Effect of local vibration therapy on blood flow and muscle oxygenation following exercise induced muscle damage

Abstract

Purpose: Exercise induced muscle damage (EIMD) is a common condition that reduces muscular function and consequently athletic performance. Previous research suggests deleterious changes in peripheral microvasculature could be a major contributing factor. Local vibration therapy (VT) can enhance skeletal muscle blood flow and oxygenation. Using near infrared spectroscopy (NIRS), the aim of this study was to investigate whether direct application of VT to the extensor Carpi Ulnaris muscle post EIMD could attenuate these negative effects and enhance muscle oxygenation and performance.

Methods: Ten healthy participants (male n=7, female n=3; age=38 ± 15yrs; height 1.72 ± 0.48m; weight 72 ± 10.4kg) were randomly assigned to control (CON) and experimental (EXP) groups, with further random assignment of arm usage for each participant. Baseline muscle strength performance was measured using a handgrip dynamometer. Arterial occlusions (AO) were applied and baseline NIRS data was collected to ascertain resting muscle oxygenation levels, desaturation and re-saturation rates. Each participant then performed an exercise protocol of 10 X 10 wrist extensions at 70% MVC to induce EIMD. Further NIRS and handgrip data was collected at 60mins, 24 and 48 hours post EIMD. EXP Group was prescribed ten minutes of VT intervention at 45HZ, applied 60mins post EIMD and every eight hours thereafter, with CON group receiving no VT.

Results: A two way mixed ANOVA revealed strength reduced significantly over time in both groups (P=0.001). No significant changes were observed in resting SmO_2 over time in either the control or experimental groups (P=0.193). No significance was observed in nadir SmO_2 over time within groups, however nadir SmO_2 reduced significantly in the experimental group at 60 minutes versus control group (P=0.027). During and post arterial occlusion, SmO_2 desaturation and re-saturation oxygen kinetics became significantly faster in the experimental group ($p<0.001$).

Conclusions: The results of this study showed that the application of local VT therapy at a frequency of 45Hz for 10 minutes at intervals of eight hours was successful in attenuating the effects of EIMD. Muscle oxygen re-saturation was considerably

faster and muscle strength did not significantly decline in the EXP group post EIMD. Including local VT as part of a recovery post exercise could be a beneficial strategy for strength and endurance athletes.
Key words: NIRS, Vibration, local vibration, muscle oxygen, EIMD, delayed onset muscle soreness, near infra red spectroscopy, oxygen delivery.

Introduction

There has been extensive research into the phenomenon known as exercise induced muscle damage (EIMD), with the main focus being on the aetiology and its acute negative effects on skeletal muscle and consequently an athletes exercise physiology. Current research objectively reports many negative effects of EIMD, with these ranging from reductions in limb range of motion, inflammation, muscular power output and force generating capacity. Significant evidence also exists from other studies measuring variables of a more subjective nature such as residual muscle soreness and stiffness ranging from 1-5 days, tenderness, and pain (Bryne et al., 2004; Caldwell et al., 2016; Davies et al., 2008, Pournot et al., 2016; Souza et al., 2011). Although the exact aetiology of EIDM is still unknown, most of the research suggests that the underlying mechanisms are due to a prolonged or acute bout of unaccustomed eccentric exercise.

This growing body of EIMD research evidence suggests that the eccentric muscle contraction causes an acute change in local skeletal muscle morphology, which negatively impacts the local and systemic haemodynamics. Consequently the supply of blood and oxygen to the active muscle cells is in some way compromised. Researchers have suggested that eccentric exercise induces 'arterial stiffness' and inflammation, which reduces the diameter of the arteries supplying the affected muscles. This is observed both centrally and locally and alters the normal blood flow and oxygen kinetics of the active skeletal muscle (Caldwell et al., 2016; Barnes et al., 2010). Local changes in the macro and micro-vascular morphology also caused deleterious changes in the flow of blood to the muscle and subsequently normal oxygenation was affected (Davies et al., 2008). Moreover, normal hyperaemic responses post muscle contraction were blunted 1-2 days post EIMD due to a slowed local micro-vascular reactivity, which altered oxygen delivery and utilisation

matching within the muscle (Larsen at al., 2015). In direct contrast to these studies, Ahmadi et al. (2017) reported a significant 'increase' in oxygen delivery 48 hours post EIMD. The authors stated that inflammation in the muscle vasculature was the causal factor for the increased oxygenation; nevertheless, this was still reported as a negative result because performance was reduced in other tasks within the study. It is also worth noting that non-human research report similar findings when measuring muscle blood flow and oxygenation after EIMD protocols. A seminal study involving rats during downhill running also report impaired muscle microcirculatory flow and an imbalance between oxygen delivery and utilisation at the onset of muscle contraction. The underlying mechanism stated in this research was, 'increased capillary distensibilty' (Kano et al., 2005). Thus, if EIMD temporarily alters the delivery of blood and oxygen to the muscle and this leads to impaired performance, it would be advantageous to athletes if these effects could in some way be reduced.

