). Hotelling's T^2 test revealed a significant group effect on the size of force fluctuations (Wilk $\Lambda = 0.770$, $P = 0.023$). *Post-hoc* analysis revealed that the normalized force fluctuation (FF-$RMS_{T2/T0}$) of

TABLE 1. Means and SD of MVC for the LLL and control groups.

MVC Characteristics	Control	LLL	Hotelling's T^2 Test	*Post Hoc*
Baseline MVC_{T0} (NT)	93.78 ± 30.01	88.61 ± 31.39	**Wilk Λ = 0.707, P = 0.005**	t_{38} = 0.553, P = 0.597
Postcontraction $MVC_{T1/T0}$ (%)	75.42 ± 12.40	77.79 ± 17.85		t_{38} = –0.489, P = 0.628
After-recovery $MVC_{T2/T0}$ (%)	**71.70 ± 13.56**	**86.22 ± 12.59***		**t_{38} = –3.551, P = 0.001**

T0, T1, and T2 are MVC measures at the time of baseline, postcontraction, and after-recovery, respectively). The values in bold format are significant.
*LLL > control, $P < 0.005$.

the LLL group (94.76% ± 21.95%) was significantly smaller than that of the sham group (121.37% ± 29.02%) at T2 ($t_{38} = 3.271$, $P = 0.002$). However, baseline RMS of force fluctuations (FF-RMS_{T0}) and normalized RMS of force fluctuations (FF-$RMS_{T1/T0}$) did not vary between groups ($P > 0.05$). Collectively, the application of LLL during fatigue recovery after BFR resistance training added to the force generation capacity and force precision control.

In terms of RMS, the contrasts of mean amplitude of EMG between the sham and LLL groups at T0, T1, and T2 are summarized in Table 3. Hotelling's T^2 test revealed a significant group effect on the EMG RMS (Wilk $\Lambda = 0.616$, $P < 0.001$). Specifically, the LLL group (94.33% ± 14.69%) exhibited a higher normalized EMG RMS at T2 (EMG-$RMS_{T2/T0}$) than that of the sham group (73.57% ± 14.94%; $t_{38} = -4.430$, $P < 0.001$), rather than EMG-RMS_{T0} and EMG-$RMS_{T1/T0}$ ($P > 0.05$; Table 3).

The decomposition results of the surface EMG showed that the total numbers of MU of all subjects for the three experimental trials at the baseline (T0) in the sham and LLL groups were 1142 and 1155, respectively. The total numbers of MU investigated at postcontraction (T1) of the sham and LLL groups were 1096 and 1173, respectively. The total numbers of MU investigated at after-recovery (T2) for the sham and LLL groups were 1112 and 1030, respectively. A permutation *t*-test was used to examine the group effect on all MU variables at T0, T1, and T2. For the average recruitment threshold of all MUs (Rec_{TH}), no group effect on Rec_{TH} was noted at T0 or T1 ($P > 0.05$; Table 4). The group-dependent difference in Rec_{TH} was significant only at T2. The Rec_{TH} of the LLL group (11.61 ± 12.68 %MVC) was greater than that of the sham group (10.27 ± 12.73 %MVC; $P = 0.003$). For the M-ISI, there were no significant group effects on M-ISI at T_0, T_1, or T_2 (Table 4). Finally, the results of the permutation *t*-test revealed a group-dependent difference in the mean CV (ISI-CV). The LLL group demonstrated a lower ISI-CV than the sham group did at T2 (LLL, 0.202 ± 0.053; sham, 0.208 ± 0.048; $P = 0.004$), rather than at T0 and T1 ($P > 0.05$; Table 4).

DISCUSSION

This study revealed that LLL therapy could more effectively restore muscle contractability after low-load resistance exercise with ischemic preconditioning than that after passive recovery with sham therapy. With the photobiomodulation effect, LLL exposure during the postcontraction recovery led to greater MVC and less force fluctuation in the after-recovery test. The superior force precision control was attributable to the recruitment of MU with higher recruitment thresholds and lower discharge variability.

