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***Title:*** Local vibration therapy increases oxygen re-saturation rate and maintains muscle strength following exercise-induced muscle damage



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Effect of vibration therapy on muscle damage

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# Local vibration therapy increases oxygen re-saturation rate and maintains muscle strength following exercise-induced muscle damage


## *Abstract*

### Context

Exercise induced muscle damage (EIMD) is associated with transient reductions in strength and athletic performance. Studies conclude aetiology is due in part to muscle micro vascular damage and disruption of blood flow. Previous research on vibration therapy reports modulation in muscle blood flow, oxygenation and strength.

### Objective

The aim of this study was to observe if local vibration therapy (VT) alleviates the impairments and haemodynamic changes associated with EIMD.

### Design

Controlled laboratory study

### Setting

Laboratory and public gymnasium

### Patients or other participants

Ten healthy participants (6 males: 4 females; age: 38±15 yrs; height: 1.72±0.48 m; mass 72.0±10.4 kg) were randomized into experimental (VT) and control (CON) groups.

### Interventions

Both groups performed 10 sets of 10 eccentric wrist flexions at 70% of 1-repetition maximum to induce muscle damage. Subsequent assessment of wrist flexor strength and flexor carpus ulnaris (FCU) muscle oxygen saturation ($SmO_2$) occurred at 1-, 24- and 48 hr-post exercise. VT group underwent 10 min of local VT (45 Hz) starting 1 hr-post exercise and applied twice daily (separated by 8 hrs) for 48 hrs during habitual waking hours. CON group received no local VT.

**Main outcome measure(s)**

Grip strength, resting muscle oxygen ($SmO_2$), muscle oxygen de-saturation and re-saturation rate.

**Results**

No difference in grip strength observed pre EIMD, but the VT group demonstrated greater strength at 1 hr (P=0.004), 24 hr (P=0.031) and 48 hr (P=0.021) post EIMD compared to controls. No difference in $SmO_2$ re-saturation over time (P>0.05), but the VT group had a greater re-saturation rate compared to controls at 1 hr (P=0.007, $d$ = 2.6), 24 hr (P=0.001 $d$ = 3.1) and 48 hr (P=0.035, $d$ = 1.7) post EIMD.

**Conclusions**

Local VT successfully attenuated the effects of EIMD and increased $SmO_2$ re-saturation in FCU muscles. Including local VT as part of a recovery protocol post-EIMD could be beneficial for rehabilitation and athletic training purposes.

**Key words:** muscle oxygen saturation, vibration therapy, exercise induced muscle damage, near infrared spectroscopy, occlusion

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**Key points:**

- 10 minutes of intermittent local vibration therapy (45Hz) attenuated the effects of EIMD throughout the duration of the study.
- Greater muscle oxygen re-saturation rates post EIMD were observed via near infra-red spectroscopy following vibration therapy compared to a control group.
- Including local intermittent vibration therapy as part of post-exercise recovery strategy for smaller muscle groups could be beneficial for rehabilitation and athletic training purposes.

Exercise induced muscle damage (EIMD) is commonly associated with delayed onset of muscle soreness (DOMS); a phenomenon that results in reductions in joint range of motion (ROM)[1] muscular power and force generation[2] and increased inflammation.[3] Evidence from previous research suggests that eccentric muscle contraction causes a greater level of EIMD symptoms than concentric contraction, by negatively impacting local and systemic haemodynamic and macro and micro-vascular morphology.[3] Consequently, EIMD following eccentric exercise typically compromises the supply of oxygenated blood to active muscles for between 24-72 hrs.[4] In terms of athletic performance, the primary symptom of EIMD is the impairment of muscle function and strength, hereby defined as reduced capacity of muscle force production. Findings from previous studies, in which the researchers induced local muscle ischemia, suggest that the initial reduction in strength in the working skeletal muscle occurs due to reduced oxygen availability.[5] Thus, it would be advantageous for individuals who experience EIMD to reduce these negative effects on performance, which may be achieved by increasing the availability of oxygen within the muscle.

