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Local Vibration Therapy, Oxygen Resaturation Rate, and Muscle Strength After Exercise-Induced Muscle Damage

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Context: Exercise-induced muscle damage (EIMD) is associated with transient reductions in strength and athletic performance. Muscle microvascular damage and disruption of blood flow are believed to be among the causes. Previous researchers reported modulations in muscle blood flow, oxygenation, and strength with vibration therapy (VT).

Objective: To observe whether local VT alleviated the impairments and hemodynamic changes associated with EIMD.

Design: Controlled laboratory study.

Setting: Laboratory and public gymnasium.

Patients or Other Participants: A total of 10 healthy participants (6 men and 4 women; age = 38 ± 15 years; height = 1.72 ± 0.48 m; mass = 72.0 ± 10.4 kg) were randomized into experimental (VT) and control groups.

Interventions: Both groups performed 10 sets of 10 eccentric wrist flexions at 70% of their 1-repetition maximum to induce muscle damage. Subsequent assessment of flexor carpus ulnaris muscle oxygen saturation and wrist-flexor strength occurred at 1, 24, and 48 hours postexercise. The experimental group underwent 10 minutes of local VT (45 Hz) starting 1 hour postexercise and applied twice daily (separated by 8 hours) for 48 hours during habitual waking hours. The control group received no local VT.

Main Outcome Measure(s): Resting muscle oxygen saturation (SmO_2), grip strength, and muscle oxygen desaturation and resaturation rates.

Results: No difference in SmO_2 resaturation was evident over time ($P > .05$), but the VT group had a greater resaturation rate than the control group at 1 hour ($P = .007$, $d = 2.6$), 24 hours ($P = .001$, $d = 3.1$), and 48 hours ($P = .035$, $d = 1.7$) post-EIMD. No difference in grip strength was observed pre-EIMD, but the VT group demonstrated greater strength at 1 hour ($P = .004$), 24 hours ($P = .031$), and 48 hours ($P = .021$) post-EIMD than did the control group.

Conclusions: Local VT successfully attenuated the effects of EIMD and increased SmO_2 resaturation in flexor carpus ulnaris muscles. Including local VT as part of a recovery protocol post-EIMD could be beneficial for rehabilitation and strength training purposes.

Key Words: muscle oxygen saturation, near-infrared spectroscopy, occlusion

Key Points

- Ten minutes of intermittent local vibration therapy (45 Hz) attenuated the effects of exercise-induced muscle damage throughout the study.
- Based on near-infrared spectroscopy, we noted greater muscle oxygen resaturation rates after exercise-induced muscle damage in the experimental group after vibration therapy than in the control group.
- Including local, intermittent vibration therapy as part of a postexercise recovery strategy for smaller muscle groups could be beneficial for rehabilitation and strength training purposes.

Exercise-induced muscle damage (EIMD) is commonly associated with the delayed onset of muscle soreness, a phenomenon that results in reduced joint range of motion[1] and muscular power and force generation[2] and increased inflammation.[3] Previous researchers[3] suggested that eccentric muscle contraction caused a greater level of EIMD symptoms than concentric contraction by negatively affecting local and systemic hemodynamic and macrovascular and microvascular morphology. Consequently, EIMD after eccentric exercise typically compromised the supply of oxygenated blood to active muscles from 24 to 72 hours.[4] In terms of athletic performance, the primary symptom of EIMD is *impaired muscle function and strength*, herein defined as reduced capacity for muscle force production. Investigators[5] who induced local muscle ischemia proposed that the initial strength reduction in the working skeletal muscle was due to reduced oxygen availability. Thus, it would be advantageous for individuals who experience EIMD to reduce these negative effects on performance by increasing the availability of oxygen within the muscle.

To date, several ergogenic aids to help attenuate the effects of EIMD have been used by athletes. One such aid is massage. However, access to a trained and sometimes costly massage therapist is often limited to athletes with high levels of support. Foam rolling is a more readily available and cheaper alternative form of deep tissue massage that is effective in reducing EIMD symptoms. Improved outcomes in performance-related variables such as vertical jump height have been recorded after its use

subsequent to damage-inducing exercise.[2] However, foam rolling can induce considerable mechanical pressure on the underlying tissues, more than twice the pressure used during occlusion and 10-fold higher than the highest medical compression category.[6] It is not surprising that foam rolling is often painful, particularly when swelling and tenderness are present with EIMD.[2] Considering the potential risks to the underlying vascular and lymphatic structures, foam rolling should be used with caution.[6]