Rapid return of maximal force for postcontraction recovery with LLL. Previous systematic reviews have revealed that LLL treatment before and during exercise provides ergogenic effects to skeletal muscle for performance improvement or fatigue prevention (17,18,32), although the effect seems to depend on many factors (such as light source, dose, population, and duration). However, the photobiomodulation effect of LLL on postexercise recovery has seldom been explored, especially the effect on the decline in endurance and postexercise discomfort primed with ischemic preconditioning (11–13). Because of low microvascular oxygenation, early fatigue development in local muscle is a potential barrier to muscle training with BFR. Risks associated with excessive metabolic stress prevent increases in training volume (33–35) and/or delay-onset muscle soreness (36) under the hypoxic condition. The decline in MVC force after strength training with ischemic preconditioning is of peripheral and/or central origins, such as the accumulation of lactate (37) and decline in corticospinal excitability (10). This study also showed evident postexercise loss of muscle-force production in the sham and LLL groups after three short bouts of intermittent low-load resistance exercise with ischemic preconditioning (Table 1). The MVC of postcontraction was roughly three-quarters of the baseline MVC.

However, it is worthy of note that exposure to LLL during the recovery on relaxation could enhance the regenerative process, and $MVC_{T2/T0}$ was significantly greater in the LLL group in the after-recovery stage (LLL, 86.22% ± 12.59%; sham, 71.70% ± 13.56%; Table 1). Some photobiomodulation effects of LLL responsible for soft tissue metabolism can explain the restoration of after-recovery MVC. First, LLL can augment cytochrome *c*-oxidase activity in intact skeletal muscle after irradiation (18,32). Mitochondrial activity is upregulated with increases in the mitochondrial respiratory chain, adding to ATP production within muscle tissues and offsetting a detrimental depletion of the muscle energy. Next, LLL

TABLE 2. Contrast of FF-RMS for the LLL and control groups.

Force Fluctuations	Control	LLL	Hotelling T^2 Test	*Post Hoc*
Baseline FF-RMS_{T0} (%MVC)	0.600 ± 0.201	0.657 ± 0.194	**Wilk Λ = 0.770, P = 0.023**	t_{38} = –0.909, P = 0.369
Postcontraction FF-$RMS_{T1/T0}$ (%)	123.07 ± 29.99	120.70 ± 47.82		t_{38} = 0.188, P = 0.852
After-recovery FF-$RMS_{T2/T0}$ (%)	**121.37 ± 29.02**	**94.76 ± 21.95***		**t_{38} = 3.271, P = 0.002**

T0, T1, and T2 are the trapezoidal isometric contraction measures at the time of baseline, postcontraction, and after-recovery, respectively. The values in bold format are significant.
*LLL < control, $P < 0.005$.

TABLE 3. Contrast of RMS of surface EMG (EMG-RMS) for the LLL and control groups.

	Control	LLL	Hotelling T^2 Test	*Post Hoc*
Baseline EMG-RMS_{T0} (μV)	35.80 ± 17.54	37.89 ± 16.32	**Wilk Λ = 0.615, P < 0.001**	t_{38} = –0.390, P = 0.699
Postcontraction EMG-$RMS_{T1/T0}$ (%)	77.15 ± 16.72	81.54 ± 12.88		t_{38} = –0.928, P = 0.359
After-recovery EMG-$RMS_{T2}/_{T0}$ (%)	**73.57 ± 14.94**	**94.33 ± 14.69***		**t_{38} = –4.430, P < 0.001**

T0, T1, and T2 are the trapezoidal isometric contraction measures at the time of baseline, postcontraction, and after-recovery, respectively.
*LLL > control, P < 0.001.

enhances the release of nitric oxide (NO) from muscle tissue and vessel endothelium (38,39). NO produces a potent vasoactive effect, increasing muscle microcirculation at the cellular level. With NO, earlier recovery of the MU recruitment threshold with LLL can also be ascribed to fast interstitial homeostasis, which contributes sufficient potassium to decrease inhibitory activity (such as group III/IV afferent feedback) on the central drive (9). In addition, blood lactate is attenuated in favor of greater force genesis due to improved sensitivity of myofibrils and Ca^{2+} uptake (40,41). Increases in ATP production and NO release jointly contribute to faster recovery of MVC force with LLL after ischemic contraction.

