# Human Physiological and Biomechanical Responses to Vibration Exercise

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A thesis submitted in partial fulfilment of the requirements of the University of Greenwich for the Degree of Doctor of Philosophy

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# DECLARATION

I certify that this work has not been accepted in substance for any degree, and is not concurrently being submitted for any degree other than that of Doctor of Philosophy being studied at the University of Greenwich. I also declare that this work is the result of my own investigations except where otherwise identified by references and that I have not plagiarised the work of others

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#### ABSTRACT

The role of vibration in exercise is controversial, with much debate about its potential benefits. The aim of the research reported in this PhD thesis was to inform evidence based practice by investigating the underlying responses of the human body during exercise with vibration. Human neuromuscular and cardiovascular systems were investigated using 3D motion analysis, near infra-red spectroscopy (NIRS), laser Doppler blood flow analysis and electromyography (EMG).

Analysis of a prototype vibrating stationary cycle identified significant increases in muscle activation. However, the validity of the results was limited by a confounding issue of increasing resistance with increasing cadence due to the cycle's vibration mechanism.

Consistency of exercise performance on vibration platforms was measured by 3D analysis; vibration did not affect the kinematic parameters of exercises such as heel raises or press ups, even though significant physiological changes occurred. NIRS indicated a significant reduction in the depletion of oxygenated haemoglobin, total haemoglobin and the normalised tissue haemoglobin index of the lateral gastrocnemius in heel raise exercises.

During quiet standing laser Doppler measurements of the dorsalis pedis artery indicated that the NIRS results were not a consequence of vasospastic responses or increased resistance to blood flow in response to vibration. Whilst heart rate and blood pressure remained constant, blood flow velocity significantly increased, suggesting the peripheral changes occurred independently of central cardiovascular function.

Heel raise exercises with whole body vibration showed significant increases in muscle activation of the soleus, but not the gastrocnemius, indicating varied muscular responses to vibration. The influence of blood flow and tissue oxygenation on EMG parameters was demonstrated via the protection of muscle conduction velocity during static squats, despite a downward shift in median frequency of the EMG power spectra.

Analysis of upper body muscles during press ups yielded significant increases in muscle activation, equivalent to increasing the load of the bench press by 10% of the one repetition maximum. The results indicate that vibration influenced the dynamic muscles more than stabiliser muscles; reinforcing the lower body studies showing that vibration has a varied influence on muscle function. The aforementioned results demonstrate the ability of vibration to augment the effects of exercise on the muscular and vascular physiological systems of the human body.

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#### ABBREVIATIONS

30L	Vibration at 30 Hz 1.2 mm	
40H	Vibration at 40 Hz 1.9 mm	
BFV	Blood flow velocity	
cHb	Total haemoglobin	
CV	Conduction velocity	
EMG	Electromyography	
EMD	Electromechanical delay	
FO	Fibre orientation	
g	Hedge's g	
g	Acceleration in multiples of -9.81 ms <sup>-2</sup>	
Hb	Haemoglobin	
HHb	Deoxyhaemoglobin	
HRmax	Maximum heart rate	
Hz	Hertz	
k	1000	
m	Metre	
MAD	Median absolute deviation	
Mb	Myoglobin	
MDF	Median frequency	
min	Minute(s)	
MNF	Mean frequency	
MVC	Maximal voluntary contraction	
nTHI	Normalised tissue haemoglobin index	
NVIB	No vibration	
O2Hb	Oxyhaemoglobin	
PV	Pooled variance	
RMS	Root mean square	
rpm	Revolutions per minute	
S	Second(s)	

SD	Standard deviation
SEM	Standard error of the mean
TDC	Top dead centre
ТОІ	Tissue oxygenation index
VIB	Vibration
W	Watts
W	Coefficient of concordance
WBV	Whole body vibration
Δ	Delta i.e. change in values
$\widehat{\omega}^{2}$	Omega squared

#### PhD OUTPUT

### Peer reviewed publications

**Robbins, D;** Adams, R and Goss-Sampson, M (2013) Can bench press exercises be used to quantify changes in EMG during press ups on a vibrating platform? *European Journal of Sports Sciences* (under review).

**Robbins D**, Chapman, M and Goss-Sampson MA, (2013) Effects of vibration on dynamic and stabiliser muscle activities during the press up *Physical Therapy In Sport* (under review).

**Robbins, D**; Yoganathan, P and Mark Goss-Sampson (2013) The influence of whole body vibration on the central and peripheral cardiovascular system. *Clinical Physiology and Functional Imaging* (accepted pending minor revisions).

**Robbins D**, Goss-Sampson MA, (2013) The influence of whole body vibration on myoelectric properties of the vastus lateralis *International Journal of Sports Science* Volume 3, Number 4,135-140.