Vibration therapy (VT) is one popular tool used in the rehabilitation and sports performance arenas as part of training programs to attenuate the effects of EIMD (Cheatham et al., 2015). Research methods involving direct local application (Cochrane. 2017), or indirect, whole body VT (Games and Sefton., 2011; Kerschan-Schindl et al., 2001) have been reported as successful. The evidence presented shows increases in performance during strength-based tasks (Bosco et al., 1999; Coza et al., 2011), improved blood flow to the peripheral tissues (Beijer et al., 2014) and greater muscle oxygenation in active skeletal muscles (Ahmadi et al., 2007). Prophylactic usage of VT pre EIMD protocol has also been studied, with researchers claiming it was possible to even prevent the symptoms of EIMD (Legleu et al., 2016; Veqar, 2014). In contrast to these studies, other researchers measured the effects of whole body VT (Cardinale et al. 2007) and local VT (Pournot et al., 2016) on muscle oxygenation but failed to find any significant differences over control groups. Paradoxically, VT has also been shown to have negative harmful effects on the body with acute exposure to very high frequencies or chronic exposure to lower frequencies over time. Authors suggested aetiology was an interrupted blood flow and concomitant acute vasoconstriction, with symptoms manifesting as vasospasms and 'white finger syndrome' (Bovenzi and Griffen. 1997).

Overall there is strong evidence to suggest that EIMD causes disturbances in the normal functioning of peripheral oxygen delivery and utilisation. In addition to this, VT has been shown to increase blood flow and oxygen delivery and potentially attenuate the effects of EIMD. Local vibration has been found to be more effective than whole body due to the effects of dissipation with whole body vibration techniques (Cochrane, D. 2017). To date there has been limited research that measures the effects of local vibration on EIMD, with respect to non-invasive measures of muscles oxygenation (NIRS), and direct performance of the affected muscle in strength based tasks. The aim of this study, therefore, was to determine if EIMD has deleterious effects on the microvasculature and supply of oxygen to the forearm muscles, which then reduces performance in a strength-based task of handgrip score. Finally, the second aim of the study is to investigate whether VT will attenuate these effects if applied locally to the affected musculature.

Materials and methods

Participants

The Manchester Metropolitan University ethics committee approved the study. Ten participants (male n=7, female n=3, age 38 ± 15years, height 1.72 ± 0.48m, weight 72 ± 10.4kg [mean ± SD]) were recruited to take part in the study. Participants completed a pre assessment questionnaire (PAR-Q) and were determined to be free from known diseases involving pulmonary, cardiovascular, bone, muscles and joints. Only participants with no history of smoking were included as evidence from previous research showed smoking related peripheral arterial disease (PAD) impaired limb blood flow (Walker et al., 2016). No participants had undertaken any structured upper body resistance exercise within the previous 6 months or completed any vigorous exercise 48 hours prior to the assessment. During the course of the study participants were instructed to refrain from drinking alcohol and taking anti-inflammatory medication that might attenuate the inflammatory effects of EIDM. No other exercise beyond what was prescribed by the lead researcher was permitted during the study. Finally, participants were randomly assigned to one of two groups, ‘experimental’ group (EXP) or ‘control’ group (CON). Participants in the

EXP group received EIMD and VT, with the CON group receiving only EIMD. Written informed consent was obtained from each participant prior to commencement of the study.

Experimental Design

Assessments were undertaken in two locations, a laboratory (temperature controlled to 23°C) and a local gymnasium. Participants were further randomly assigned an arm to use for the study to prevent dominance bias. Participants reported to the laboratory on four separate occasions for physiological assessment; baseline (1), 60 minutes-post (2), 24 hours-post (3) and 48 hours-post (4). Height, weight and BMI were recorded at baseline only, with handgrip score, blood pressure and muscle oxygen saturation assessed at all four visits.

The protocol at each laboratory assessment was strictly adhered to. Participants lay supine on a massage bed with their arm relaxed parallel to their trunk. Baseline blood pressure readings were undertaken and the supra-systolic pressure required for arterial occlusion calculated. The cuff was then left in-situ on the upper arm. Next the MOXY monitor © sensor was placed on the skin midway between the wrist and elbow on the wrist flexor muscles (Extensor Carpi Ulnaris) and the area marked by drawing around the device in indelible marker. This was to ensure the 24 and 48 hour readings were also taken from the same area of muscle, as previous research revealed significant heterogeneity of blood flow and oxygen utilisation within a muscle (Vogiatzis et al., 2015). The Moxy © device was then secured in place with a light shield and adhesive dressings to prevent ambient light pollution, which is known to give erroneously high NIRS readings (Kovalenko et al., 2015). Five minutes of resting SmO_2 data was then collected at which point an arterial occlusion (AO) was administered. Pressure in the cuff was quickly inflated (<3seconds) to a supra-systolic level of 30mgHg above baseline to ensure cessation of blood flow in brachial artery. The occlusion was maintained for 3 minutes and then quickly released; a further 2 minutes of MOXY recovery data was collected.