Vibration therapy (VT) is another technique known to improve muscle blood flow and oxygenation.[7] Vibration therapy is administered to either the whole body, typically via plates through closed chain positions (ie, hands or feet on the plate), or locally, in which a device applies VT directly to a specific region of the body.[8] Irrespective of mode, VT is accessible and can be administered consistently at varying intensities according to individual comfort. This modality is already used in athletic rehabilitation and sports performance settings to enhance strength,[9] manage recovery from injury,[10] and increase joint range of motion.[11] Of note, both whole-body[12] and local VT[8] alleviated the effects of EIMD when administered before and after EIMD protocols. Although the 2 modes have not been directly compared, the size of the vibration reaching the target tissue from whole-body VT has been suggested to be less than that of local VT, due to signal dissipation into the surrounding unaffected tissues.[8] In addition, whole-body VT is usually limited to large commercial gyms; local VT is more accessible due to its relatively lower cost and high portability. Furthermore, Games et al[13] concluded that local VT, which was applied to unloaded body segments, might be more effective than whole-body VT, which is applied to loaded body segments. Unloaded muscles are relaxed; thus, the small blood vessels supplying these muscles are not subject to the same levels of pressure from the surrounding muscle tissue otherwise observed during contraction.[14] Consequently, with less vascular compression, improved blood flow through the muscle microvasculature can be expected; however, this hypothesis is speculative given the lack of direct assessment of muscle blood flow using local VT after EIMD.

Therefore, the aim of our study was to determine whether local VT modulated oxygenation to the muscle and attenuated the strength loss associated with EIMD in the wrist-flexor muscle group more than no VT. We hypothesized that local VT would modulate muscle oxygenation and aid in maintaining strength after EIMD.

METHODS

Participants

A total of 10 participants (men = 6, women = 4; age = 38 ± 15 years, height = 1.72 ± 0.48 m, mass = 72.0 ± 10.4 kg) with no previous or current upper body musculoskeletal conditions, who described themselves as healthy, and who had no previous experience with resistance training that specifically targeted the arms[15] were recruited for the study. Additional inclusion criteria were no history of smoking, which is known to impair peripheral blood flow,[16] and not using anti-inflammatory medication, which has been shown to reduce the effects of EIMD.[17] Participants were randomly allocated to the treatment group (VT, n = 5) or control group (no VT, n = 5). All participants gave written consent, ethical approval was granted by the local ethics committee of Manchester Metropolitan University, and all procedures complied with the Declaration of Helsinki.

Experimental Procedure

Participants were required to attend 4 testing sessions: at baseline and 1, 24, and 48 hours post-EIMD protocol. Anthropometric assessment of height and mass and administration of an EIMD protocol were conducted during the baseline session only, whereas muscle oxygen saturation, wrist-flexor strength, and all exercise protocols were conducted at each session. Participants were advised to avoid vigorous exercise for 48 hours before and throughout the study.

Muscle Oxygen Measures

After arrival at the laboratory for baseline data collection, participants assumed a supine position for the assessment of blood pressure in the right arm. After a 10-minute rest period to allow blood flow to return to normal,[18] muscle oxygen saturation (SmO_2) of the flexor carpi ulnaris (FCU) was measured using a portable near-infrared spectroscopy (NIRS) sensor (Moxy Monitor, Fortiori Design LLC). The NIRS sensor was placed on the skin of the wrist flexors, midway between the styloid process of the wrist and the superior radio-ulnar joint process, using an adhesive dressing. A light shield was placed over the monitor to prevent ambient light pollution.[19] The NIRS sensor placement was identified with a permanent marker to ensure the reliability of sensor placement on subsequent testing days, particularly because blood flow and oxygen utilization (9%–13%) are known to be heterogeneous within a muscle.[20] With participants remaining supine, we recorded SmO_2 for 5 minutes; resting SmO_2 was determined as the peak value recorded during this period once stability was achieved (no greater than 3%–5% fluctuation in 30 seconds[4]). Subsequently, the brachial artery was occluded using a manual sphygmomanometer cuff placed 2 to 3 cm above the antecubital fold. In line with previous research,[5] pressure in the sphygmomanometer cuff was quickly inflated (<3 seconds) to a suprasystolic level of 30 mm Hg above the baseline systolic blood pressure (150–180 mm Hg) to ensure cessation of blood flow in the brachial artery. The occlusion was maintained for 3 minutes and immediately released; desaturation and resaturation rates were then measured to express the rate of change (kinetics) of SmO_2. During occlusion, SmO_2 was continuously recorded for 3 minutes, with the lowest value obtained identified as the nadir. The absolute difference between peak resting SmO_2 and nadir SmO_2 values was then used to calculate the rate of desaturation ($\%\cdot min^{-1}$) as (peak SmO_2 − nadir SmO_2) / 3. After deflation of the arm cuff, SmO_2 "recovery" was measured for 3 minutes, with the SmO_2 at 3 minutes recorded and used to calculate the rate of resaturation ($\%\cdot min^{-1}$) as (recovery SmO_2 − nadir SmO_2) / 3. Data were collected in real time by Bluetooth transmission between the NIRS device and a separate computer via an ANT+ sensor (Garmin Ltd). The data were processed through Peripedal computer software (version 2.4) and saved in .csv format. Near-infrared spectroscopy has been validated against