Rapid return of force precision for postcontraction recovery with LLL. Regarding postcontraction recovery, few works have addressed the restoration of force precision control because of underlying modification of a muscle's force output with rate coding or recruitment of the MU, or both. In reference to baseline force fluctuations, both groups exhibited postexercise loss of force precision control, as indicated by the comparably greater FF-$RMS_{T1/T0}$ (Table 2). The increase in force fluctuations did not vary by group, indicating a tendency of neuromuscular fatigue (42) in relation to increases in discharge variability for shifts in low-frequency common drive to MUs (43). Compared with the baseline EMG, both groups also demonstrated a comparably decreasing trend of EMG amplitude (EMG-$RMS_{T1/T0}$) in the postcontraction stage (Table 3), concomitant with a decline in gross nervous system output. However, postcontraction recovery of force precision varied by group. The LLL group regained most of the force precision control, showing significantly smaller force fluctuations (FF-$RMS_{T2/T0}$) than those of the sham group in the after-recovery stage (Table 2).

The increase in force steadiness of the LLL group was physiologically supported by relatively greater EMG-$RMS_{T2/T0}$ (Table 3) for reinstatement of the gross nervous system output and smaller ISI-CV (Table 4) due to the lower discharge variability than that of the sham group. Because the MU model based on computer simulations and experimental findings predicts that discharge rate variability is a major determinant of isometric force variability (44,45), the decline in ISI-CV could be linked to superior force gradation and steadiness for the LLL group in the after-recovery stage (Table 4). In addition, LLL intervention during recovery on relaxation globally increased the recruitment threshold (Rec_{TH}) of active MU (Table 4), indicative of a recovery process of neuromuscular fatigue. It is known that the central nervous system lowers the recruitment threshold of MU in compensation for diminution of the force contributions of individual MU so that muscle force output can be sustained via increasing recruitment of MU (46,47). Hence, the scenario implies that the central nervous system relatively raises the recruitment threshold of MU when the twitch force of MU is potentiated with the photobiomodulation effect for rapid ATP synthesis in the after-recovery stage (32,43). Probably because the regulation of the recruitment threshold and rate coding of MU were independent processes (48), we noted an insignificant increase in discharge rate in the LLL group during the recovery stage relative to that of the sham group (P = 0.094; Table 4). Nevertheless, at least for some participants, an increase in the discharge rate of MUs in the after-recovery stage could help to smooth the summation of several twitch forces, resulting in smaller force fluctuations in the after-recovery stage (Table 2). The neural adaptation with LLL could increase the central drive to MU to counter peripheral fatigue and/or negative inhibitory impacts on the working muscles (30,49) when group III/IV inhibitory feedback is down-regulated (9).

Some methodological issues merit concern. First, the relatively small sample size may have caused sampling bias and unrepresentativeness of our observations in this study. However, this study has developed initial evidence of the beneficial effects of LLL irradiation for rapid return of force control after BFR resistance training. Next, the MU investigated might not have been identical at each time point (T0, T1, and T2). However, its possible impact was unlikely to affect the present

TABLE 4. Means and SD of MU variables for the LLL and control groups.

MU Properties		Control	LLL	Permutation Test
Rec_{TH} (%MVC)	Baseline (T0)	16.69 ± 13.97	16.23 ± 16.48	P = 0.405
	Postcontraction (T1)	9.69 ± 10.91	9.98 ± 10.44	P = 0.440
	After-recovery (T2)	**10.27 ± 12.73**	**11.61 ± 12.68***	***P* = 0.003**
M-ISI (ms)	Baseline (T0)	56.9 ± 18.4	57.4 ± 21.3	P = 0.414
	Postcontraction (T1)	52.6 ± 18.3	53.5 ± 18.8	P = 0.199
	After-recovery (T2)	54.1 ± 15.0	53.0 ± 18.6	P = 0.094
ISI-CV	Baseline (T0)	0.203 ± 0.050	0.202 ± 0.054	P = 0.892
	Postcontraction (T1)	0.206 ± 0.055	0.209 ± 057	P = 0.186
	After-recovery (T2)	**0.208 ± 0.048**	**0.202 ± 0.053****	***P* = 0.004**

T0, T2, and T3 are the trapezoidal isometric contraction measures at the time of baseline, postcontraction, and after-recovery, respectively.
*LLL > control, P < 0.005.
**LLL < control, P < 0.005.

results of MU behaviors, as the EMG electrode was not removed from the recording muscle at any time during the short-period experiment. In addition, the MU behaviors were estimated based on more than 1000 decomposed MU with permutation test. Finally, the effect of LLL on postcontraction with ischemic preconditioning is still not fully understood, as we did not repeatedly measure force and MU behaviors until full recovery.