After the baseline data (1) had been collected, both groups underwent a session of resistance exercise in the gymnasium to induce EIMD. The next assessment data was

collected within 60 minutes (2) after finishing the EIMD protocol in the laboratory and repeated at 24 (3) and 48 hours (4) thereafter.

Handgrip

Baseline handgrip scores were recorded for all participants pre EIMD and at each retest (60mins, 24 and 48hours) time point thereafter. A Camry constant digital hand dynamometer was used as this is engineered as a professional training device suitable for handgrip strength testing. In line with previous studies that observed significant differences in handgrip scores based on changing positions (Mogk et al., 2003), participants sat in an upright position with the forearm resting on an adjustable horizontal table. The upper arm was inline with torso and the elbow flexed and resting on the table at 90°. The hand was pronated with the dorsal aspect on the table and in neutral parallel alignment with the forearm. Participants were asked to squeeze the dynamometer and produce their maximum grip force, which was recorded electronically on the device.

EIMD

Participants were seated with the randomly assigned forearm flexed at 90° to the upper arm and resting on the plinth of a bicep curl machine and their hand over the edge. Maximum voluntary contraction (MVC) was determined by lowering a dumbbell using only the hand and wrist extensors. An estimated weight was passed to the participant when they were in the prescribed starting position with the wrist parallel to the forearm in neutral alignment. The dumbbell was then lowered to the furthest comfortable range and then returned back to the starting neutral alignment position. This procedure was repeated until the correct one repetition maximum was determined. After a 10-minute recovery period all participants then underwent an acute bout of novel eccentric exercise to induce EIMD. A protocol of 10 sets of 10 repetitions with 60 seconds recovery between sets was prescribed. Wrist extension using a dumbbell load of 70% MVC was used throughout. During the extension phase participants were instructed to take three seconds to lower the load to the maximal comfortable range and then slowly return to neutral over one to two seconds. This

was in line with previous research involving EIMD using the forearm muscle (Kraemer et al., 2002; Pournot et al., 2016).

Near Infra Red spectroscopy

Muscle oxygen saturation data was collected using a near infrared spectroscopy (NIRS) MOXY monitor blue-tooth wearable sensor. NIRS has previously been validated as an accurate device for measuring forearm blood flow and muscle oxygenation (De Blasi., 1997; Gayda et al., 2014; Van Beekvelt et al., 2001;). MOXY operates on the principle that near infrared light (670 to 810 nm) penetrates human tissue, which has a low optical absorbance. The light signal is then either scattered or absorbed, this occurs in either the myoglobin (Mb) or haemoglobin (Hb) respectively. How much of this infrared light the chromophores of Mb and Hb absorb, depends on whether they are carrying oxygenated (OHB) or deoxygenated blood (HHB). The values obtained from the MOXY monitor represent the relative amounts of OHB and HHB present at that instant in the small capillaries, arterioles and venules of the muscle (Ferrari et al. 2004). This allows us to study the differences in oxygen delivery and muscle oxygen utilisation. Total haemoglobin (Thb) is calculated from the sum of OHB and HHB which enables us to determine a third metric, SmO_2 (muscle oxygenation), calculated as follows, Thb/OHB *100.

SmO_2 reflects the interaction between oxygen supply and oxygen utilisation, therefore the data presented in this study is obtained from the relative changes in SmO_2 at rest and during AO before and after EIMD. A further step was taken to enable us to appreciate oxygen kinetics during AO by calculating the rate of change of SmO_2. Peak nadir was defined as the lowest SmO_2 observed during occlusion. Desaturation was defined as the rate of muscle oxygen consumption, and calculated as the difference in resting SmO_2 and nadir in the three minutes during occlusion. Re-saturation was defined as the rate of muscle oxygen consumption and calculated as the difference in Nadir SmO_2 and recovery SmO_2 at three minutes post occlusion (fig 1).

Data was collected in real time using Peripedal © PC software and saved in CSV format for later Statistical analysis using SPSS IBM software version 25.