**Robbins D**, Goss-Sampson MA, (2013) The influence of whole body vibration on the plantar flexors during heel raise exercise, *Journal of Electromyography and Kinesiology* 23 614–618

**Robbins, D**; Zeinstra, E, B; Jimenez, A; Goss-Sampson, M (2012) Does whole body vibration have clinically significant neurophysiological and neurovascular implications? *Journal of Prevention and Treatment* Volume 1, Number 2, 18-26.

**Robbins, D.** Elwell, C. Jimenez, A and Goss-Sampson, M. (2012) Localised muscle tissue oxygenation during dynamic exercise with whole body vibration. *Journal of Sports Science and Medicine* 11, 346-351.

#### **Industrial Scientific Reports**

The Effects of PowerBIKE<sup>™</sup> on Cyclical Muscle Activation Patterns, *produced for Power Plate International Ltd.* 

Annual summary of vibration exercise research in 2011, produced for Power Plate International Ltd.

Resources for Research and Practice, *The Sport and Exercise Scientist* issue 35 2013.

## **Conference Presentations**

**Robbins, D**; Yoganathan, P and Mark Goss-Sampson, (July 2013): The influence of whole body vibration on the central and peripheral cardiovascular system. IUPS conference, Birmingham; **poster presentation.** 

**Robbins D,** Chapman, M and Goss-Sampson MA, (April 2013): Effects of vibration on dynamic and stabiliser muscle activities during the press up. BASES Student Conference, Cardiff; oral presentation.

**Robbins D,** Goss-Sampson MA, (April 2012): The influence of whole body vibration on the plantarflexors during heel raise exercise. BASES Student Conference, London, Docklands; oral presentation, *Winner of best presentation in applied research award category*.

**Robbins D,** Matharoo J and Goss-Sampson MA, (March 2012): Progressive vibration exercise research. Kent county local network research, Medway; **poster presentation**.

**Robbins D,** Jimenez A, Elwell C and Goss-Sampson MA, (September 2011): Acute effects of vibration on muscle oxygenation during plantar flexion exercise. BASES Annual Conference, Colchester; oral presentation.

Goss-Sampson MA, **Robbins D**, Matharoo J, Filingeri D and Jimenez A, (September 2011): The effects of powerBIKE on cyclical muscle activation patterns. BASES Annual Conference, Colchester; **poster presentation**.

## **CHAPTER 1: INTRODUCTION**

Despite the fact that the role of vibration in health and exercise has been explored for over 100 years, the fundamental mechanisms of the biomechanical and physiological changes are still not clearly understood. The earliest records of the application of vibration for health benefits is that of Jean-Martin Charcot, who in 1880 noted greater improvements in his pilgrim Parkinson's patients who had to travel from a distance, leading to the assumption the improvements were related to vibrations from horse-drawn or railway carriages <sup>1</sup>. Based on this observation, Charcot produced a vibrating chair (see Figure 1) to simulate the vibrations of travel <sup>2</sup> and inspired Gilles de la Tourette, a younger colleague of



Figure 1. Charcot's vibrating chair, taken from Goetz<sup>1</sup>.

Charcot, to invent a motorized hat (see Figure 2) lined with adjustable steel plates to adjust to the size of the patients head. The vibrating helmet was used for the treatment of neurasthenia, which includes characteristic symptoms of fatigue, anxiety, headache, neuralgia, depressed mood, and migraines <sup>1</sup>.



Figure 2. de la Tourette vibrating hat<sup>1</sup>.

The idea of utilising vibration as a clinical application was continued throughout the early twentieth century by Dr J.H Kellogg, who invented a range of devices including: an updated vibrating chair, a platform, a bar used to massage the feet and lower legs and a belt used to vibrate different body sections <sup>3</sup>.

The first application of vibration in relation to sport and exercise is credited to Vladimir Nasarov in the 1970's who applied vibrations to athletes during eccentric training movements to increase athletes power and flexibility <sup>4</sup>. Current applications of vibration involve a wide range of products based on different methods of training, potential applications include:

- Whole body vibration (WBV) where platforms vibrate while participants complete training movements whilst standing on the platform.
- Partial body vibration whereby a participant is half on and half off a vibrating platform whilst completing training movements such as press ups or lunges.
- Local vibration; where a handheld device applies vibration to specific body regions for therapeutic or training purposes.

There are also different methods of generating vibration, including:

- Synchronous vertical vibration
- Oscillating vertical vibration
- Rotational vibration.

Out of the varying types of equipment synchronous research on vertical vibration and oscillating vertical vibration platforms are by far the most common, which is mostly a consequence of the fact they are also the most widely available commercially.