To date, several ergogenic aids to help attenuate the effects of EIMD are utilised by athletes. One such aid is the use of massage. However, access to a trained and sometimes costly masseur is often limited to athletes that have access to high levels of support. Foam rolling is a more readily available and cheaper alternative form of deep tissue massage that is effective at reducing the symptoms of EIMD. Improved outcomes in performance-related variables such as vertical jump height have been recorded following its usage subsequent to damage-inducing exercise.[2] Foam rolling, however, can induce considerable mechanical pressure on the underlying tissues; exceeding twice the pressure used during occlusion and 10-fold higher

than the highest medical compression category.[6] Unsurprisingly, foam rolling is often painful, particularly when swelling and tenderness are present with EIMD.[2] Considering the potential risks to underlying vascular and lymphatic structures, the use of foam rolling should be done with caution.[6]

Vibration therapy (VT) is another alternative technique known to improve muscle blood flow and oxygenation.[7] VT is administered to either the whole-body, typically via plates through closed-chain positions (i.e. hands or feet on the plate), or locally, where 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. VT 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] Importantly, both whole body[12] and local VT[8] have been shown to alleviate the effects of EIMD when administered before and after EIMD protocols. Whilst there are no direct comparisons between the two modes, it has been suggested that the size of the vibration reaching the target tissue from whole-body VT is most likely reduced compared to local VT, due to signal dissipation into the surrounding, non-affected, tissues.[8] Additionally, whole body VT, is usually limited to large commercial gyms, with local VT more accessible due to relatively lower cost and high portability. Furthermore, Games et al.[13] concluded that application of local VT, which occurs on 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 could be expected, however this hypothesis is speculative as there appears to be no direct assessment of muscle blood flow using local VT following EIMD.

Therefore, the aim of this study was to determine whether local VT modulates oxygenation to the muscle and attenuates the strength loss associated with EIMD in the wrist flexor muscle group compared to no VT. It was hypothesised that local VT would modulate muscle oxygenation and aid in maintaining strength following EIMD.

## *Methods*

### *Participants*

Ten participants (mean ±SD; male N = 6; female N = 4; age 38 ±15 yrs; height 1.72 ±0.48 m; mass 72.0 ±10.4 kg) with no previous or current upper body musculoskeletal conditions, described themselves as healthy and without previous experience of resistance training that specifically targeted the arms[15] were recruited for the study. Inclusion required that all participants had no prior history of smoking, which is known to impair peripheral blood flow,[16] or had not been using anti-inflammatory medication which has been shown to reduce the effects of EIMD.[17] Participants were randomly allocated to a treatment group (VT, N = 5) or control group (i.e., 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*

All participants were required to attend four testing sessions, at baseline, and 1 hr, 24 hr and 48 hr post EIMD protocol. Anthropometric assessment of height and mass and administration of an EIMD protocol were conducted during the baseline session only, whilst muscle oxygen saturation, wrist flexor strength and all exercise protocols were conducted at each session. Participants were advised to avoid vigorous exercise for 48 hrs prior to and throughout the study duration.

***Muscle Oxygen Measures***

Following arrival at the laboratory for baseline data collection, participants assumed a supine position for the assessment of blood pressure, taken from the right arm. Following a 10 min 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 portable near infra-red spectroscopy (NIRS) sensor (MOXY monitor© Fortiori design LLC, Hutchinson, Minnesota 55350). 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 adhesive dressings. A light shield was also 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 as there is known heterogeneity of blood flow and oxygen utilisation (9-13%) within a muscle.[20] With participants remaining supine, $SmO_2$ was recorded for 5 mins, with resting $SmO_2$ determined as the peak value recorded during this period once stability was achieved (no greater than 3-5% fluctuation in 30 seconds[4]). Subsequently, occlusion of the brachial artery was undertaken using a manual sphygmomanometer cuff placed approximately 2-3 cm above the antecubital fold. In line