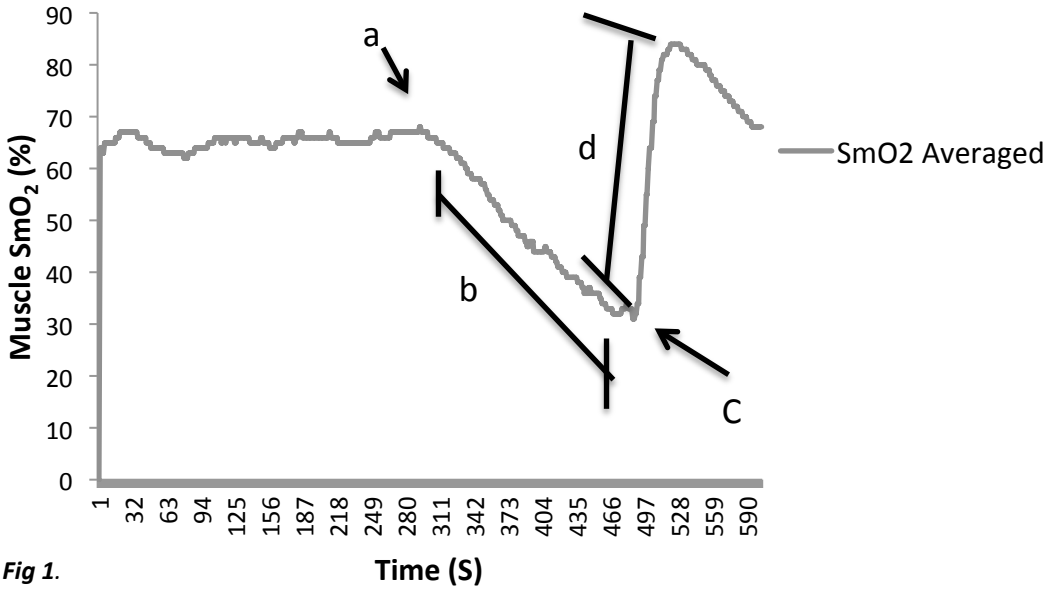

Fig 1.
Example of NIRS SmO_2 data obtained during AO. (a) peak resting SmO_2 (b) desaturation SmO_2 (c) nadir SmO_2 (d) re-saturation SmO_2

Vibration Therapy

VT was administered using a Pulseroll © commercial vibrating foam roller. The roller has four settings (1-4), which allow variable frequencies (Hz) to be administered. VT was self-administered for durations of 10 minutes at 30 minutes, and three further bouts at 8-hour intervals during the 48 hours post EIMD. A demonstration of the correct procedure was given at the first intervention by the lead researcher and VT was self-administered thereafter. The technique involved slowly moving the Pulesroll across the wrist flexor muscles, focusing on the marked area and the muscle belly. Participants were instructed to use setting two, as this supplied VT at a frequency of 45Hz. Previous research suggested that frequencies within the range of 15-50Hz Hz with a duration of greater than five minutes but less than 120minutes was most effective in the treatment and prevention of EIMD (Bovenzi and Griffen. 1997; Cardinale et al. 2007; Games and Sefton., 2011; Kerschan-Schindl et al., 2001; Legleu et al., 2016).

Data analysis

Statistical analysis was performed using SPSS IBM software version 25. The following variables were obtained from the data: Handgrip performance and SmO_2 values for resting, nadir, desaturation and re-saturation SmO_2. Resting SmO_2 was determined

as the peak value recorded during the five-minute rest phase once stability was achieved (no greater than 3-5% fluctuation in 30 seconds (Ahmadi et al., 2007)). Nadir was the lowest SmO_2 value at the end point of occlusion. Desaturation SmO_2 was calculated as the difference between resting SmO_2 and nadir SmO_2 divided by 180 (the total time of occlusion in seconds). Re-saturation was calculated as the difference between nadir and peak SmO_2 during the recovery phase (fig 1).

All output sample data was independently obtained from participants and Levene's tests reported normally distributed data therefore parametricity was assumed. Data was further tested for equality of variance using Mauchlys test of Sphericity (P>0.05) with all data sets displaying homoscedasticity.

A two-way mixed ANOVA was conducted to investigate the effects of local vibration therapy on muscle oxygen saturation (SmO_2) and performance during handgrip assessment pre and post EIMD intervention.

Post-hoc pairwise comparisons were completed on any data sets showing significance to further investigate group's differences using Bonferroni comparisons. Alpha was set at p<0.05, appraisal of the between and within subject's data is now presented (M±SD)

Results

Handgrip summary

Handgrip strength reduced significantly over time within the CON group versus baseline ($F(3,24)=7.414$, $p=0.001$ $\eta^{2=}0.967$). Pairwise comparisons revealed that handgrip strength for the CON group reduced significantly at 60mins (P=0.044), 24 hours (P=0.003) and 48 hours (P=0.035) versus baseline SmO_2 scores. Contrastingly, there was no significant difference between baseline handgrip strength and any time point observed in the EXP group (P =0.172).

