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

4
5 *Abstract*

6 **Context**

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

11 **Objective**

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

14 **Design**

15 Controlled laboratory study

16 **Setting**

17 Laboratory and public gymnasium

18 **Patients or other participants**

19 Ten healthy participants (6 males; 4 females; age: 38 ± 15 yrs; height: 1.72 ± 0.48 m; mass
20 72.0 ± 10.4 kg) were randomized into experimental (VT) and control (CON) groups.

21 **Interventions**

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

28 **Main outcome measure(s)**

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

31 **Results**

32 No difference in grip strength observed pre EIMD, but the VT group demonstrated greater
33 strength at 1 hr ($P=0.004$), 24 hr ($P=0.031$) and 48 hr ($P=0.021$) post EIMD compared to
34 controls. No difference in SmO_2 re-saturation over time ($P>0.05$), but the VT group had a
35 greater re-saturation rate compared to controls at 1 hr ($P=0.007$, $d = 2.6$), 24 hr ($P=0.001$ $d =$
36 3.1) and 48 hr ($P=0.035$, $d = 1.7$) post EIMD.

37 **Conclusions**

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

41

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

44 Abstract Word Count: 298

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46 **Key points:**

- 47 • 10 minutes of intermittent local vibration therapy (45Hz) attenuated the effects of
48 EIMD throughout the duration of the study.
- 49 • Greater muscle oxygen re-saturation rates post EIMD were observed via near infra-
50 red spectroscopy following vibration therapy compared to a control group.
- 51 • Including local intermittent vibration therapy as part of post-exercise recovery
52 strategy for smaller muscle groups could be beneficial for rehabilitation and athletic
53 training purposes.

54

55

56 Exercise induced muscle damage (EIMD) is commonly associated with delayed onset of
57 muscle soreness (DOMS); a phenomenon that results in reductions in joint range of motion
58 (ROM)¹ muscular power and force generation² and increased inflammation.³ Evidence from
59 previous research suggests that eccentric muscle contraction causes a greater level of EIMD
60 symptoms than concentric contraction, by negatively impacting local and systemic
61 haemodynamic and macro and micro-vascular morphology.³ Consequently, EIMD following
62 eccentric exercise typically compromises the supply of oxygenated blood to active muscles
63 for between 24-72 hrs.⁴ In terms of athletic performance, the primary symptom of EIMD is
64 the impairment of muscle function and strength, hereby defined as reduced capacity of
65 muscle force production. Findings from previous studies, in which the researchers induced
66 local muscle ischemia, suggest that the initial reduction in strength in the working skeletal
67 muscle occurs due to reduced oxygen availability.⁵ Thus, it would be advantageous for
68 individuals who experience EIMD to reduce these negative effects on performance, which
69 may be achieved by increasing the availability of oxygen within the muscle.

70

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

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

83

84 Vibration therapy (VT) is another alternative technique known to improve muscle blood flow
85 and oxygenation.⁷ VT is administered to either the whole-body, typically via plates through
86 closed-chain positions (i.e. hands or feet on the plate), or locally, where a device applies VT
87 directly to a specific region of the body.⁸ Irrespective of mode, VT is accessible and can be
88 administered consistently at varying intensities according to individual comfort. VT is
89 already used in athletic rehabilitation and sports performance settings to enhance strength,⁹
90 manage recovery from injury¹⁰ and increase joint range of motion.¹¹ Importantly, both whole
91 body¹² and local VT⁸ have been shown to alleviate the effects of EIMD when administered
92 before and after EIMD protocols. Whilst there are no direct comparisons between the two
93 modes, it has been suggested that the size of the vibration reaching the target tissue from
94 whole-body VT is most likely reduced compared to local VT, due to signal dissipation into
95 the surrounding, non-affected, tissues.⁸ Additionally, whole body VT, is usually limited to
96 large commercial gyms, with local VT more accessible due to relatively lower cost and high
97 portability. Furthermore, Games et al.¹³ concluded that application of local VT, which occurs
98 on unloaded body segments, might be more effective than whole-body VT, which is applied
99 to loaded body segments. Unloaded muscles are relaxed, thus the small blood vessels
100 supplying these muscles are not subject to the same levels of pressure from the surrounding
101 muscle tissue otherwise observed during contraction.¹⁴ Consequently, with less vascular

102 compression, improved blood flow through the muscle microvasculature could be expected,
103 however this hypothesis is speculative as there appears to be no direct assessment of muscle
104 blood flow using local VT following EIMD.