with previous research, pressure in the sphygmomanometer cuff was quickly inflated (<3 seconds) to a supra-systolic level of 30 mmHg above the baseline systolic blood pressure (150-180 mmHg) to ensure cessation of blood flow in the brachial artery.[5] The occlusion was maintained for 3 min and immediately released, desaturation and re-saturation rates were then measured to express the rate of change (kinetics) of muscle oxygen saturation. During occlusion, $SmO_2$ was continuously recorded for 3 mins, with the lowest value obtained determined 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. Following deflation of the arm cuff, $SmO_2$ 'recovery' was measured for 3 min, with the $SmO_2$ at 3 min recorded and used to calculate rate of re-saturation ($\%\cdot min^{-1}$) as: (recovery $SmO_2$ – nadir $SmO_2$)/ 3. Data was collected in real time by Bluetooth transmission between the NIRS device and a separate computer via an ANT+ sensor (Garmin Ltd ©, Schaffhausen, Switzerland). The data was processed through Peripedal© computer software and saved in .csv format. NIRS has previously been validated as an accurate device for measuring forearm blood flow and muscle oxygenation against magnetic resonance spectroscopy ($r = 0.965$)[21] and strain-gauge plethysmography.[22] Measures of muscle oxygen saturation were repeated at 1, 24 and 48 hours post damage inducing exercise protocol.

***Strength Measures***

Following assessment of muscle oxygenation, wrist flexor strength was measured using a constant digital handheld dynamometer (Camry Scale, EH101, South El Monte, CA, USA). Participants sat in an upright position with their upper arm relaxed by the side of the torso and the elbow flexed to 90°. Their hand was supinated, with the dorsal surface placed on a

table and in neutral alignment with the forearm. After a demonstration, participants were instructed to squeeze the dynamometer for ~5 s with verbal encouragement given to all participants. This was repeated three times and the peak force (N) of the three trials was recorded. Assessment of peak wrist flexor strength was repeated at 1, 24 and 48 hrs post-EIMD protocol.

***Determination of One Repetition Maximum (1RM)***

In order to determine the exercise load to be used for the muscle damaging protocol, participants completed an assessment of their one repetition maximum (1RM) for the wrist flexors. Initially, participants were seated with their elbow flexed at 90° to the upper arm and forearm resting on the plinth of a bicep curl machine. In order to isolate control of the movement to the wrist flexors, the distal part of the limb (wrist to fingers) was not supported by the plinth. Following a series of warm-up contractions, participants self-selected a starting dumbbell weight to commence the assessment of 1RM, which was passed to the participant when they were in the prescribed starting position, i.e., with the wrist and forearm parallel (neutral alignment) and rested supine on the table (Figure 1A). Initially, the dumbbell was lowered over 3 s to the end range of motion of wrist extension (Figure 1B), before being returned to the starting position over 1s whilst maintaining the supinated arm position, in line with previous protocols.[5] Participants completed one repetition of each weight and, if successful, this was increased by 1 kg and the procedure was repeated following a 2 min rest. 1RM was identified as the final load completed without failing to return the dumbbell to the starting position within 1 second. Consistent verbal encouragement was given to each

192 participant during the assessment. Following identification of the 1RM, participants rested for
193 10 min before undergoing the muscle-damaging protocol.

194

195 ***Exercise Induced Muscle Damage Protocol***

196 Using the same set-up as described above for the identification of 1RM, participants
197 completed 10 sets of 10 eccentric wrist flexion repetitions, with 60 seconds recovery between
198 sets using a load of 70% of 1RM in line with previous research that induced muscle
199 damage.[15] As previously stated, participants were instructed to take 3 s to lower the dumbbell
200 to the maximal comfortable range and then return to neutral over 1 s. [22]