Table 1. Handgrip strength at baseline 60 minutes, 24 hours and 48 hours post EIMD in control and experimental groups

	Handgrip	
	EXP	CON
Baseline	45.0 ± 4.56	37.9 ± 12.07
60 minutes	43.8 ± 5.09	36.1 ± 11.26 *
24 hours	44.6 ± 4.50	34.7 ± 10.72 *
48 hours	44.4 ± 4.68	36.0 ± 11.74 *

* Denotes within group significance versus baseline (P<0.05)

Resting SmO_2 summary

There were no significant differences in resting SmO_2 levels over time post EIMD between the EXP and CON groups ($F(3,24)=1.703$, $P=0.193$ $\eta^{2=}0.388$). Similarly there were no significant differences between SmO_2 values at any time point in the EXP ($P=0.053$) or CON ($P=0.053$) groups when compared to baseline.

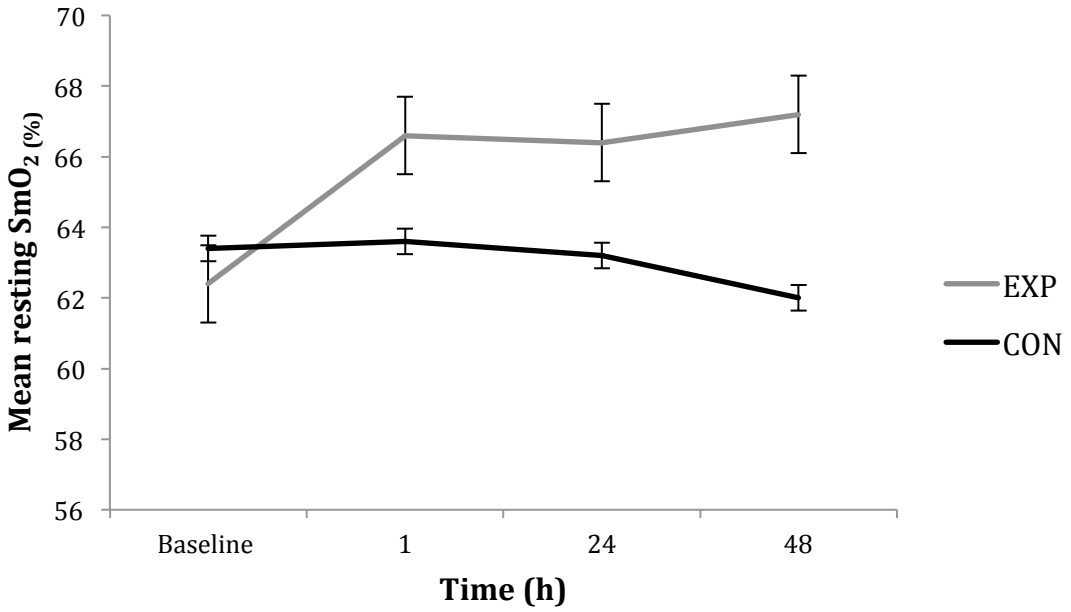

Fig. 2 Effect of EIMD on mean resting Sm02 values for EXP and CON groups

Nadir SmO_2 summary

Nadir SmO_2 values did not significantly differ over time versus baseline in either the EXP or the CON group (F(3,24)=1.560, P=0.225, $\eta^{2=}$0.358). However there was a significant decrease in nadir SmO_2 value observed between groups (F(3,24)=8.359, P=0.001, $\eta^{2=}$0.982). Further pairwise comparisons revealed that SmO_2 nadir was significantly lower in EXP group at the 60-minute assessment than in CON group (P= 0.027).

Desaturation summary

During AO, a faster rate of SmO_2 desaturation post EIMD was observed over time in both the EXP and CON groups when compared to baseline (F(3,24)=52.854, *p*<0.001, $\eta^{2=}$1.000). Additionally there was a significant increase in the rate of $Sm0_2$ desaturation observed between EXP and CON groups (F(3,24)=84.581, *p*<0.001, $\eta^{2=}$1.000). Further pairwise comparisons revealed that SmO_2 desaturation rate during arterial occlusion at 60min post EIMD was significantly faster in the EXP group than in the CON group. No significance was observed across other time points between groups.