CONCLUSIONS

Ischemic preconditioning usually brings about early development of muscle fatigue during strength training. This study reveals that LLL therapy has potential to expedite postcontraction recovery for low-resistance exercise with ischemic preconditioning. LLL exposure during recovery on relaxation improves both force generation capacity and force precision control. The improved force precision control is linked principally to the activation of MUs, with a greater recruitment threshold with lower MU discharge variability. LLL therapy has a bioenergetic effect that resuscitates the contractibility of those MUs susceptible to fatigue and the discharge stability against exhaustion. It seems that recovery from postcontraction loss of contractibility may be facilitated with LLL when strength training with ischemic preconditioning is conducted, but further studies will be needed to confirm the effect.

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REFERENCES

1. Lixandrão ME, Ugrinowitsch C, Berton R, et al. Magnitude of muscle strength and mass adaptations between high-load resistance training versus low-load resistance training associated with blood-flow restriction: a systematic review and meta-analysis. *Sports Med*. 2018; 48(2):361–78.
2. Patterson SD, Hughes L, Warmington S, et al. Blood flow restriction exercise: considerations of methodology, application, and safety. *Front Physiol*. 2019;10:533.
3. Ozaki H, Loenneke JP, Abe T. Blood flow-restricted walking in older women: does the acute hormonal response associate with muscle hypertrophy? *Clin Physiol Funct Imaging*. 2017;37(4):379–83.
4. Fry CS, Glynn EL, Drummond MJ, et al. Blood flow restriction exercise stimulates mTORC1 signaling and muscle protein synthesis in older men. *J Appl Physiol (1985)*. 2010;108(5):1199–209.
5. Pearson SJ, Hussain SR. A review on the mechanisms of blood-flow restriction resistance training-induced muscle hypertrophy. *Sports Med*. 2015;45(2):187–200.
6. Pope ZK, Willardson JM, Schoenfeld BJ. Exercise and blood flow restriction. *J Strength Cond Res*. 2013;27(10):2914–26.
7. Fatela P, Mendonca GV, Veloso AP, Avela J, Mil-Homens P. Blood flow restriction alters motor unit behavior during resistance exercise. *Int J Sports Med*. 2019;40(9):555–62.
8. Farina D, Ferguson RA, Macaluso A, De Vito G. Correlation of average muscle fiber conduction velocity measured during cycling exercise with myosin heavy chain composition, lactate threshold, and VO_{2max}. *J Electromyogr Kinesiol*. 2007;17(4):393–400.
9. Teixeira AL, Fernandes IA, Vianna LC. $GABA_A$ receptors modulate sympathetic vasomotor outflow and the pressor response to skeletal muscle metaboreflex activation in humans. *J Physiol*. 2019;597(16): 4139–50.
10. Peyrard A, Willis SJ, Place N, Millet GP, Borrani F, Rupp T. Neuromuscular evaluation of arm-cycling repeated sprints under hypoxia and/or blood flow restriction. *Eur J Appl Physiol*. 2019;119(7): 1533–45.