### **CHAPTER 2: LITERATURE REVIEW**

#### 2.1 Application of vibration in exercise research

Vibration is the mechanical process of reciprocating oscillations. In 2011 there were 103 scientific publications based on, or relating to, vibration exercise (unpublished research by the thesis author). Of these 103 articles, approximately 14% were reviews and 86% were original research. Over half of the original research articles, approximately 61%, used either synchronous vertical vibration or oscillating (alternating sides) vertical vibration (see Figure 3). The remaining studies utilised custom built or local vibration equipment – many of which utilised vibration which was not explicitly defined as synchronous or oscillatory;



Figure 3. Types of vibrating platform used in research during 2011.

While synchronous and oscillating vibration are both in essence providing a vertical vibrations to the body, the style in which the vibration is introduced is fundamentally different. Synchronous vibration is a uniform vibration produced evenly across the entire platform, whereas oscillating vibration is produced via a pivoting platform which moves about a central point between user's feet. (see Figure 4).



Figure 4. Synchronous vertical vibration (left) and oscillating platforms (right) (taken from Cardinale and Wakeling 2005).

Recently Power Plate Ltd began the process of developing a novel form of whole body vibration via a stationary cycle with a mechanical mechanism providing the option of introducing vibration. However, to date there have not been any published studies to provide peer reviewed conclusions about the effects of this form of vibration exercise.

#### 2.2 Physiological responses to different types of platform

Recent meta analyses considering the effect of WBV on muscular strength <sup>5</sup> and power <sup>6</sup> concluded that synchronous vertical vibration platforms elicit a significantly larger effect for chronic adaptations, where chronic adaptation were defined as those repeatable after one week, compared to oscillating platforms in both measures (muscular strength and power). However, oscillating platforms elicit a greater treatment effect for acute, i.e. less than one week, strength training adaptations. The difference in strength and power adaptations is potentially due a difference in the ratio of force to acceleration, known as mechanical impedance, which has been shown to be lower in oscillating platforms  $^{7}$ . Rittweger  $^{8}$ combined the theory of oscillating platforms generating lower acceleration forces with the observation that the oscillating platform created rotation about the hips and concluded that this was the underlying mechanism for attenuating vertical accelerations at the spinal level and above. Although conversely, Pel et al.<sup>9</sup> recently reported that oscillating platforms produced higher accelerations of up to 15 q compared to vertical vibrating platforms which produced accelerations at approximately half the value (up to 8 g). These higher accelerations could potentially explain the increased acute effects reported <sup>6</sup>. However, the ultimate differences and the underlying mechanisms between exposure to

the different styles of vibration types have yet to be ascertained <sup>10</sup>. It should also be noted oscillating platforms tend to operate at lower frequencies than synchronous vertical vibrating platforms and the reported studies used for the meta-analysis on strength <sup>5</sup> typically were based on lower exposure durations than that of synchronous vibration. The effects of synchronous and oscillating WBV on heart rate and VO2 were later compared by Gojanovic and Henchoz<sup>11</sup>, who found that oscillating WBV produced greater increases heart rate than synchronous WBV. The authors' intimated that the increased heart rate could result in elevated VO2, provided the intervention lasted longer than 20 minutes. Abercromby *et al.* <sup>12</sup> reported increased electromyographic (EMG) activity in the lower limb extensors, but not flexors, during oscillating WBV compared to that of synchronous vertical WBV. This paper received some criticism for using digital band stop filters which been known to result in signal attenuation<sup>8</sup>, therefore potentially excessively reducing signal amplitude. However, since the conclusion was based on *increased* signal values, accounting for potential signal attenuation would only increase the significance of the findings. In order to ascertain the implications of EMG signal changes, one must first consider the neurophysiological influences of vibration.

#### 2.2.1 Neurophysiological responses to vibration

The underlying neuromuscular responses to vibration have been studied extensively for approaching 100 years. However, originally local vibration was applied directly to the muscle belly or the muscle tendon. Generally local vibration is more effective when the tendon is vibrated, however if the intensity of the vibration is high enough the reflexes also appear when the vibration is introduced to the muscle belly <sup>13</sup>. In 1938 Echlin and Fessard <sup>14</sup> investigated the effects of introducing vibration to the tendons of cats, rabbits and frogs on the output of muscle stretch receptors. Their findings suggested that not only did vibration result in increased neural output of muscle stretch receptors, but that if the discharge rate of the receptor was activated by pre-stretching, to an output level similar to that of the vibration frequency, the threshold for activation of the receptor was drastically lowered. These findings were confirmed in 1951, when Kuffler *et al.* <sup>15</sup> performed similar experiments and noted high sensitivity of mammalian muscle spindles to vibration. As

anatomical knowledge progressed it was discovered that muscle stretch receptor components and their innervations reacted differently to vibration. Stretch receptor components that have high conduction velocity afferent nerves are the most responsive to vibration (see Table 1).