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

109 *Methods*

110 *Participants*

111 Ten participants (mean \pm SD; male N = 6; female N = 4; age 38 ± 15 yrs; height 1.72 ± 0.48 m;
112 mass 72.0 ± 10.4 kg) with no previous or current upper body musculoskeletal conditions,
113 described themselves as healthy and without previous experience of resistance training that
114 specifically targeted the arms¹⁵ were recruited for the study. Inclusion required that all
115 participants had no prior history of smoking, which is known to impair peripheral blood
116 flow,¹⁶ or had not been using anti-inflammatory medication which has been shown to reduce
117 the effects of EIMD.¹⁷ Participants were randomly allocated to a treatment group (VT, N = 5)
118 or control group (i.e., no VT, N = 5). All participants gave written consent, ethical approval
119 was granted by the local ethics committee of Manchester Metropolitan University and all
120 procedures complied with the Declaration of Helsinki.

121

122 *Experimental Procedure*

123 All participants were required to attend four testing sessions, at baseline, and 1 hr, 24 hr and
124 48 hr post EIMD protocol. Anthropometric assessment of height and mass and administration
125 of an EIMD protocol were conducted during the baseline session only, whilst muscle oxygen
126 saturation, wrist flexor strength and all exercise protocols were conducted at each session.
127 Participants were advised to avoid vigorous exercise for 48 hrs prior to and throughout the
128 study duration.

129

130 *Muscle Oxygen Measures*

131 Following arrival at the laboratory for baseline data collection, participants assumed a supine
132 position for the assessment of blood pressure, taken from the right arm. Following a 10 min
133 rest period, to allow blood flow to return to normal,¹⁸ muscle oxygen saturation (SmO₂) of
134 the flexor carpi ulnaris (FCU) was measured using portable near infra-red spectroscopy
135 (NIRS) sensor (MOXY monitor© Fortiori design LLC, Hutchinson, Minnesota 55350). The
136 NIRS sensor was placed on the skin of the wrist flexors, midway between the styloid process
137 of the wrist and the superior radio-ulnar joint process, using adhesive dressings. A light shield
138 was also placed over the monitor to prevent ambient light pollution.¹⁹ The NIRS sensor
139 placement was identified with a permanent marker to ensure the reliability of sensor
140 placement on subsequent testing days, particularly as there is known heterogeneity of blood
141 flow and oxygen utilisation (9-13%) within a muscle.²⁰ With participants remaining supine,
142 SmO₂ was recorded for 5 mins, with resting SmO₂ determined as the peak value recorded
143 during this period once stability was achieved (no greater than 3-5% fluctuation in 30
144 seconds⁴). Subsequently, occlusion of the brachial artery was undertaken using a manual
145 sphygmomanometer cuff placed approximately 2-3 cm above the antecubital fold. In line

146 with previous research, pressure in the sphygmomanometer cuff was quickly inflated (<3
147 seconds) to a supra-systolic level of 30 mmHg above the baseline systolic blood pressure
148 (150-180 mmHg) to ensure cessation of blood flow in the brachial artery.⁵ The occlusion was
149 maintained for 3 min and immediately released, desaturation and re-saturation rates were then
150 measured to express the rate of change (kinetics) of muscle oxygen saturation. During
151 occlusion, SmO₂ was continuously recorded for 3 mins, with the lowest value obtained
152 determined as the nadir. The absolute difference between peak resting SmO₂ and nadir SmO₂
153 values was then used to calculate the rate of desaturation (%·min⁻¹) as: (peak SmO₂ – nadir
154 SmO₂)/3. Following deflation of the arm cuff, SmO₂ ‘recovery’ was measured for 3 min, with
155 the SmO₂ at 3 min recorded and used to calculate rate of re-saturation (%·min⁻¹) as: (recovery
156 SmO₂ – nadir SmO₂)/ 3. Data was collected in real time by Bluetooth transmission between
157 the NIRS device and a separate computer via an ANT+ sensor (Garmin Ltd ©, Schaffhausen,
158 Switzerland). The data was processed through Peripedal© computer software and saved in
159 .csv format. NIRS has previously been validated as an accurate device for measuring forearm
160 blood flow and muscle oxygenation against magnetic resonance spectroscopy (r = 0.965)²¹
161 and strain-gauge plethysmography.²² Measures of muscle oxygen saturation were repeated at
162 1, 24 and 48 hours post damage inducing exercise protocol.