201

202 ***Vibration Therapy***

203 Following the EIMD protocol, all participants were asked to refrain from completing any
204 strenuous exercise or consumption of pain relief and anti-inflammatory medication during the
205 48 hrs post protocol.[17] The control group were asked to continue with their usual habitual
206 activity during this time and return for assessments of muscle oxygen saturation and strength
207 at 1 hr, 24 hr and 48 hr post-EIMD. The VT group self-administered VT using a Pulseroll©
208 (Shenzen technologies, Shenzen, China) standard commercial vibrating foam roller twice
209 daily (separated by 8 hours) for 48 hrs post EIMD, as the effects of EIMD are known to
210 manifest between this time. [23,24] A demonstration of the correct procedure was given to all
211 participants in the VT group prior to self-administration and all participants were supervised
212 during their first VT to ensure the application of pressure and region of administration were
213 correct, whilst the remaining VT treatments were completed unsupervised. VT involved

focussed application of the Pulseroll© on the previously marked area of the FCU muscle belly using the non-involved arm to ensure that only vibration was applied and no external pressure to the muscle.[8] Participants were instructed to administer VT at a frequency of 45 Hz for 10 mins during each administration. The first VT treatment occurred at 1 hr post EIMD, and the timing of all VT treatments was the same on each day. To ensure participants administered the VT at the correct time, they received a text reminder approximately 1 hr prior to each treatment.

### *Data analysis*

Statistical analysis was performed using SPSS (IBM SPSS statistics for Mac, version 25. Armonk, NY: IBM corp). Wrist flexor strength and $SmO_2$ values were tested for normality (Shapiro-Wilk), equal variance (Levene's) and sphericity (Mauchly's) before being tested for effects using a 2x4 (group x time) mixed measures ANOVA. Bonferroni adjusted post-hoc pairwise comparisons were completed on significant main effects. Alpha was set at $P < 0.05$ and all data are presented as mean ± standard deviation. Effect sizes for pairwise comparisons were calculated using Cohen's *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 and >0.81 very large. In addition, partial eta squared ($\eta^2$) was used to show the magnitude of the effect between each condition and classified as 0.01 (small), 0.09 (medium) and 0.25 (large).

## *Results*

### *Wrist Flexor Strength*

There was a significant effect of time ($F_{(3,24)} = 7.414$, $P = 0.001$ $\eta^2 = 0.481$) and group*time interaction for strength ($F_{(3,24)} = 4.338$, $P = 0.014$ $\eta^2 = 0.352$). There was no change in strength of the VT group over time ($P > 0.005$) whereas strength of the control group was lower at 1 hr (4%, $P = 0.044$, $d = 0.98$), 24 hr (8%, $P = 0.003$, $d = 1.34$) and 48 hr-post EIMD (5%, $P = 0.035$, $d = 1.06$) compared to baseline. There were no other strength differences between time points ($P > 0.05$, Figure 2).

< INSERT FIGURE 2 NEAR HERE >

***$SmO_2$***

There was no effect of time ($F_{(3,24)} = 1.703$, $P = 0.193$, $\eta^2 = 0.388$) or group ($F_{(1,8)} = 0.33$, $P = 0.578$, $\eta^2 = 0.040$, Table 1) for resting $SmO_2$ (Figure 3). Nadir $SmO_2$ did not differ significantly between groups ($F_{(1,8)} = 2.495$, $P = 0.153$, $\eta^2 = 0.238$) or over time when compared to baseline ($F_{(3,24)} = 1.$, $P = 0.225$, $\eta^2 = 0.163$, Table 1). There was, however, a group*time interaction ($F_{(3,24)} = 8.359$, $P = 0.001$, $\eta^2 = 0.511$). Post hoc analyses revealed that nadir $SmO_2$ was lower in the VT group compared to the control group at 1 hr post EIMD only ($P = 0.027$, Table 1).

< INSERT FIGURE 3 NEAR HERE >

< INSERT TABLE 1 NEAR HERE >

There was a significant main effect in the rate of $SmO_2$ desaturation post EIMD protocol ($F_{(3,24)} = 3.030$, $P = 0.049$, $\eta^2 = 0.275$), but this did not differ between groups ($F_{(1,8)} = 2.906$, $P = 0.127$, $\eta^2 = 0.266$) (Figure 4). Rate of desaturation was faster at 24 hrs than at baseline ($P = 0.037$) while no other differences existed ($P > 0.05$, Table 1).