	Nuclear Bag	Nuclear Chain Central	Nuclear Chain Polar	Myotube
Conduction velocity	High	Low	Low	Low
Vibrator response	Yes	Yes	No	Yes
Rapid stretch response to vibration	Phasic	Tonic	Tonic	Phasic

#### Table 1. Differentiating characteristics of spindle end organs.

Table taken from Bianconi & van der Meulen<sup>16</sup>

Furthermore it has also been suggested that vibration can initiate bidirectional stimulation of nerve fibres via ephaptic transmission, where orthodromic stimulation of an axonal branch of a nerve results in antidromic transmission along a different local axonal branch of a nerve <sup>17</sup>. While this theory is speculative, it can be suggested that the process would therefore initiate an enhancing effect, where once transmission began there would be an increase in neural activity until a threshold point was reached.

In 1974, a review of vibration studies <sup>18</sup> reported that typically there are three motor effects resulting from the introduction of vibration to a muscle:

- 1. Muscle contractile activity is increased due to the tonic vibration reflex. The increase is a gradual process occurring over 20 -60 seconds
- 2. The excitability of motor neurons innervating the antagonistic muscles is depressed via reciprocal inhibition
- 3. The monosynaptic stretch reflexes of the vibrated muscle are suppressed.

This summary would suggest that ephaptic transmission is a potential underlying mechanism for muscle activation as a consequence of vibration. However, ephaptic transmission contraction enhancement will only apply to local or direct vibration, as the stimulus of WBV is not applied directly to a muscle but transmitted via whichever body part

is in contact with the source of vibration (usually the feet). More recent technological advances, such as ultrasound, have allowed detailed non-invasive analysis of muscle function during WBV. Whole body exposure to oscillating vibrations at 6 Hz has been shown to elicit 50% greater contractile tissue displacement than that caused by an isometric contraction created by static posture in quiet standing. An increase in the changes of muscle fibre length activates muscle spindles, which in turn generate muscular tone; this therefore providing an explanation for the underlying mechanism for stretch receptor output and the consequential increase in muscle tone following both direct and indirect vibration exposure <sup>19</sup>. In addition to intramuscular receptors there are also cutaneous receptors known as Pacinian corpuscles which can detect vibration <sup>20</sup>. While Pacinian corpuscles can detect vibrations on the surface of the skin, they are not the receptor responsible for the tonic vibration reflex <sup>18</sup>. However, Pacinian corpuscles have been linked to sympathetic nervous activity, such as sweating and changes in vascular function <sup>21</sup> providing a potential explanation of the underlying mechanisms for additional physiological responses to WBV exercise.

It is of interest to note that despite the obvious similarities between the proposed underlying mechanism and that of stretch reflexes, WBV does not appear to facilitate reflexes. To date there have been two key studies looking at the effect of vibration on reflex activity. In 1966 De Gail *et al.*<sup>22</sup> looked specifically at reflex function during vibration. During this investigation reflexes and responses to local vibration were tested while the participants were administered pharmaceutical interventions known to influence peripheral neurophysiology in a specific manner. The interventions and responses are summarised in Table 2.

Intervention	Neurophysiological Effect	Effect of Reflex During Vibration
Procaine block	Selective block of gamma efferent fibres	Tonic contraction decreased to one third or less of the control level, tendon jerks were reduced to about half.
Barbiturate block	Block polysynaptic pathways	Tonic contraction was abolished, tendon jerk preserved.
Ciba 28,882-Ba	Block polysynaptic pathways, static stretch receptors and discharge rate of gamma motor neurones	Tonic contraction markedly reduced, tendon jerk response unchanged.

Table 2. Pharmaceutical effects on reflexes.

Information taken from De Gail et al. 22

In the absence of pharmaceutical intervention the researchers also noted a depression of the tendon jerk reflex by local vibration. However, this depression could be reduced, or even removed, by the Jendrassik manoeuvre where participants clasp hands together and generate a small amount of force by pulling their hands apart and/or clench their jaw. It has been suggested that remote muscle activation results in the reduced presynaptic inhibition of 1a afferents allowing facilitation of reflex activity <sup>23</sup>; Although it should be noted the influence of the Jendrassik manoeuvre has been shown to reduce with age <sup>24</sup>, which could have implications if testing with participants of heterogeneous age groups.

From these results the following conclusions were drawn:

- 1. The tonic vibration reflex involves excitation of muscle spindles, as the reflex is reduced during a procaine block.
- The tonic vibration reflex involves more than one interneuron, as the reflex is also reduced by a barbiturate block and Ciba 28,882-Ba.
- The tonic vibration reflex is influenced by supraspinal levels as the Jendrassik manoeuvre influences tendon jerk depression.