163

164 *Strength Measures*

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

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

174

175

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

177 In order to determine the exercise load to be used for the muscle damaging protocol,
178 participants completed an assessment of their one repetition maximum (1RM) for the wrist
179 flexors. Initially, participants were seated with their elbow flexed at 90° to the upper arm and
180 forearm resting on the plinth of a bicep curl machine. In order to isolate control of the
181 movement to the wrist flexors, the distal part of the limb (wrist to fingers) was not supported
182 by the plinth. Following a series of warm-up contractions, participants self-selected a starting
183 dumbbell weight to commence the assessment of 1RM, which was passed to the participant
184 when they were in the prescribed starting position, i.e., with the wrist and forearm parallel
185 (neutral alignment) and rested supine on the table (Figure 1A). Initially, the dumbbell was
186 lowered over 3 s to the end range of motion of wrist extension (Figure 1B), before being
187 returned to the starting position over 1s whilst maintaining the supinated arm position, in line
188 with previous protocols.⁵ Participants completed one repetition of each weight and, if
189 successful, this was increased by 1 kg and the procedure was repeated following a 2 min rest.
190 1RM was identified as the final load completed without failing to return the dumbbell to the
191 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, Shenzhen, 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

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

221

222 *Data analysis*

223 Statistical analysis was performed using SPSS (IBM SPSS statistics for Mac, version 25.
224 Armonk, NY: IBM corp). Wrist flexor strength and SmO₂ values were tested for normality
225 (Shapiro-Wilk), equal variance (Levene's) and sphericity (Mauchly's) before being tested for
226 effects using a 2x4 (group x time) mixed measures ANOVA. Bonferroni adjusted post-hoc
227 pairwise comparisons were completed on significant main effects. Alpha was set at $P < 0.05$
228 and all data are presented as mean \pm standard deviation. Effect sizes for pairwise comparisons
229 were calculated using Cohen's d to determine the magnitude of the difference between
230 groups and were classified as: <0.2 low, 0.21-0.5 medium, 0.51-0.8 large and >0.81 very
231 large. In addition, partial eta squared (η^2) was used to show the magnitude of the effect
232 between each condition and classified as 0.01 (small), 0.09 (medium) and 0.25 (large).

233

234 *Results*

235 *Wrist Flexor Strength*

236 There was a significant effect of time ($F_{(3,24)} = 7.414$, $P = 0.001$, $\eta^2 = 0.481$) and group*time
237 interaction for strength ($F_{(3,24)} = 4.338$, $P = 0.014$, $\eta^2 = 0.352$). There was no change in
238 strength of the VT group over time ($P > 0.005$) whereas strength of the control group was
239 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
240 (5%, $P = 0.035$, $d = 1.06$) compared to baseline. There were no other strength differences
241 between time points ($P > 0.05$, Figure 2).

242

243 < INSERT FIGURE 2 NEAR HERE >

244

245 ***SmO₂***

246 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
247 $= 0.578$, $\eta^2 = 0.040$, Table 1) for resting SmO_2 (Figure 3). Nadir SmO_2 did not differ
248 significantly between groups ($F_{(1,8)} = 2.495$, $P = 0.153$, $\eta^2 = 0.238$) or over time when
249 compared to baseline ($F_{(3,24)} = 1.$, $P = 0.225$, $\eta^2 = 0.163$, Table 1). There was, however, a
250 group*time interaction ($F_{(3,24)} = 8.359$, $P = 0.001$, $\eta^2 = 0.511$). Post hoc analyses revealed
251 that nadir SmO_2 was lower in the VT group compared to the control group at 1 hr post EIMD
252 only ($P = 0.027$, Table 1).

253 < INSERT FIGURE 3 NEAR HERE >

254 < INSERT TABLE 1 NEAR HERE >

255

256 There was a significant main effect in the rate of SmO₂ desaturation post EIMD protocol
257 ($F_{(3,24)} = 3.030$, $P = 0.049$, $\eta^2 = 0.275$), but this did not differ between groups ($F_{(1,8)} = 2.906$,
258 $P = 0.127$, $\eta^2 = 0.266$) (Figure 4). Rate of desaturation was faster at 24 hrs than at baseline (P
259 $= 0.037$) while no other differences existed ($P > 0.05$, Table 1).