Re-saturation summary

No significant difference in SmO_2 re-saturation rate was observed over time in the EXP or CON groups versus baseline (F(3,24)=2.096, *p*=0.127 $\eta^{2=}$0.468). However there was a significant increase observed in the rate of SmO_2 re-saturation between groups, with the rate of SmO_2 re-saturation increasing faster in the EXP group when compared to the CON group (F(3,24)=7.321, *p*<0.001, $\eta^{2=}$0.965). Pairwise comparisons revealed $Sm0_2$ re-saturation rates increasing faster in EXP group than CON group at 60 min (P=0.01), 24 hours (P=0.001) and 48 hours (P=0.37). No significance was observed between EXP and CON baseline scores though (P=0.354)

Table 2.
NIRS derived data at rest and post EIMD during AO in Experimental and Control groups

	Resting SmO_2 (%)		Nadir SmO_2 (%)		Desaturation SmO_2 (%)		Re-Saturation SmO_2 (%)	
	EXP	CON	EXP	CON	EXP	CON	EXP	CON
Baseline	62.4 ± 5.87	63.4 ± 6.35	42.2 ± 3.56	40.8 ± 4.49	0.11 ± 0.04	0.13 ± 0.03	0.20 ± 0.03	0.12 ± 0.12
60	66.6 ± 6.47	63.6 ± 8.17	33.4 ± 3.36 ◆	46.2 ± 8.84 ◆	0.27 ± 0.05◆	0.12 ± 0.03◆	0.27 ± 0.05◆	0.16 ± 0.15◆
24 hours	66.4 ± 5.81	63.2 ± 8.93	35.2 ± 8.76	42.8 ± 5.26	0.17 ± 0.06	0.20 ± 0.04	0.27 ± 0.03◆	0.15 ± 0.17◆
48 hours	67.2 ± 5.89	62 ± 10.30	39.4 ± 4.93	43.8 ± 9.31	0.15 ± 0.05	0.13 ± 0.07	0.23 ± 0.034◆	0.14 ± 0.14◆

◆ Denotes between group significant difference (P<0.05)

Discussion

The purpose of this study was to examine whether EIMD has a deleterious effect on the oxygen kinetics within local muscle vasculature, and if this reduces performance in a strength-based task. Further to this, we investigated whether local VT could attenuate these effects. Primary findings of this study reveal that EIMD altered the muscle SmO_2 dynamics as observed during supra systolic arterial occlusion in the forearm muscles.

Overall these results suggest that eccentric exercise increased the metabolic requirements of the resting muscle, observed by greater oxygen utilisation levels within the muscle. Further to this, VT may have improved the ability of the muscle to utilise oxygen due to an increased delivery. Finally, the strength performance decline normally associated with EIMD was attenuated in the EXP group after receiving VT.

The effects of EIMD and VT on resting, desaturation and re-saturation SmO_2

Findings revealed that muscle resting SmO_2 levels did not significantly change in either group post EIMD. Evidence from previous research, investigating the effects of only EIMD (no VT interventions) protocols on SmO_2 showed similar responses to those described above (fig 2) in the CON group (Ahmadi et al., 2007). Authors observed an acute peak in resting oxygen level immediately post EIMD followed by a significant decline at all further time points. The researchers attributed this initial increase to an acute exercise hyperemia, a recognised reaction whereby an increase in local muscle metabolism initiates a compensatory vasodilation (Joyner et al.,

2015). The declining resting SmO_2 levels at 24 and 48 hours (fig 2), would reflect previous research that concludes EIMD interferes will local blood flow and oxygenation (Souza-Silva et al., 2017). In contrast to this, the VT group resting muscle sm02 post EIDM would not fit this theory, as it remained higher throughout the study (fig 2). A possible explanation for this observed phenomenon might be evidence of a 'functional hyperemia', whereby increased local metabolic activity, in this case induced by EIMD, initiates an enhanced vasomotor response. Local muscle oxygen requirements increase in response to EIMD due to aerobic repair processes that take place within the muscle (Ahmadi et al., 2007). In addition to this, release of inflammatory metabolites could induce a local reactive vasodilation, which then facilitates a greater delivery of oxygen to meet this new higher utilisation (Barnes et al., 2010). Integrity of the local muscle vasculature would be required for this vasodilation to be successful, which is a possible reason why the CON group SmO_2 declines post EIMD. It is worth noting here that the lack of significance between groups is most likely due to the small sample size and high variations within the groups, so future studies with larger cohorts or increased homogeneity would be beneficial.

The current study also examined the effects of EIMD on the rate of oxygen consumption. Application of the supra systolic arterial occlusion allowed further investigation of muscle oxygen kinetics. In theory this allows the cessation of inflow and outflow of blood posterior to the cuff. Once this is achieved it is then possible to observe local muscle oxygen consumption via the NIRS device in terms of changes in relative amounts and rates of oxygen usage. One caveat is that NIRS signals cannot differentiate between whether the oxygen signal is from the myoglobin (Mb) and/or hemoglobin (Hb) chromophores. Throughout this study, and in other similar studies, it assumed that any changes to the SmO_2 value are a consequence of changes in the saturation of the Hb in the blood and thus can be attributed to blood flow. Previous research suggests that up to 80% of the NIRS optical signal is from Hb and not Mb (Hamaoka et al., 2011) but this needs further investigation and should be factored in when considering SmO_2 as a proxy for changes in blood flow.