More recently in 2011 Ritzmann *et al.*<sup>25</sup> investigated the effect of WBV on the H-reflex, the stretch reflex and the short latency response during hopping. The logic for this somewhat more functional approach was that the H-reflex bypasses the muscles spindles by direct electrical stimulation, whilst still operating at a spinal level via gamma motor neurons. The stretch reflex is modulated by alpha-gamma linkage and hopping is a complex motor task modulated at all levels i.e. spinal and supraspinal centres. The results obtained displayed significant reductions in the H-reflex, which occurred during WBV and had not recovered after 5 min rest. The stretch reflexes at the ankle (soleus and medial gastrocnemius muscles) were significantly reduced in a sitting position but not whilst standing; suggesting that active muscles, or neurophysiologically complex tasks increase reflex recovery. Finally, the short latency response of the muscle during hopping was not affected. Based on these results the following conclusions were drawn:

- 1. The basic responses of muscles to indirect vibration are similar to that of direct vibration.
- 2. The greater influence of WBV on the H-reflex is a consequence of the H-reflex sensitivity towards pre-synaptic inhibition.
- Tasks which involve greater motor complexity e.g. standing or hopping, involve supraspinal centres which increase reflex recovery in active muscles via tonic vibration reflex inhibitory mechanisms.

Despite being published 45 years apart, the findings from these studies reflect each other very well. Both studies indicate that the greatest responses i.e. reflex depression, occur in inactive muscles and that pre-activation will diminish the effect of vibration on reflexes. Both studies also agree that underlying neurophysiological response is polysynaptic in nature and vibration does not facilitate reflexes under any conditions. Finally both papers agree that supraspinal centres influence the neurophysiological response to the effect of vibration.

# 2.2.2 Neurophysiological risks and considerations in relation to exercise related vibration

The risks and consequences of occupational vibration have been acknowledged and addressed by researchers and government organisations, such as the UK's Health and Safety Executive, who provide information about the risks of industrial vibration and exposure during work commitments. This has been deemed necessary as both WBV and local vibration can lead to Hand and Arm Vibration Syndrome (HAVS) and/or debilitating conditions such as back pain (www.hse.gov.uk/vibration/). In addition, there are various methods by which nerve injuries can occur e.g. compression, traction and/or friction, often resulting from repetitive forceful motions in awkward or unusual positions <sup>26</sup> which are an integral part of sports activities, particularly during learning stages and/or regular participation that can be aggravated by vibration. Despite this, the neurophysiological and neuromechanical implications with regards to vibration during exercise have still received scant attention. One area which has been investigated is the risks involved with excessive vibration during cycling. Regular cyclists, particularly those who receive higher levels of

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vibration e.g. off road cyclists, are at risk of upper limb compression neuropathy <sup>27–30</sup>. Nerve injury is more frequently seen in the ulnar nerve than the median nerve, <sup>31,32</sup> with symptoms including weakness of grip and occasionally numbness of the fourth and fifth fingers <sup>33</sup>. Prevention or management of the condition, if caught early, is relatively simple with recommendations including: use of correct protective equipment (padded gloves and handlebars), ensuring the correct set up of the bicycle e.g. seat and handlebar positions, regularly changing the position of hands whilst riding <sup>34</sup>. Wilmarth and Nelson <sup>31</sup> performed a prospective study before and immediately after a four day 600 Km bicycle race and found 70% of the study participants experienced either upper limb ulnar nerve motor, sensory or both symptoms following the race. These considerations are particularly relevant with the current development of Power Plate Ltd's new PowerBIKE which has a vibrating mechanism acting upon the pedal crank. Of all the aetiological factors considered, the most common for ulnar nerve compression injuries is pressure on the heel of the hand during vibration <sup>32</sup>. This obviously applies to other exercise situations, such as during press ups where even performing the exercise on a hard floor has been shown to cause ulnar nerve injury, <sup>35</sup>. The presence of vibration, for example when completing push ups with hands on a vibrating platform, therefore increases the relative risk. This risk can again be exaggerated if the hands also receive impact e.g. during plyometric press ups, potentially leading to a debilitating condition of the ulnar nerve known as Hypothenar Hammer Syndrome<sup>33</sup>. The ulnar nerve is not the only nerve at risk during vibration exercise. During press ups participants are required to place their hands directly upon the platform in a position similar to that of the 'provocative positions' which are positions which exacerbate symptoms used in the clinical diagnosis. Examples of provocative positions in carpal tunnel syndrome are the "reverse Phalen's manoeuvre" (see Figure 5) where hands are held at shoulder height, wrist extended, palms touching with fingers pointing to the ceiling and has been shown to produce significantly higher carpal tunnel pressures than the normal Phalen's manoeuvre which is flexed wrists with the backs of the hands placed together <sup>36</sup>; as this position is utilised for its ability to generate unusual or uncomfortable sensations.