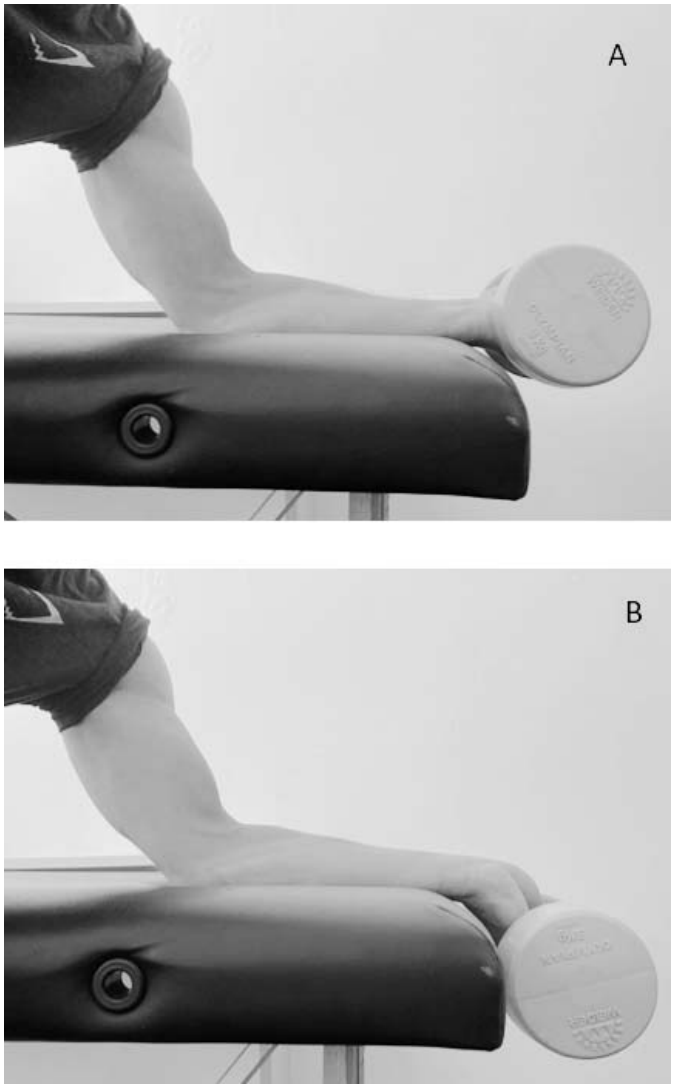

Figure 1. A) Starting position with the forearm resting on a plinth and the wrist in neutral alignment. B) End range of motion with maximum wrist extension and the forearm in neutral alignment.

magnetic resonance[21] spectroscopy ($r = .965$) and strain-gauge plethysmography as an accurate device for measuring forearm blood flow and muscle oxygenation.[22] Measures of muscle oxygen saturation were repeated at 1 hour and 24 and 48 hours post–damage-inducing exercise protocol.

Strength Measures

After assessment of muscle oxygenation, wrist-flexor strength was measured using a constant digital handheld dynamometer (model EH101; Camry Scale). Participants sat in an upright position with the upper arm relaxed by the side of the torso and the elbow flexed to 90°. The hand was supinated, with the dorsal surface placed on a table and in neutral alignment with the forearm. After a demonstration, participants were instructed and orally encouraged to squeeze the dynamometer for approximately 5 seconds. This was repeated 3 times, and the peak force (N) of the 3 trials was recorded. Peak wrist-flexor strength was assessed at 1, 24, and 48 hours post-EIMD protocol.