< INSERT FIGURE 4 NEAR HERE >

There was a main effect of time on $SmO_2$ re-saturation rate ($F_{(3,24)} = 4.339$, $P = 0.014$, $\eta^2 = 0.352$) and the VT group had a significantly greater re-saturation rate compared to the controls ($F_{(1,8)} = 10.35$, $P = 0.012$, $\eta^2 = 0.564$) (Figure 5). There was no difference between groups for rate of $SmO_2$ re-saturation at baseline ($P = 0.611$), but rate of $SmO_2$ re-saturation at 1 hr ($P = 0.007$, $d = 2.6$), 24 hr ($P = 0.001$, $d = 3.1$) and 48 hrs ($P = 0.035$, $d = 1.7$) post EIMD was higher in the VT group compared to controls (Table 1). For the VT group, re-saturation of $SmO_2$ was greater at 1 ($P = 0.04$, $d = 1.5$), 24 ($P = 0.001$, $d = 2.0$) and 48 hrs ($P = 0.018$, $d = 1.0$) compared to baseline, whereas there was no difference in re-saturation rate for controls between any baseline and any time point ($P > 0.05$, Table 1).

< INSERT FIGURE 5 NEAR HERE >

## *Discussion*

The aim of this study was to determine whether intermittent administration of local VT modulates blood flow and oxygenation to the FCU muscle and attenuates the strength loss associated with EIMD compared to no VT. The main findings were that application of intermittent local VT after a muscle damage-inducing exercise protocol was more effective at

277 maintaining strength of the wrist flexors than no VT, and that FCU $SmO_2$ re-saturation was
278 greater with local VT compared to no VT.

279

280 The aetiology of EIMD is multifactorial with many seeking to find the underlying factors that
281 contribute to this syndrome.[25] Strength reduction is considered one of the most valid markers
282 of EIMD,[26] with many studies demonstrating strength losses following EIMD.[2] Within the
283 current study, strength reduction observed within the wrist flexor muscle group was
284 significantly reduced from baseline at 1, 24 and 48 hr post EIMD protocol for the control
285 group only, which suggests the muscle damaging protocol that was administered was
286 appropriate. Interestingly, however, this trend was not observed in the experimental group
287 who underwent local VT. The magnitude of strength reduction ($\eta^2$ = 0.352) observed within
288 the control group is in line with previous research investigating the effects of local VT on
289 muscle strength post damage, which also reported moderate effect sizes (d= 0.44) in strength
290 within VT group versus control groups.[10]

291 One plausible explanation as to why muscle strength was maintained in the local VT group is
292 due to the improved re-saturation rate of $SmO_2$ observed within the VT compared to the
293 control group. Previous research by Moraleda et al[34] measured the positive effects of VT on
294 EIMD, concluding a possible cause was the beneficial effect on $SmO_2$ (d= 0.96). In that
295 research, $SmO_2$ data was obtained using NIRS in real time during rest and activity without
296 occlusion. An even larger effect on $SmO_2$ was also observed within the current study (d= 1.7-
297 3.1), except via occlusion, which is used to calculate rate of change of $SmO_2$ thus providing
298 better insight into oxygen kinetics and blood flow.

Maximal muscle contraction relies on the continuous utilisation 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 (PCr) to adenosine diphosphate (ADP), previous research clearly shows that this energy system is 'reloaded' by oxidative means and is therefore heavily dependent on the availability of oxygen.[27,28] In addition to this, any breakdown in the transport of oxygen, such as damage to the peripheral local muscle microcirculation would compromise muscle function.[29] Relative increase in the re-saturation rate of $SmO_2$ would suggest greater oxygen delivery and therefore blood flow to the muscle.[22,30] In the current study, $SmO_2$ increased by 78% from baseline to 48 hrs post EIMD following administration of local VT, compared to only a 31% increase in the control group. Assuming $SmO_2$ is an appropriate surrogate for blood flow, the results of the current study may suggest the VT group experienced increased blood flow to their damaged wrist flexors. Although blood flow was not measured directly in the current study, previous investigations have demonstrated that increased flow to damaged muscles following EIMD reduces the level of muscle damage biomarkers, such as creatine kinase, more rapidly than when no significant increase in blood flow is observed.[30] Increased blood flow and reduced levels of damage biomarkers also correlate with improved recovery from EIMD[30] and would explain the aforementioned maintenance of muscle strength in the VT group compared to the reduction in strength observed in the control group following EIMD.