260 < INSERT FIGURE 4 NEAR HERE >

261 There was a main effect of time on SmO₂ re-saturation rate ($F_{(3,24)} = 4.339$, $P = 0.014$, $\eta^2 =$
262 0.352) and the VT group had a significantly greater re-saturation rate compared to the
263 controls ($F_{(1,8)} = 10.35$, $P = 0.012$, $\eta^2 = 0.564$) (Figure 5). There was no difference between
264 groups for rate of SmO₂ re-saturation at baseline ($P = 0.611$), but rate of SmO₂ re-saturation
265 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
266 EIMD was higher in the VT group compared to controls (Table 1). For the VT group, re-
267 saturation of SmO₂ was greater at 1 ($P = 0.04$, $d = 1.5$), 24 ($P = 0.001$, $d = 2.0$) and 48 hrs (P
268 $= 0.018$, $d = 1.0$) compared to baseline, whereas there was no difference in re-saturation rate
269 for controls between any baseline and any time point ($P > 0.05$, Table 1).

270 < INSERT FIGURE 5 NEAR HERE >

271

272 *Discussion*

273 The aim of this study was to determine whether intermittent administration of local VT
274 modulates blood flow and oxygenation to the FCU muscle and attenuates the strength loss
275 associated with EIMD compared to no VT. The main findings were that application of
276 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 SmO₂ 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 SmO₂ observed within the VT compared to the
293 control group. Previous research by Moraleda et al³⁴ measured the positive effects of VT on
294 EIMD, concluding a possible cause was the beneficial effect on SmO₂ ($d = 0.96$). In that
295 research, SmO₂ data was obtained using NIRS in real time during rest and activity without
296 occlusion. An even larger effect on SmO₂ 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 SmO₂ thus providing
298 better insight into oxygen kinetics and blood flow.

299 Maximal muscle contraction relies on the continuous utilisation of energy stores, which in
300 turn relies on the adequate delivery of oxygen. Even during anaerobic bouts, when energy is
301 derived from the transfer of phosphate from phosphocreatine (PCr) to adenosine diphosphate
302 (ADP), previous research clearly shows that this energy system is ‘reloaded’ by oxidative
303 means and is therefore heavily dependent on the availability of oxygen.^{27,28} In addition to
304 this, any breakdown in the transport of oxygen, such as damage to the peripheral local muscle
305 microcirculation would compromise muscle function.²⁹ Relative increase in the re-saturation
306 rate of SmO₂ would suggest greater oxygen delivery and therefore blood flow to the
307 muscle.^{22,30} In the current study, SmO₂ increased by 78% from baseline to 48 hrs post EIMD
308 following administration of local VT, compared to only a 31% increase in the control group.
309 Assuming SmO₂ is an appropriate surrogate for blood flow, the results of the current study
310 may suggest the VT group experienced increased blood flow to their damaged wrist flexors.
311 Although blood flow was not measured directly in the current study, previous investigations
312 have demonstrated that increased flow to damaged muscles following EIMD reduces the
313 level of muscle damage biomarkers, such as creatine kinase, more rapidly than when no
314 significant increase in blood flow is observed.³⁰ Increased blood flow and reduced levels of
315 damage biomarkers also correlate with improved recovery from EIMD³⁰ and would explain
316 the aforementioned maintenance of muscle strength in the VT group compared to the
317 reduction in strength observed in the control group following EIMD.

318

319 According to their local inflammation theory, Gulick and Kimura³¹ suggest that increased
320 permeability of local vasculature after eccentric muscle damage leads to an efflux of
321 metabolites and oedema formation within the damaged muscle. Subsequently, a cascade of

322 events occur that leads to increased neutrophil release, macrophage formation and breakdown
323 of muscle tissue. Furthermore, Egners et al.³² explained that oedema formation compromises
324 muscle perfusion and contributes to local hypoxia, compounding muscle damage.
325 Considering SmO₂ was increased at 48 hr post-EIMD in the VT group only within the current
326 study, it is reasonable to assume that VT attenuated the inflammatory cascade, extent of
327 neutrophil margination and local hypoxia following EIMD. Local VT is known to increase
328 the internal diameter of the vasculature serving damaged muscles,¹² resulting in transient
329 increases in the relative blood flow and ultimately enhanced oxygen delivery to this area.²⁵
330 Acute changes in SmO₂ are reflective of the dynamic local vascular tone, which controls
331 blood flow, oxygenation and perfusion rates within the muscle tissue.²² Increases in relative
332 SmO₂ would be expected with greater blood flow, thus, providing an explanation for the
333 attenuation of the inflammatory cascade and higher SmO₂ 48 hr post-EIMD in the VT group
334 only.