Determination of 1-Repetition Maximum

To determine the exercise load to be used for the muscle-damaging protocol, participants completed an assessment of their 1-repetition maximum (1-RM) for the wrist flexors. They were initially seated with the elbow flexed to 90° and the forearm resting on the plinth of a biceps-curl machine. To isolate control of movement of the wrist flexors, the distal part of the limb (wrist to fingers) was not supported by the plinth. After a series of warm-up contractions, each participant self-selected a starting dumbbell weight for the assessment of 1-RM. The weight was passed to the participant when he or she was in the prescribed starting position: that is, with the wrist and forearm parallel (neutral alignment) and resting supine on the table (Figure 1A). The dumbbell was lowered over 3 seconds to the end range of wrist extension (Figure 1B) before being returned to the starting position over 1 second while the supinated arm position was maintained, in line with earlier protocols.[5] If the participant completed 1 repetition of each weight, this was increased by 1 kg, and the procedure was repeated after a 2-minute rest. The 1-RM was identified as the final load completed without failing to return the dumbbell to the starting position within 1 second. Consistent oral encouragement was given to each participant during the assessment. After determination of the 1-RM, participants rested for 10 minutes before undergoing the muscle-damage protocol.

Exercise-Induced Muscle-Damage Protocol

Using the same setup as described for the identification of 1-RM, participants completed 10 sets of 10 eccentric wrist-flexion repetitions, with a 60-second recovery between sets using a load of 70% of 1-RM, consistent with a study that involved induced muscle damage.[15] Participants were instructed to take 3 seconds to lower the dumbbell to the maximal comfortable range and then return to neutral over 1 second.[22]

Vibration Therapy

After the EIMD protocol, participants were asked to refrain from any strenuous exercise or consuming pain relief or anti-inflammatory medication during the subsequent 48 hours.[17] Those in the control group were asked to continue with their usual habitual activity during this time and return for assessments of SmO_2 and strength at 1, 24, and 48 hours post-EIMD. The VT group self-administered VT using a Pulseroll (Shenzen Technologies) standard commercial vibrating foam roller twice daily (separated by 8 hours) for 48 hours post-EIMD because the effects of EIMD are known to manifest during this time.[23,24] The procedure was demonstrated for all participants in the VT group before self-administration. They were supervised during their first VT to ensure that the application of pressure and region of administration were correct, whereas the remaining VT treatments were completed unsupervised. The VT focused application of the Pulseroll on the previously marked area of the FCU muscle belly using the uninvolved arm to ensure that only vibration was applied without external pressure on the muscle.[8] Participants were instructed to administer VT at a frequency of 45 Hz for 10 minutes. The first VT treatment occurred at 1

Table. Near-Infrared Spectroscopy Data by Group During Occlusion at Rest and After Exercise-Induced Muscle Damage

	SmO_2							
	%				%·min^{-1}			
	Resting		Nadir		Desaturation Rate		Resaturation Rate	
Time	VT	Control	VT	Control	VT	Control	VT	Control
Baseline	62.4 ± 5.9	63.4 ± 6.4	42.2 ± 3.6	40.8 ± 4.5	6.7 ± 2.4	7.5 ± 2.0	12.0 ± 2.4	11.2 ± 2.3
1 h	66.6 ± 6.5	63.6 ± 8.2	33.4 ± 3.4[a]	46.2 ± 8.8	10.5 ± 3.5	6.9 ± 1.8	16.0 ± 3.2[a]	10.4 ± 1.4
24 h	66.4 ± 5.8	63.2 ± 8.9	35.2 ± 8.8[a]	42.8 ± 5.3[a]	11.0 ± 2.3	5.7 ± 2.8	16.5 ± 1.8[a]	9.3 ± 2.3
48 h	67.2 ± 5.9	62.0 ± 10.3	39.4 ± 4.9[c]	43.8 ± 9.3[b]	9.3 ± 3.3	6.1 ± 3.8	14.3 ± 2.1[a]	9.8 ± 3.4

Abbreviation: VT, vibration therapy.

* Between-groups differences ($P < .05$). Differences from [a] baseline, [b] 1 h, and [c] 24 h.

hour post-EIMD, and the timing of all VT treatments was the same on each day. To ensure that participants administered the VT at the correct time, they received a text reminder approximately 1 hour before each treatment.