Figure 5. Reverse Phalens's position.

When combined with vibration it has potential to be an issue for carpal tunnel patients. Another test is the carpal compression test or Durkan's test (see Figure 6) where direct pressure is placed upon base of the palm just distal to the wrist  $^{37}$ .



Figure 6. Durkan's carpal pressure test.

The carpal compression test has been shown to achieve a sensitivity of 87%, and a specificity of 95% <sup>38</sup>, again confirming the risks of added pressure if the participant has a pre-existing condition. Median nerve compression injuries at the wrist are reported in athletes who perform repetitive gripping or sustained wrist hyperflexion or hyperextension or who are exposed to vibration <sup>26</sup>. It should also be noted that the most severe nerve injury and structural changes (demyelination, interstitial and perineurial fibrosis) occur just

proximal to the wrists <sup>39</sup>. If large diameter nerve fibres are affected prior to small fibres, as is often the case in carpal tunnel syndrome <sup>40</sup>; then proprioceptive input will be reduced. In study considering the outcome of 55 carpal tunnel release operations performed due to vibration induced carpal tunnel syndrome, Hagberg et al. <sup>41</sup> found that the level of exposure prior to operation influenced the recovery after the surgery. These findings again confirm that neural structural changes occur in response to vibration. When considering the neurophysiological basis for nociceptive signals the gate theory suggested by Melzack and Wall <sup>42</sup> and further refined by Wall <sup>43</sup>, suggests that there is a control system at the spinal level helping to modulate the signals transmitted to the brain. Essentially signals from small and large diameter fibres (noxious and non-noxious sensory signals respectively) initially communicate with an inhibitory interneuron within the spinal cord (see Figure 7). These inhibitory interneurons alter the ratio of the final ascending signal sent from the projection neuron. This process therefore regulates the intensity of the signal travelling up the spine to the brain. Consequently, if large nerve fibres are less active, there is less inhibition of nociception, in this instance it is said 'the gate is open'.



Figure 7. Representation of the gate control of pain theory.

Given that some pathological conditions, such as peripheral neuropathies and carpal tunnel syndrome <sup>40</sup>, have been shown to damage large nerve fibres prior to small nerve fibres; this potentially provides one underlying mechanism for some clinical populations finding vibration exercise unpleasant. Although the concept that finding vibration unpleasant should not be restricted to clinical populations, Rittweger et al. <sup>44</sup> reported most subjects felt uncomfortable after approximately five minutes of WBV if they were not allowed to change position. It should also be noted that when considering the effect of vibration on peripheral nerves, Goldsmith et al. <sup>45</sup> suggested that the primary nerves affected by vibration white finger were the small nerve fibres. As nerve conduction studies displayed lower range sensory action potentials and nerve biopsies illustrated that neuronal damage had to be quite severe before large diameter nerve fibres were affected, despite conclusions being based on a small sample group (n= 6), the findings must be considered. A lack of nociceptive input decreases the effectiveness of the body's protective mechanisms. This was recently confirmed in a study by Sandén et al.<sup>46</sup> who tested a cohort of office and manual workers for effects of combined hand/arm vibration on nerve conduction, especially the conduction of the large diameter nerve fibres; no relationship between exposure and distal neuropathy was observed. While this would suggest that vibration induced injury is unlikely to result in a condition that will further 'open the gate', it has clearly been shown that carpal tunnel syndrome can result in increased pain levels. However, it must also be considered that any condition which previously resulted in damage to large nerve fibres has potential to 'open the gate' (see Table 3 for conditions which 'open/close the gate').

Table 3. Conditions which open/close the gate in pain theory.

Pactors that Regulate Spinal Gate Control			
Gate Open		Gate Closed	
	Aδ and/or C fibres active	A $\alpha$ or A $\beta$ fibres active	
	Overuse	Relaxation	
Physiological	Fatigue	Strengthening/ Conditioning	
	Improper Mechanics	Monitored exercise	
	Tired	Rested	
Medical	Extent of injury/-	Medication	
	Pathological condition	Cooling/Heating	
Cognitive	Focussing on Pain	Distraction	
	Anxiety/Fear	Relaxation	
	Depression	Happiness	
	Negative attitude	Positive attitude	
	Stress	Prior Experience	

<b>Factors that</b>	Regulate	Sninal	Gate	Control
i actors that	Regulate	Spinai	Jace	Control

Compiled from 47-49

There is also potential that a change in the ratio of peripheral stimulation results in 'undue perception of exertion' as described in Rittweger *et al.*<sup>44</sup>. It should also be noted that this process can be beneficial in pain reduction if large nerve fibres are not damaged or in neuropathies such as in diabetic small fibre neuropathy <sup>50</sup>. Therefore, while gate theory has the potential to act as an underlying mechanism for perception of a WBV experience, more studies considering the perception of WBV exercise are needed. It is of interest to note that while the potentially different frequencies of vibration during exercise, compared to that of industrial applications, in combination with reduced exposure time, might not exaggerate pre-existing conditions; the process may be uncomfortable for the participants. The key points from these studies are summarised in Table 4.