According to their local inflammation theory, Gulick and Kimura[31] suggest that increased permeability of local vasculature after eccentric muscle damage leads to an efflux of metabolites and oedema formation within the damaged muscle. Subsequently, a cascade of

events occur that leads to increased neutrophil release, macrophage formation and breakdown of muscle tissue. Furthermore, Egners et al.[32] explained that oedema formation compromises muscle perfusion and contributes to local hypoxia, compounding muscle damage. Considering $SmO_2$ was increased at 48 hr post-EIMD in the VT group only within the current study, it is reasonable to assume that VT attenuated the inflammatory cascade, extent of neutrophil margination and local hypoxia following EIMD. Local VT is known to increase the internal diameter of the vasculature serving damaged muscles,[12] resulting in transient increases in the relative blood flow and ultimately enhanced oxygen delivery to this area.[25] Acute changes in $SmO_2$ are reflective of the dynamic local vascular tone, which controls blood flow, oxygenation and perfusion rates within 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 hr post-EIMD in the VT group only.

The timing and dosage of VT application appears to be significant and should not be overlooked. Previous investigation demonstrated that administering a single bout of VT pre-EIMD was ineffective at maintaining muscle strength when assessed at 24, 48 and 72 hours post-EIMD.[33] More recently, Moraleda et al.[34] demonstrated that a single bout of local VT administered as late as 48 hrs post EIMD was sufficient to improve $SmO_2$ above baseline (~12%), albeit to a lesser extent than that observed in the current study. 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 seen within the current 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 would explain the higher $SmO_2$ observed within this study, 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 as these diminish when no reapplication occurs.[35] Thus, providing evidence that multiple bouts of VT may be more effective than single bouts for improving $SmO_2$ following EIMD, but further research is still required to ascertain optimal windows of application, and identify the mechanisms underpinning a potential summative effective of the therapy.

An alternative explanation for the results in the current study is the facilitation of 'functional hyperaemia', a recognised reaction whereby an increase in local muscle metabolism initiates compensatory vasodilation.[25] The normal inflammatory process seen in EIMD is well documented to hinder local blood flow[25,26] and lead to unfavourable leakage of intramuscular cell contents, ultimately inhibiting normal muscular contraction and causing loss of strength.[26] The higher $SmO_2$ observed following EIMD in the VT group of the current study suggest that local VT enhances vasomotor response, increasing local muscle oxygen level and reverses some of these inflammatory processes post-EIMD.[4] Kerschan-Schindl et al.[12] reported that whole body VT post EIMD enhanced vasodilation of small arterioles and capillaries. With a more intense vibration likely to be experienced by the target muscles than that from whole body VT,[8] local VT may induce local reactive vasodilation to a greater extent than that observed following whole-body VT previously. It should be noted that no biomarkers of muscle damage or inflammation were measured in the current study so this explanation remains speculative and would benefit from further investigation. Nonetheless,

368 repeated applications of local VT are preferable to whole body VT when attempting to limit
369 the extent of EIMD following unaccustomed eccentric exercise.

370 ***Limitations***

371 The current study controlled blood flow through the use of a single cuff on the upper arm.
372 While this worked well in creating arteriole occlusion, a more appropriate method would
373 have been to place a second cuff on the wrist to occlude the venous circulation. Without this
374 second occlusion point, it must be assumed that some blood moved out of the compartment
375 into the venous system, potentially affecting the NIRS data and subsequent inferences
376 relating to muscle metabolism. However, there is no data pertaining to the size of this effect.