11. Hammer SM, Alexander AM, Didier KD, Barstow TJ. Influence of blood flow occlusion on muscular recruitment and fatigue during maximal-effort small muscle-mass exercise. *J Physiol*. 2020;598(19): 4293–306.
12. Oranchuk DJ, Koral J, da Mota GR, et al. Effect of blood flow occlusion on neuromuscular fatigue following sustained maximal isometric contraction. *Appl Physiol Nutr Metab*. 2020;45(7):698–706.
13. Copithorne DB, Hali K, Rice CL. The effect of blood flow on tibialis anterior motor unit firing rates during sustained low-intensity isometric contractions. *Appl Physiol Nutr Metab*. 2021;46(1):63–8.
14. de Queiros VS, de França IM, Trybulski R, et al. Myoelectric activity and fatigue in low-load resistance rxercise with different pressure of blood flow restriction: a systematic review and meta-analysis. *Front Physiol*. 2021;12:786752.
15. Badier M, Guillot C, Lagier-Tessonnier F, Jammes Y. EMG changes in respiratory and skeletal muscles during isometric contraction under normoxic, hypoxemic, or ischemic conditions. *Muscle Nerve*. 1994; 17(5):500–8.
16. Hashmi JT, Huang YY, Osmani BZ, Sharma SK, Naeser MA, Hamblin MR. Role of low-level laser therapy in neurorehabilitation. *PM R*. 2010;2(12 Suppl 2):S292–305.
17. Fisher SR, Rigby JH, Mettler JA, McCurdy KW. The effectiveness of photobiomodulation therapy versus cryotherapy for skeletal muscle recovery: a critically appraised topic. *J Sport Rehabil*. 2019;28(5): 526–31.
18. Nampo FK, Cavalheri V, Dos Santos Soares F, de Paula Ramos S, Camargo EA. Low-level phototherapy to improve exercise capacity and muscle performance: a systematic review and meta-analysis. *Lasers Med Sci*. 2016;31(9):1957–70.
19. Hayworth CR, Rojas JC, Padilla E, Holmes GM, Sheridan EC, Gonzalez-Lima F. In vivo low-level light therapy increases cytochrome oxidase in skeletal muscle. *Photochem Photobiol*. 2010; 86(3):673–80.
20. Leal Junior EC, Lopes-Martins RA, Dalan F, et al. Effect of 655-nm low-level, laser therapy on exercise-induced skeletal muscle fatigue in humans. *Photomed Laser Surg*. 2008;26(5):419–24.
21. Moopanar TR, Allen DG. Reactive oxygen species reduce myofibrillar Ca^{2+} sensitivity in fatiguing mouse skeletal muscle at 37°C. *J Physiol*. 2005;564(Pt 1):189–99.
22. Vanin AA, Verhagen E, Barboza SD, Costa LOP, Leal-Junior ECP. Photobiomodulation therapy for the improvement of muscular performance and reduction of muscular fatigue associated with exercise in healthy people: a systematic review and meta-analysis. *Lasers Med Sci*. 2018;33(1):181–214.
23. Vassão PG, Toma RL, Antunes HK, Tucci HT, Renno AC. Effects of photobiomodulation on the fatigue level in elderly women: an isokinetic dynamometry evaluation. *Lasers Med Sci*. 2016;31(2): 275–82.
24. Tomazoni SS, Machado CDSM, De Marchi T, et al. Infrared low-level laser therapy (photobiomodulation therapy) before intense progressive running test of high-level soccer players: effects on functional, muscle damage, inflammatory, and oxidative stress markers—a randomized controlled trial. *Oxid Med Cell Longev*. 2019;2019: 6239058–12.