335

336 The timing and dosage of VT application appears to be significant and should not be
337 overlooked. Previous investigation demonstrated that administering a single bout of VT pre-
338 EIMD was ineffective at maintaining muscle strength when assessed at 24, 48 and 72 hours
339 post-EIMD.³³ More recently, Moraleda et al.³⁴ demonstrated that a single bout of local VT
340 administered as late as 48 hrs post EIMD was sufficient to improve SmO₂ above baseline
341 (~12%), albeit to a lesser extent than that observed in the current study. Dissimilar to the
342 current study, a single application of local VT 48-hours post-EIMD was not able to maintain
343 muscle strength.³⁴ Repeated bouts of VT, as seen within the current study may incrementally
344 improve the local vascular tone to create a 'summative' benefit over time. Such a

345 summation would contribute to enhanced blood flow and would explain the higher SmO₂
346 observed within this study, although this needs to be shown empirically. It is possible,
347 therefore, that the acute benefits of a single application of local VT are not sufficient to
348 attenuate the symptoms of muscle damage as these diminish when no reapplication occurs.³⁵
349 Thus, providing evidence that multiple bouts of VT may be more effective than single bouts
350 for improving SmO₂ following EIMD, but further research is still required to ascertain
351 optimal windows of application, and identify the mechanisms underpinning a potential
352 summative effective of the therapy.

353

354 An alternative explanation for the results in the current study is the facilitation of ‘functional
355 hyperaemia’, a recognised reaction whereby an increase in local muscle metabolism initiates
356 compensatory vasodilation.²⁵ The normal inflammatory process seen in EIMD is well
357 documented to hinder local blood flow^{25,26} and lead to unfavourable leakage of intramuscular
358 cell contents, ultimately inhibiting normal muscular contraction and causing loss of
359 strength.²⁶ The higher SmO₂ observed following EIMD in the VT group of the current study
360 suggest that local VT enhances vasomotor response, increasing local muscle oxygen level
361 and reverses some of these inflammatory processes post-EIMD.⁴ Kerschman-Schindl et al.¹²
362 reported that whole body VT post EIMD enhanced vasodilation of small arterioles and
363 capillaries. With a more intense vibration likely to be experienced by the target muscles than
364 that from whole body VT,⁸ local VT may induce local reactive vasodilation to a greater
365 extent than that observed following whole-body VT previously. It should be noted that no
366 biomarkers of muscle damage or inflammation were measured in the current study so this
367 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 ≥ 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 al³⁴ 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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417

418 ***Declaration of Interests***

419 The authors have declared that no competing interests exist.

420

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423

424 **References**

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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).