377 In comparison to other studies, the current sample size is smaller.[7] This is the first study to
378 show that VT improves the effects of EIMD and blood oxygenation. While the effects of this
379 study are positive, we accept that these findings are within a relatively small sample size and
380 within a relatively small muscle group. A post hoc G*Power analysis was performed, and the
381 effect was found to be strong enough to avoid a type I error (N = 6). Further to this, the
382 statistical analysis used is non-parametric which helps to ensure that the error rate is nullified
383 as much as possible, although the results should therefore be interpreted with caution. Future
384 research involving additional muscle groups is required to ensure the positive effects
385 observed here are observed in larger muscle groups that more commonly exhibit EIMD, such
386 as the quadriceps.

387 A future consideration would be to include subjective pain scoring to assess effectiveness of
388 treatment for EIMD as this has been included in other studies to evaluate efficacy of
389 interventions.[11] The authors appreciate that pain is often used as a proxy marker for
390 'recovery' from EIMD within similar research. Nonetheless, previous research specifically

391 assessing measurement tools used within EIMD studies, such as muscle torque, range of
392 motion and histological changes, argue that subjective 'soreness' scores correlated poorly
393 with actual muscle function and therefore subsequent damage from eccentric loading.[36,37]
394 Strength has been shown to be a more reliable marker (i.e. intraclass correlation coefficients
395 $\geq 0.85$) to measure muscle function and resultant recovery post EIMD.[36] Further to this, peak
396 loss of muscle function due to EIMD reportedly occurs within the first 24-48 hours, whereas
397 the time course for peak soreness occurs later between 48-72 hours.[36] This effect was
398 observed within the Moraleda et al[34] study, which reported 30.2% less pain, reported using
399 VAS scores, 48 hours after EIMD protocol and VT intervention. In line with this research,
400 specifically the 48 hours timeline of the study, the authors feel the objective measure of
401 strength is the best tool for quantifying the effects of EIMD and determining the efficacy of
402 the intervention.

403

404 ***Conclusion***

405 Application of local VT therapy appears to have contributed to attenuating the effects of
406 EIMD on muscle strength and blood oxygenation in wrist flexor muscles. Notably, we
407 believe this study is the first to show that VT contributes to alleviating some EIMD
408 symptoms when administered multiple times post EIMD, which could be due to a summative
409 effect over time. Including local VT as part of post-exercise recovery strategies for smaller
410 muscle groups could be beneficial for rehabilitation and athletic training purposes, although
411 more work is warranted in this area to substantiate the current findings and apply them to
412 larger muscle groups.

413

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***Declaration of Interests***

The authors have declared that no competing interests exist.

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Table 1. NIRS Derived Data During Atrial Occlusion at Rest and at Time Points Post EIMD in the VT and Control Groups.

| | Resting $SmO_2$ (%) | | Nadir $SmO_2$ (%) | | $SmO_2$ Desaturation Rate (%·min$^{-1}$) | | $SmO_2$ Re-saturation Rate (%·min$^{-1}$) | |
|---|---|---|---|---|---|---|---|---|
| | 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 hrs | 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 hrs | 67.2 ± 5.9 | 62 ± 10.3 | 39.4 ± 4.9 [c] | 43.8 ± 9.3 [c] | 9.3 ± 3.3 | 6.1 ± 3.8 | 14.3 ± 2.1 * [a] | 9.8 ± 3.4 |

Between group differences at each time point given as * P<0.05. Differences to baseline given as [a], differences to 1 hr as [b], differences to 24 hrs given as [c] (P<0.05).

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

Figure 2. Handgrip strength relative to baseline levels in the vibration therapy (VT) group (grey) and control group (black) following exercise induced muscle damage. There are no differences between groups, * $P < 0.05$ compared to baseline in the VT group only.

Figure 3. Resting $SmO_2$ in the vibration therapy (VT) group (grey) and control group (black) following exercise induced muscle damage.

Figure 4. Desaturation rate in the vibration therapy (VT) group (grey) and control group (black) following exercise induced muscle damage.

Figure 5. Re-saturation rates in the vibration therapy (VT) group (grey) and control group (black) following exercise induced muscle damage.