25. Stamborowski SF, de Oliveira Spinelli BM, Lima FPS, et al. The influence of photobiomodulation on the temperature of the brachial biceps during muscle fatigue protocol. *Lasers Med Sci*. 2021;36(8):1741–9.
26. Leal-Junior EC, Vanin AA, Miranda EF, de Carvalho Pde T, Dal Corso S, Bjordal JM. Effect of phototherapy (low-level laser therapy and light-emitting diode therapy) on exercise performance and markers of exercise recovery: a systematic review with meta-analysis. *Lasers Med Sci*. 2015;30(2):925–39.
27. Higashi RH, Toma RL, Tucci HT, et al. Effects of low-level laser therapy on biceps braquialis muscle fatigue in young women. *Photomed Laser Surg*. 2013;31(12):586–94.
28. De Luca CJ, Adam A, Wotiz R, Gilmore LD, Nawab SH. Decomposition of surface EMG signals. *J Neurophysiol*. 2006;96(3):1646–57.
29. Nawab SH, Chang SS, De Luca CJ. High-yield decomposition of surface EMG signals. *Clin Neurophysiol*. 2010;121(10):1602–15.
30. Chen YC, Su YH, Lin YT, Huang CC, Hwang IS. Acute physiological responses to combined blood flow restriction and low-level laser. *Eur J Appl Physiol*. 2020;120(6):1437–47.
31. Slifkin AB, Vaillancourt DE, Newell KM. Intermittency in the control of continuous force production. *J Neurophysiol*. 2000;84(4):1708–18.
32. Alves AN, Fernandes KP, Deana AM, Bussadori SK, Mesquita-Ferrari RA. Effects of low-level laser therapy on skeletal muscle repair: a systematic review. *Am J Phys Med Rehabil*. 2014;93(12):1073–85.
33. Das A, Paton B. Is there a minimum effective dose for vascular occlusion during blood flow restriction training? *Front Physiol*. 2022;13:838115.
34. Kolind MI, Gam S, Phillip JG, et al. Effects of low load exercise with and without blood-flow restriction on microvascular oxygenation, muscle excitability and perceived pain. *Eur J Sport Sci*. 2023;23(4):542–51.
35. Winchester LJ, Morris CE, Badinger J, Wiczynski TL, VanWye WR. Blood flow restriction at high resistance loads increases the rate of muscular fatigue, but does not increase plasma markers of myotrauma or inflammation. *J Strength Cond Res*. 2020;34(9):2419–26.
36. Brandner CR, Warmington SA. Delayed onset muscle soreness and perceived exertion after blood flow restriction exercise. *J Strength Cond Res*. 2017;31(11):3101–8.
37. Cayot TE, Lauver JD, Silette CR, Scheuermann BW. Effects of blood flow restriction duration on muscle activation and microvascular oxygenation during low-volume isometric exercise. *Clin Physiol Funct Imaging*. 2016;36(4):298–305.
38. Lohr NL, Ninomiya JT, Warltier DC, Weihrauch D. Far red/near infrared light treatment promotes femoral artery collateralization in the ischemic hindlimb. *J Mol Cell Cardiol*. 2013;62:36–42.
39. Moriyama Y, Nguyen J, Akens M, Moriyama EH, Lilge L. In vivo effects of low level laser therapy on inducible nitric oxide synthase. *Lasers Surg Med*. 2009;41(3):227–31.
40. Ferraresi C, Hamblin MR, Parizotto NA. Low-level laser (light) therapy (LLLT) on muscle tissue: performance, fatigue and repair benefited by the power of light. *Photonics Lasers Med*. 2012;1(4):267–86.
41. Ørtenblad N, Nielsen J, Saltin B, Holmberg HC. Role of glycogen availability in sarcoplasmic reticulum Ca^{2+} kinetics in human skeletal muscle. *J Physiol*. 2011;589(Pt 3):711–25.
42. Huang CT, Hwang IS, Huang CC, Young MS. Exertion dependent alternations in force fluctuation and limb acceleration during sustained fatiguing contraction. *Eur J Appl Physiol*. 2006;97(3):362–71.
43. Hwang IS, Lin YT, Huang CC, Chen YC. Fatigue-related modulation of low-frequency common drive to motor units. *Eur J Appl Physiol*. 2020;120(6):1305–17.
44. Enoka RM, Christou EA, Hunter SK, et al. Mechanisms that contribute to differences in motor performance between young and old adults. *J Electromyogr Kinesiol*. 2003;13(1):1–12.
45. Moritz CT, Barry BK, Pascoe MA, Enoka RM. Discharge rate variability influences the variation in force fluctuations across the working range of a hand muscle. *J Neurophysiol*. 2005;93(5):2449–59.
46. Carpentier A, Duchateau J, Hainaut K. Motor unit behaviour and contractile changes during fatigue in the human first dorsal interosseus. *J Physiol*. 2001;534(Pt 3):903–12.
47. Sale DG. Influence of exercise and training on motor unit activation. *Exerc Sport Sci Rev*. 1987;15:95–151.
48. Jensen BR, Pilegaard M, Sjøgaard G. Motor unit recruitment and rate coding in response to fatiguing shoulder abductions and subsequent recovery. *Eur J Appl Physiol*. 2000;83(2–3):190–9.
49. Miller JD, Lippman JD, Trevino MA, Herda TJ. Neural drive is greater for a high-intensity contraction than for moderate-intensity contractions performed to fatigue. *J Strength Cond Res*. 2020;34(11):3013–21.