Data Analysis

We performed the statistical analysis using SPSS (version 25; IBM Corp). The SmO_2 and wrist-flexor strength values were tested for normality (Shapiro-Wilk), equal variance (Levene), and sphericity (Mauchly) before being tested for effects using a 2 × 4 (group × time) mixed-measures analysis of variance. Bonferroni adjusted post hoc pairwise comparisons were conducted when significant main effects were present. The α value was set at $P < .05$. All data are presented as mean ± standard deviation. Effect sizes for pairwise comparisons were calculated using the Cohen *d* to determine the magnitude of the difference between groups and were classified as <0.2, *low*; 0.21–0.5, *medium*; 0.51–0.8, *large*; or >0.81, *very large*. In addition, partial η^2 was used to indicate the magnitude of the effect between conditions and classified as 0.01, *small*; 0.09, *medium*; or 0.25, *large*.

RESULTS

Oxygen Saturation

No effect of time ($F_{3,24} = 1.703$, $P = .193$, $\eta_p^2 = 0.388$) or group ($F_{1,8} = 0.33$, $P = .578$, $\eta^2 = 0.040$; Table) was present for resting SmO_2 (Figure 2). Nadir SmO_2 did not differ between groups ($F_{1,8} = 2.495$, $P = .153$, $\eta_p^2 = 0.238$) or over time when compared with baseline ($F_{3,24} = 1$, $P = .225$, $\eta_p^2 = 0.163$). However, we observed a group × time interaction ($F_{3,24} = 8.359$, $P = .001$, $\eta_p^2 = 0.511$). Post hoc analyses revealed that nadir SmO_2 was lower in the VT group than in the control group only at 1 hour post-EIMD ($P = .027$).

A significant main effect in the rate of SmO_2 desaturation was demonstrated post-EIMD protocol ($F_{3,24} = 3.030$, $P = .049$, $\eta_p^2 = 0.275$), but this did not differ between groups ($F_{1,8} = 2.906$, $P = .127$, $\eta_p^2 = 0.266$; Figure 3). The rate of desaturation was faster at 24 hours than at baseline ($P = .037$); no other differences existed ($P > .05$; Table).

We found a main effect of time on the SmO_2 resaturation rate ($F_{3,24} = 4.339$, $P = .014$, $\eta_p^2 = 0.352$) and a greater resaturation rate in the VT group than in the control group ($F_{1,8} = 10.35$, $P = .012$, $\eta_p^2 = 0.564$; Figure 4). The groups did not differ in the rate of SmO_2 resaturation at baseline ($P = .611$), but at 1 hour ($P = .007$, $d = 2.6$), 24 hours ($P = .001$, $d = 3.1$), and 48 hours ($P = .035$, $d = 1.7$), post-EIMD was higher in the VT group than in the control group (Table). For the VT group, resaturation of SmO_2 was greater at 1 hour ($P = .04$, $d = 1.5$), 24 hours ($P = .001$, $d = 2.0$), and 48 hours ($P = .018$, $d = 1.0$) than at baseline; the resaturation rate did not differ for the control group between baseline and any other time point ($P > .05$).

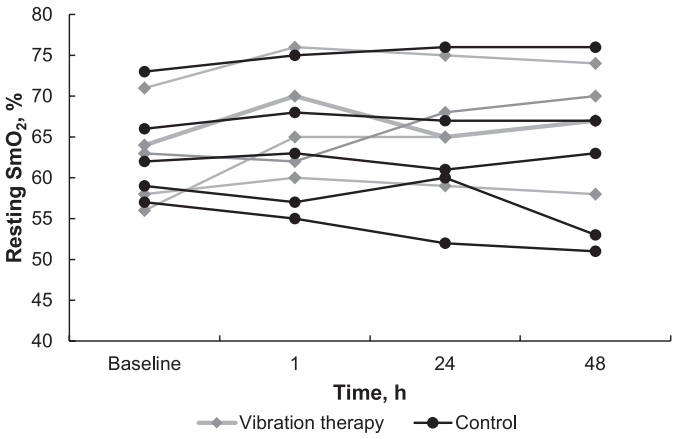

Figure 2. Resting oxygen saturation (SmO_2) in the vibration therapy (VT) group and control group postexercise-induced muscle damage.