Table 4. Summary of key findings neurophysiological studies which suggest vibration is an influencin	g factor.

Author	Publication date	Ν	Relevant points
Eckman	1975	3	Clinical case study of cyclists who developed ulnar neuropathy
Noth	1980	4	Clinical case study of cyclists who developed deep and distal ulnar neuropathy
Weiss	1985	132	Groin numbness and palmar pain or paraesthesia each occurred in approximately 10% or bicycle riders during a 500 mile race
Wilmarth	1988	25	Sensory nerve conduction velocity of the ulnar nerve following cycling
Walker	1988	1	Clinical case study of ulnar nerve injury caused by push ups on a hard surface
Hagberg	1991	41	Level and intensity of vibration exposure influences recovery after carpal tunnel surgery
Richmond	1994	Review	Compression neuropathy, more commonly ulnar than median, is frequent but seldom produces permanent injury or deficit if promptly recognized and managed
Goldsmith	1994	47	Primary nerves affected by vibration are small nerve fibres
Rittweger	2001	12	5 min WBV results in most subjects feeling uncomfortable
Patterson	2003	25	Cyclist's palsy occurs at high rates in both experienced and inexperienced cyclists.
Sandén	2010	155	no exposure-response association between hand-arm vibration exposure and distal neuropathy of the large myelinated fibres
Hong	2011	1	Clinical case study describing pain reduction in diabetic small fibre neuropathy following WBV

#### 2.2.3 Neurovascular risks and considerations in relation to exercise related vibration

While generally neurovascular injuries in the hands of athletes are rare <sup>51</sup>, the relatively high occurrence resulting from occupational exposure to vibration warrants consideration when introducing a participant to vibration exercise. Vibration introduced to the human body, whether whole body vibration or partial body vibration, potentially can influence peripheral vascular structure and consequently blood flow. One of the most common conditions resulting from hand/arm vibration is Raynaud's phenomenon (RP) and/or its secondary form Vibration White Finger (VWF). Both these are vasospastic conditions of the extremities for which the greatest risk occurs at frequencies 40-125 Hz<sup>52</sup>. Widely available recreational vibration equipment can operate inside this range, therefore exposure risks further vasospastic responses. Typically, a vasospastic response, or excessive constriction of a blood vessel, will result in local pain, cyanosis (blue colour of the skin), pallor and altered sweat secretion <sup>30</sup>. As peripheral sweat glands are under the control of the sympathetic nervous system, this suggests a global response as opposed to a purely local response. This hypothesis is strengthened by additional global responses to upper limb vibration, such as an increased heart rate <sup>53</sup>, bilateral vasospastic responses to a unilateral hand vibration exposure <sup>54</sup> and vasospastic responses in the feet resulting from upper limb exposure to cold when assessing for hand/arm vibration syndrome <sup>55–57</sup>. Local vibration to the palm of the hand has also been shown to result global responses such as increased skin sympathetic activity in the region innervated by the tibial nerve and causing increased perspiration of the sole of the foot <sup>58</sup>. These responses have been attributed to both a global response via a centrally mediated sympathetic vasoconstrictor reflex and local responses within the blood vessels themselves <sup>53</sup>. The connection between cutaneous vibration receptors (Pacinian corpuscles) and the sympathetic nervous system has previously been identified as postganglionic sympathetic fibres within Pacinian corpuscles <sup>59</sup>. The connection between Pacinian corpuscles and sympathetic nervous activity was further supported by Hyvärinen et al. <sup>21</sup> who reported a constant relationship between vasospasms and the mean threshold for Pacinian corpuscle activation. It has therefore been suggested that continuous activation of these receptors could result in a vasoconstriction a consequence of the reflexive efferent discharges <sup>54</sup>. However, the resulting responses to vibration have been attributed to both a vasoconstrictor reflex and an active local vasodilatation, with both