No significant changes in relative muscle oxygen consumption (as expressed in peak nadir data) were observed over time, however at 60 minutes the EXP group

displayed a significant increase in oxygen consumption versus the CON group. Previous research confirms that the negative effects of EIMD manifest as early as 90minutes post EIMD, with reported reductions in muscle strength, increased soreness and increased plasma proteins within the blood (Davies et al., 2008). Reductions in blood flow were not reported until 48 hours post EIMD in the Barnes et al. (2010) study, although this is due to the fact that the study investigated central pulse wave reactivity within arteries. Arterial stiffening would most likely take longer to develop than the changes to physiology that can be detected in real time with NIRS technology. Davies et al., (2008) did use NIRS to investigate the effects of EIMD on muscle oxygen dynamics but failed to assess within this narrow window of 60-90minutes post EIMD, opting for one more assessment at 48 hours. The slowing of muscle oxygen kinetics that Davies et al., (2008) reported 48 hours post EIMD was attributed to an increased QO_2:VO_2 ratio. The underlying mechanisms attributed to structural and functional changes within the local muscle initiated by a compensatory increase in micro-vascular pressure to preserve oxygen delivery. In simple terms this meant local demand for oxygen outstripped systemic supply, which suggests an impaired delivery system and suboptimal blood flow.

The current study hypothesized that VT could reduce the effects of EIMD, primarily by reducing the damage to local microvasculature and thus enhancing delivery of oxygen to the muscle. The EXP group received their VT intervention 30 minutes post EIMD, so it must be assumed that any findings post EIMD between groups is to be due to this local application of VT. The significant differences observed at 60 minutes post EIMD therefore suggest that VT may already beginning to attenuate the effects of EIMD. In a longer term study over six weeks, Beijer et al. (2015) reported that VT enhanced vasodilation of small arterioles and capillaries, and this would be advantageous to oxygen delivery. Kerschan-Schindl et al. (2001) also reported more acute changes, with increased blood flow immediately after VT. Both of these studies used whole body and not local vibration, although Cochrane, D. (2017) suggests that local VT has a more potent effect as less vibration is dissipated throughout the body. Given that the NIRS signal measures oxygen within the small vasculature of the muscles, this could explain the detection of increased muscle oxygen consumption in the EXP group at 60 minutes. No previous research was

found that examined the effects of VT on EIMD within this short window of time, with previous studies preferring to opt for time frames between 24-72 hours later, when peak effects of EIMD are most commonly reported.

Desaturation rates increased over time in both groups, again with a significantly faster rate seen at 60 min in the EXP group. In this study the rate of desaturation and re-saturation is considered a proxy for blood flow. In the absence of invasive blood testing or more expensive equipment not available, the kinetics of oxygen during AO in this study has been considered as an indirect measure of oxygen supply via blood flow. The negative local vascular changes associated with EIMD have also been shown to manifest within 60 minutes post EIMD (Lau et al., 2011). Local VT, specifically the mechanical motion it generates, has been shown to decrease arterial stiffness (Otsuki et al., 2008) and increase the production of nitric oxide (NO), a potent vasodilator within muscle microvasculature (Maloney-Hinds et al., 2009). The first VT treatment given at 30 minutes could have stimulated NO release and concomitant vasodilation. The further treatments administered throughout the study might have boosted this effect as well as combatting the arterial stiffening previously reported (Barnes et al., 2010). This could account in part for the higher desaturation rate in the EXP group. One reason the significance may not have continued at future time points could be due to the small sample size and high variance. In future, larger cohorts should be recruited as this would assist in identifying relevant differences between the groups. A second reason may be due to Self-administration of local VT. Inevitably this means that participants will apply different pressures and durations of exposure to muscle, which might affect responses in the tissues. Muscles have been shown to display heterogeneity of blood flow (Kalliokoski et al., 2000), so uneven administration of VT could lead to some areas of the muscle not receiving as much therapy and this could lead to lower SmO_2 values. Future studies could involve using a machine to control the pressure of application.