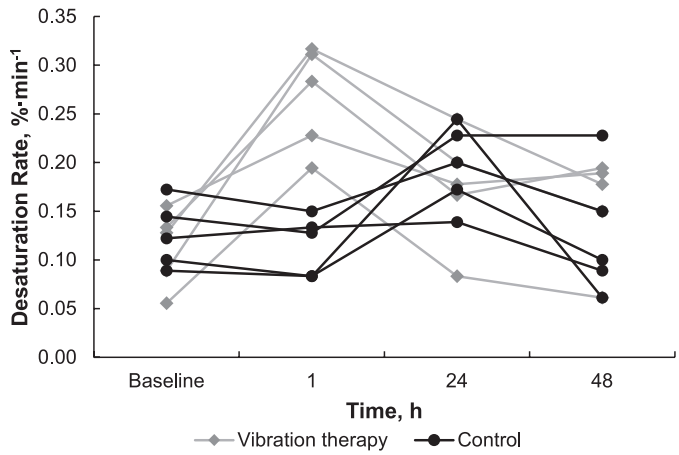

Figure 3. Desaturation rate in the vibration therapy (VT) group and control group postexercise-induced muscle damage.

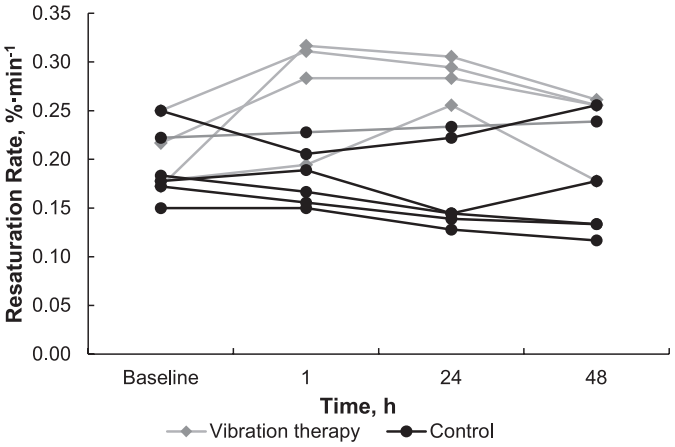

Figure 4. Resaturation rates in the vibration therapy (VT) group and control group postexercise-induced muscle damage.

Wrist-Flexor Strength

A significant effect of time ($F_{3,24} = 7.414$, $P = .001$, $\eta_p^2 = 0.481$) and group × time interaction for strength ($F_{3,24} = 4.338$, $P = .014$, $\eta_p^2 = 0.352$) were noted. The strength of the VT group over time did not change (P values $> .005$), whereas the strength of the control group was lower at 1 hour (4%; $P = .044$, $d = 0.98$), 24 hours (8%; $P = .003$, $d = 1.34$), and 48 hours (5%; $P = .035$, $d = 1.06$) post-EIMD than at baseline. No other strength differences between time points were identified (P values $> .05$; Figure 5).

DISCUSSION

The aim of our study was to determine whether intermittent administration of local VT modulated blood flow and oxygenation to the FCU muscle and attenuated the strength loss associated with EIMD more than no VT. The main findings were that application of intermittent local VT after a muscle damage–inducing exercise protocol resulted in greater FCU SmO_2 resaturation with local VT than with no VT and that VT was more effective in maintaining wrist-flexor strength than no VT.

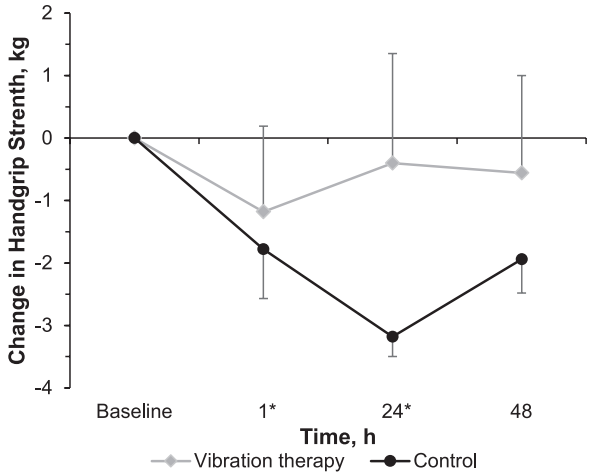

Figure 5. Handgrip strength relative to baseline levels in the vibration therapy (VT) group and control group postexercise-induced muscle damage. No differences were present between groups. * $P < .05$ compared with baseline only in the VT group.