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

529

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

534

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

537

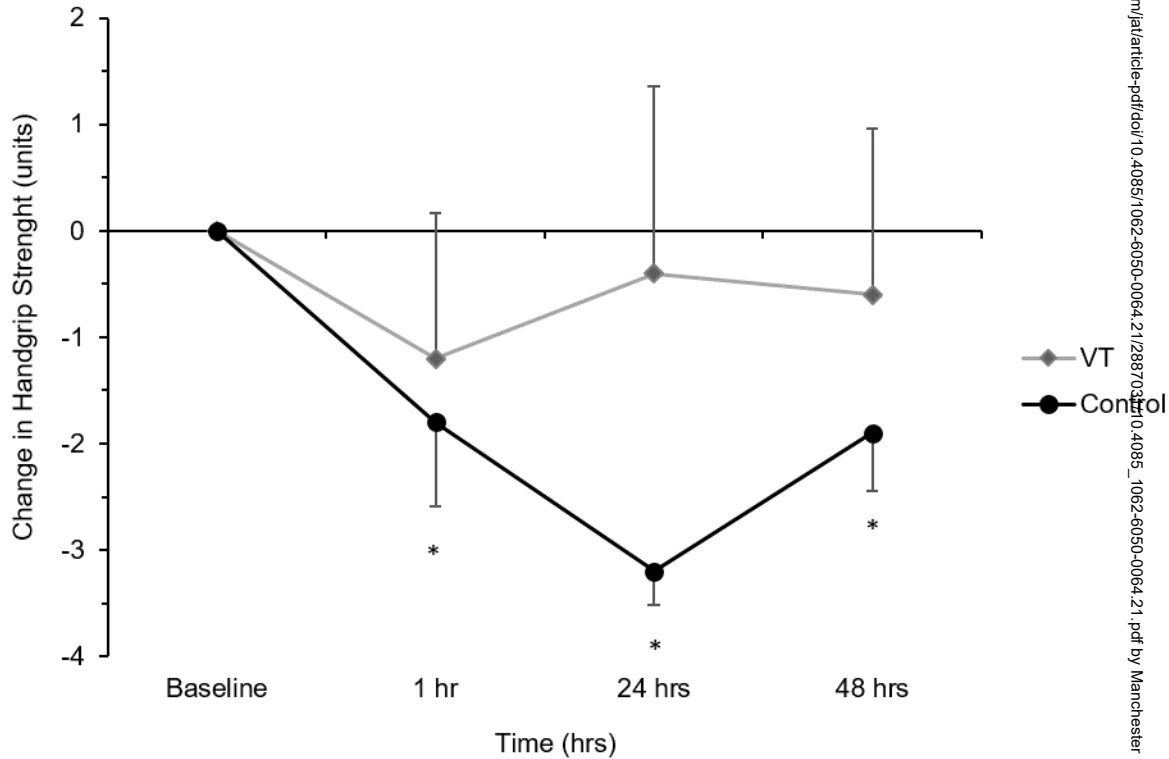
538 Figure 4. Desaturation rate in the vibration therapy (VT) group (grey) and control
539 group (black) following exercise induced muscle damage.

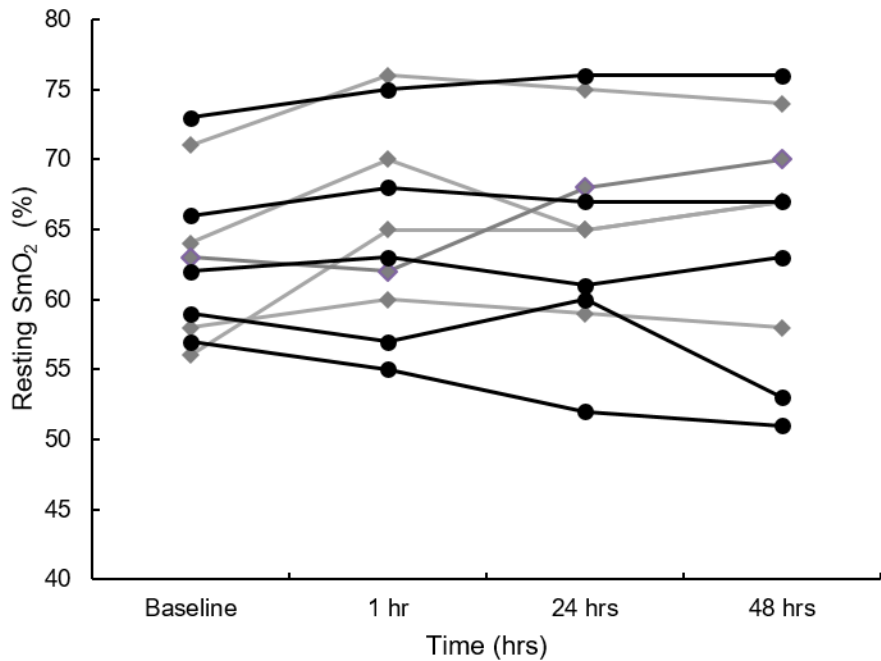
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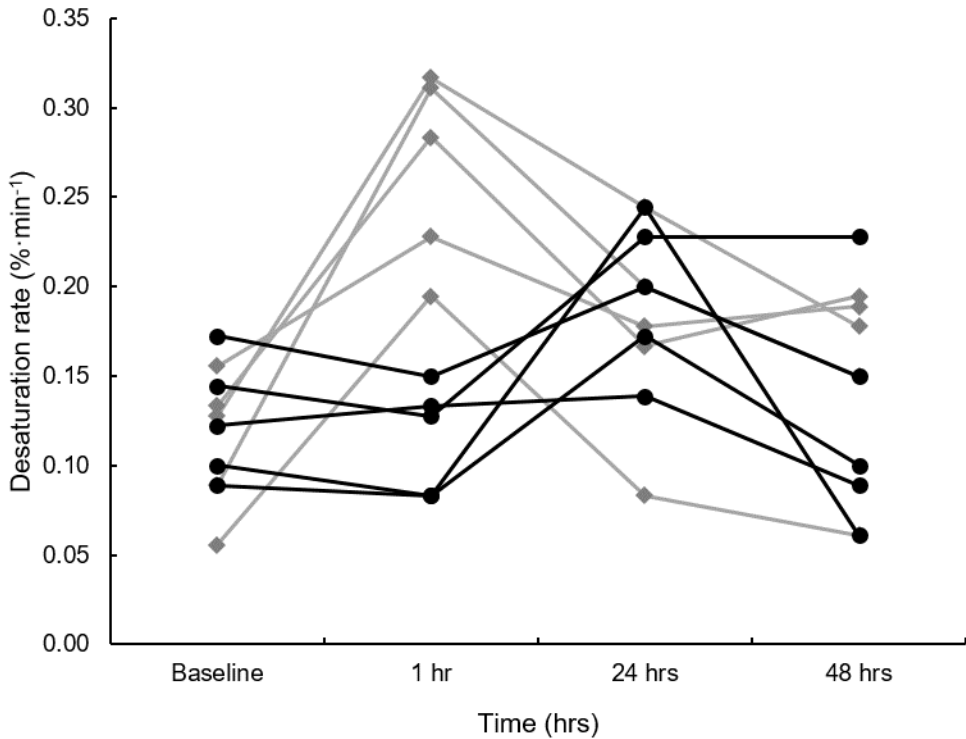
541 Figure 5. Re-saturation rates in the vibration therapy (VT) group (grey) and control
542 group (black) following exercise induced muscle damage.