Re-saturation was significantly faster throughout the study post EIMD in the EXP group versus the CON group. Removing the occlusion from the upper arm allows the immediate re-perfusion of the muscle tissue with arterial oxygenated blood during the rebound hyperemia phase. How efficiently this re-perfusion takes place is

dependent on good delivery, so reflective of the local blood flow within the microvasculature. Assuming protocols are adhered to, then any changes in the rate of re-saturation must be attributed to the effects of EIMD and VT. The CON group data shows only the effects of EIMD, therefore it can be assumed that the EXP group shows the effects that VT has on EIMD. At all given time points post EIMD the EXP group showed a faster re-oxygenation of the local tissue. The more retarded re-saturation response observed in the CON group in this study indicates that EIMD has interfered with the normal oxygenation process. Larsen at al. (2015) observed similar blunted hyperaemic responses 1-2 days post EIMD due to a slowed local micro-vascular reactivity, which altered oxygen delivery and utilisation matching within the muscle. In contrast, the VT intervention appears to attenuate these effects, as observed in the response of the EXP group. Overall the dynamics of re-saturation give the best insight into the effects of EIMD on blood flow and oxygenation. Considering the significantly faster re-oxygenation of muscle tissue this would suggest that the VT intervention of the EXP group reduced the deleterious effects of EIMD. One plausible explanation is that the mechanical effects of local VT initiates a reactive vasodilation response, possibly due to the local release of NO or some other vasomotor influencing metabolite. Continued local VT administered 24 to 48 hours post EIMD may reverse some of the negative changes to local vasculature such as arterial stiffening or capillary distensibilty (Kano et al., 2005; Otsuki et al., 2008).

Although the application of a single cuff worked well in respect of completely stopping the flow of blood (and oxygen) to the distal forearm, best practice would be to place a second cuff on the wrist to occlude the venous circulation. Without this second occlusion point it must be assumed that some blood moved out of the compartment into the venous system, thus affecting the resulting NIRS data and inferences relating to muscle metabolism. Nevertheless, EXP and CON groups underwent the same AO protocol, so observed significant differences between groups for NIRS related oxygen kinetics are most likely due to other variables, namely VT.

In accordance with previous research, these findings suggest that EIMD does impair the normal muscle blood flow and oxygenation and there is also an observed increase in the local metabolic requirements.

Muscle strength

This study proposes that the reduced oxygen availability, as a result of EIMD, is the causal factor of the diminished strength performance. Muscle contraction is heavily dependent on oxygen (Pittman et al., 2000) and the muscle microcirculation plays a critical role in delivering this oxygen for diffusion into the cell. If, as the evidence suggests, EIMD induces these negative changes then we would expect significant strength losses in both EXP and CON groups post EIMD. However, the significant loss of handgrip strength observed in the CON group was not observed in the EXP group. This study used 10 minutes of local VT in accordance with a systematic review of the effects of local VT that concluded it could enhance muscle strength, and that single applications lasting 10 minutes appeared most effective (Alghadir et al., 2018). Although this study does not report 'enhanced' strength in the EXP group, there is good evidence that local VT prevented the same performance decline observed in the CON group. Previous research has presented increases in performance during strength-based tasks with VT only interventions (Bosco et al., 1999; Coza et al., 2011). The improved delivery of oxygen in the EXP group, as observed in the re-saturation dynamics described above, is the most likely mechanism for this. Non-invasive measurement of local muscle SmO_2 levels, allows real time appreciation of the interaction between oxygen delivery and oxygen utilisation. 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 (Ferrari et al., 2004). It is worth noting at this point that NIRS does not objectively measure blood flow, so caution is required when assuming an increased oxygenation is a direct result of an increased blood supply within the results of this study. Previous research has included blood sampling alongside NIRS to give a more accurate measure of blood flow within the muscle (Kerschan-Schindl et al., 2001). Comparative studies against the industry standard Doppler and indo-cyanine green have also shown NIRS to be a valid and reliable tool for measures of blood flow and oxygenation (Boushel et al., 2000; louvaris et al., 2016; Themelis et al., 2007). Further research that includes more robust measures of blood flow would therefore add value to the current study.

Not all studies agree with the findings here, Pournot et al. (2016) reported no benefits of local VT attenuating the effects of EIMD, although the study measured 'muscle stiffness' and considering the most likely aetiology is related to local vasculature changes, this type of test might be inappropriate.

Conclusions

Overall the findings of this study support the theory that EIMD disrupts the normal flow of blood and thus oxygen delivery to the muscle. EIMD also reduces performance in strength-based tasks, the effects of which can manifest within 60 minutes and last up to 48 hours later. It could be assumed from the EXP group data that local VT attenuated micro-vascular damage attributed to EIMD, thus improving blood flow and oxygen delivery through increased vasodilation. Local VT appears to also attenuate the acute strength loss associated with EIMD.

Specifically, the authors of this study recommended that an intervention of local VT at a frequency of 45Hz for a duration of 10 minutes post training, and up to two further daily applications as part of a holistic training plan. This could increase the delivery of oxygen to the exercised tissue, assist in the removal of metabolic waste and improve muscle performance.

Future studies involving VT would be beneficial to help further understand the most appropriate interventions for athletes and the possible underlying mechanisms.

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