The cause of EIMD is multifactorial, with many researchers seeking the underlying factors that contribute to this syndrome.[25] Strength reduction is considered one of the most valid markers of EIMD,[26] as many studies have demonstrated strength losses due to EIMD.[2] In our investigation, strength in the wrist-flexor muscle group was reduced from baseline at 1 hour and 24 and 48 hours post-EIMD protocol only for the control group, which suggests that the muscle-damage protocol was appropriate. It is interesting, however, that this trend was not present in the experimental group, which received local VT. The magnitude of strength reduction ($\eta_p^2 = 0.352$) in the control group was consistent with previous research[10] on the effects of local VT on muscle strength postdamage, which also indicated a moderate effect size ($d = 0.44$) for strength in the VT versus control groups.

One plausible explanation as to why muscle strength was maintained in the local VT group is the improved resaturation rate of SmO_2 in the VT compared with the control group. In earlier work, Moraleda et al[34] measured the positive effects of VT on EIMD and concluded that a possible cause was the beneficial effect on SmO_2 ($d = 0.96$). They obtained SmO_2 data using NIRS in real time during rest and activity without occlusion. Our effect on SmO_2 was larger ($d = 1.7$–3.1) except via occlusion, which was used to calculate the rate of change of SmO_2, thereby providing better insight into oxygen kinetics and blood flow.

Maximal muscle contraction relies on the continuous use of energy stores, which in turn relies on the adequate delivery of oxygen. Even during anaerobic bouts, when energy is derived from the transfer of phosphate from phosphocreatine to adenosine diphosphate, this energy system is "reloaded" by oxidative means and is therefore heavily dependent on the availability of oxygen.[27,28] In addition, any breakdown in the transport of oxygen, such as damage to the peripheral local muscle microcirculation, compromises muscle function.[29] A relative increase in the resaturation rate of SmO_2 would suggest greater oxygen delivery and, as a result, greater blood flow to the muscle.[22,30] We found that SmO_2 increased by 78% from baseline to 48 hours post-EIMD administration of local VT versus only a 31% increase in the control group. Assuming SmO_2 is an appropriate surrogate for blood flow, this may suggest that the VT group experienced increased blood flow to their damaged wrist flexors. Although we did not directly measure blood flow, previous investigators[30] demonstrated that increased flow to damaged muscles reduced the level of muscle damage biomarkers, such as creatine kinase, more rapidly than when no increase in blood flow occurred. Biomarkers of increased blood flow and reduced levels of damage also correlated with improved recovery from EIMD[30] and would explain the aforementioned maintenance of muscle strength in the VT group versus the reduced strength in the control group after EIMD.

In their local inflammation theory, Gulick and Kimura[31] proposed that increased permeability of the local vasculature after eccentric muscle damage leads to an efflux of metabolites and edema formation within the damaged muscle. Subsequently, a cascade of events leads to increased neutrophil release, macrophage formation, and breakdown of muscle tissue. Furthermore, Egners et al[32] explained that edema formation compromises muscle perfusion and contributes to local hypoxia, compounding

muscle damage. Considering that SmO_2 increased at 48 hours post-EIMD only in our VT group, it is reasonable to assume that VT attenuated the inflammatory cascade, extent of neutrophil margination, and local hypoxia after EIMD. Local VT is known to increase the internal diameter of the vasculature serving damaged muscles,[12] resulting in transient increases in relative blood flow and ultimately enhanced oxygen delivery to this area.[25] Acute changes in SmO_2 reflect dynamic local vascular tone, which controls blood flow, oxygenation, and perfusion rates in the muscle tissue.[22] Increases in relative SmO_2 would be expected with greater blood flow, thus providing an explanation for the attenuation of the inflammatory cascade and higher SmO_2 48 hours post-EIMD only in the VT group.