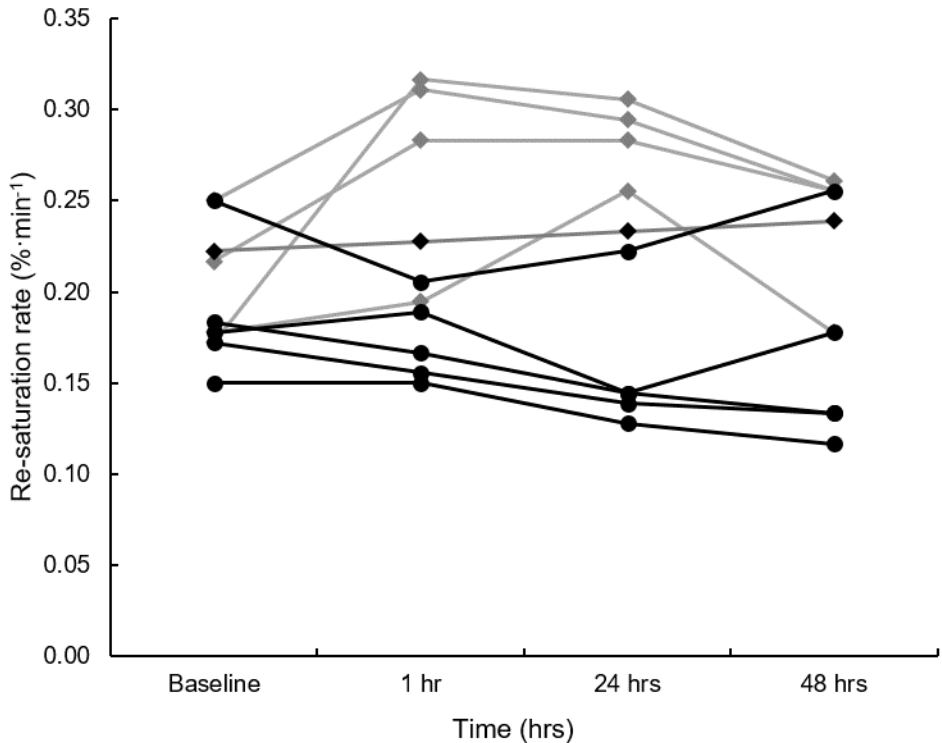
A











—◆— VT
—●— control