The timing and dosage of VT application appears to be significant and should not be overlooked. Earlier investigators[33] demonstrated that a single bout of VT pre-EIMD was ineffective in maintaining muscle strength when assessed at 24, 48, and 72 hours post-EIMD. More recently, Moraleda et al[34] showed that a single bout of local VT administered as late as 48 hours post-EIMD was sufficient to improve SmO_2 above baseline (approximately 12%), albeit to a lesser extent than we observed. Dissimilar to the current study, a single application of local VT 48 hours post-EIMD was not able to maintain muscle strength.[34] Repeated bouts of VT, as administered in our study, may incrementally improve the local vascular tone to create a "summative" benefit over time. Such a summation would contribute to enhanced blood flow and explain the higher SmO_2 observed, although this needs to be shown empirically. It is possible, therefore, that the acute benefits of a single application of local VT are not sufficient to attenuate the symptoms of muscle damage, given that these diminish in the absence of reapplication,[35] providing evidence that multiple bouts of VT may be more effective than single bouts for improving SmO_2 post-EIMD. Further research is required to ascertain the optimal windows of application and identify the mechanisms underpinning a potential summative effect of the therapy.

An alternative explanation for our results is the facilitation of "functional hyperemia," a recognized reaction whereby an increase in local muscle metabolism initiates compensatory vasodilation.[25] The normal inflammatory process seen in EIMD hinders local blood flow[25,26] and leads to unfavorable leakage of intramuscular cell contents, ultimately inhibiting normal muscular contraction and causing loss of strength.[26] The higher SmO_2 in our VT group suggests that local VT enhanced the vasomotor response, increasing the local muscle oxygen level and reversing some of these inflammatory processes post-EIMD.[4] Kerschan-Schindl et al[12] reported that whole-body VT post-EIMD enhanced the vasodilation of small arterioles and capillaries. Because the target muscles are likely to experience a more intense vibration from local VT than from whole-body VT,[8] the former therapy may induce local reactive vasodilation to a greater extent than that noted after whole-body VT. We did not measure biomarkers of muscle damage or inflammation, so this explanation remains speculative and would benefit from further examination. Nonetheless, repeated applications of local VT are preferable to whole-body VT when attempting to limit the extent of EIMD after unaccustomed eccentric exercise.

In our study, blood flow was controlled with a single cuff on the upper arm. Although this worked well to occlude the arterioles, a more appropriate method would have been to place a second cuff on the wrist to occlude the venous circulation. Without this second occlusion point, we must assume that some blood moved out of the compartment into the venous system, potentially affecting the NIRS data and subsequent inferences relating to muscle metabolism. However, the size of this effect is unknown.

Our sample size was smaller than that in other studies.[7] We are the first to show that VT improved the effects of EIMD and blood oxygenation. Although these effects were positive, we recognize that the sample size and muscle group were both small. A post hoc G*Power analysis demonstrated that the effect was strong enough to avoid a type I error ($n = 6$). Furthermore, the nonparametric test helped to ensure that the error rate was nullified as much as possible, although the results should still be interpreted with caution. Future research involving additional muscle groups is required to ensure that the positive effects we observed also occur in larger muscle groups that more commonly exhibit EIMD, such as the quadriceps.

A future consideration would be to include subjective pain scoring to assess the effectiveness of EIMD treatment, as has been done in other work evaluating the effectiveness of interventions.[11] We appreciate that pain is often used as a proxy marker for "recovery" from EIMD. However, authors[1,36] who specifically assessed measurement tools used in EIMD studies, such as muscle torque, range of motion, and histologic changes, argued that subjective "soreness" scores correlated poorly with actual muscle function and, therefore, subsequent damage from eccentric loading. Strength was a more reliable marker (ie, intraclass correlation coefficients ≥ 0.85) for measuring muscle function and resultant recovery post-EIMD.[1] Furthermore, peak loss of muscle function due to EIMD reportedly occurs within the first 24 to 48 hours, whereas peak soreness occurs later, between 48 and 72 hours.[1] This effect was observed by Moraleda et al[34]: their participants described 30.2% less pain according to visual analog scores obtained 48 hours after the EIMD protocol and VT intervention. In line with this research, specifically the 48-hour timeline, we believe the objective measure of strength is the best tool for quantifying the effects of EIMD and determining the effectiveness of the intervention.

CONCLUSIONS

Application of local VT therapy appears to have contributed to attenuating the effects of EIMD on muscle strength and blood oxygenation in the wrist-flexor muscles. To our knowledge, this study is the first to show that VT contributed to alleviating some EIMD symptoms when administered multiple times post-EIMD, which could be due to a summative effect. Including local VT as part of postexercise recovery strategies for smaller muscle groups could be beneficial for rehabilitation and strength training purposes, although more work is warranted in this area to substantiate the current findings and apply them to larger muscle groups.

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