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mechanisms competing against each other <sup>53</sup>. One potential underlying mechanism influencing local vasodilatation is local release of endothelial-derived relaxing factor (EDRF) and prostacyclin <sup>54</sup>. The endothelium itself can be considered a dynamic interface between the vascular compartment and the extravascular space, acting in the roles of assisting in the regulation of protein flux, inflammatory cells into tissues, blood flow and prevention of thrombosis <sup>60</sup>. The release of EDRF and prostacyclin can be stimulated by shear stress in isolated blood vessel sections <sup>61,62</sup>. As blood is a viscous liquid it is the principle cause of shear stress on the endothelium, particularly the blood that is contact with endothelial cell surface which does not flow at the rate of blood central to the blood vessels. However, unlike mechanical strain, shear stress is focussed on the endothelium and not transferred to local tissues <sup>60</sup>. The mechanical stresses within vasculature and the predicted consequences as described have been mathematically modelled <sup>63,64</sup>. It has also been shown that as blood flow increases an enhanced rate of prostacyclin release is observed <sup>65</sup>. Therefore, it is conceivable that the endothelin-induced release of prostacyclin results from increased shear stress <sup>66</sup>. The role endothelin-1 of has been investigated due to its ability to produce sustained vasoconstriction, therefore increasing shear stress and release of EDRF, however when endothelin-1 was introduced in the presence of a strong vasodilator (which abolished the vasoconstriction) no increase in EDRF was observed <sup>66</sup>, which indicates that shear stress not the presence of endothelin-1 influenced the increased release of EDRF. Based on these findings the endothelium clearly has an important role in the response to shear stresses, potentially by activating cell signalling pathways which trigger effector responses, unfortunately to date it not known if these responses are direct mechanosensors i.e. they automatically respond to stress/strain, or they are mechanosensitive i.e. they respond to local signals produced in response to stress/strain <sup>60</sup>. The effect of shear stresses on larger vascular structures have also been investigated by assessing pulse wave velocity and blood pressure at the ankles which can be used as predictor of peripheral arterial stiffness. To completely differentiate between peripheral and central arterial stiffness is problematical, however, it has been suggested that by obtaining wave velocity and blood pressure recordings at the ankles, peripheral influence would be greater <sup>67</sup>. The findings indicated that changes in arterial stiffness reduced approximately 20 minutes after WBV exercise sustained, with reductions lasting for circa 40 min. The authors hypothesised that the underlying mechanism is vasodilatation via vascular

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endothelial function. This hypothesis is supported by reports that vibration during cycling resulted in an increase in the release of vascular endothelial growth factor (VEGF), a mitogen regulator of angiogenesis and matrix metalloproteinases (endopeptidases) MMP-2 and MMP-9<sup>68</sup>, which have roles in vascular remodelling<sup>69</sup>. Although, it should be noted that VEGF release following cycling with vibration was related to the participants training status as greater releases were noted in higher trained participants<sup>68</sup>. It is not known if this was due to an increased production of VEGF or a release of increased stored levels of VEGF. It is possible that these changes also occur due to increased shear stresses as vibration during exercise has been shown to increase blood flow<sup>70–72</sup> and increased tissue oxygenation parameters<sup>73,74</sup>. However, it should be noted that although vasospastic responses to vibration have been noted in the extremities; it has not yet been established if changes in tissue oxygenation are in relation to increased blood flow or to vasospastic responses in the feet.

A final point of interest is that shear forces in blood vessels have also been shown to create mechanical risks which researchers and practitioners should be aware of. Two case studies have recently reported patients presenting with uniocular drops in vision clinically attributed to vitreous haemorrhage, the suspected cause in both cases was WBV. Bertschinger & Dosso<sup>75</sup> reported the case of a 43 year old man who presented with a uniocular drop in vision, the only reported change in recent activity was the introduction WBV for a period of two weeks prior to developing the condition. More recently Gillan et al.<sup>76</sup> reported the case of vitreous haemorrhage with localised posterior retinal detachment in a 52 year old male. The patient presented with a uniocular drop in vision following a single session of WBV. While the authors acknowledge that causality cannot be categorically related to WBV, previous issues such as vitreous liquefaction has been reported in workers using pneumatic drills<sup>77</sup> indicating there is a need for caution, particularly if interacting with people who have previously suffered from this condition, and further research into this area. The key points from these studies are summarised in Table 5

Author	Publication date	Ν	Relevant points	
Hyvärinen	1973	43	Traumatic vasospastic disease is a chronic over excitation of the Pacinian vibration receptors that produce spastic reactions in the vasculature through a reflex linkage with the sympathetic nervous system	
Kroemer	1989	Review	Frequencies of 40-125 provide greatest risk of vasospastic diseases in hands	
Aulicino	1990	Review	Neurovascular injuries are rare in the athlete. Modification of protective devices, alteration of technique, and education may help avoid neurovascular problems described in this article	
Sakakibara	1990	5	Vibration exposure of the hand triggers sympathetic activity in the tibial nerve innervating the foot, and causes vasoconstriction of the toe and perspiration on the sole of the foot	
Sakakibara	1991	43	Hand -arm vibration syndrome affects circulation in the feet	
Greenstein	1992	28	Unilateral hand vibration causes bilateral vasospastic responses	
Sakakibara	1994	Review	Arterial pathological changes like medial muscular hypertrophy have been observed in both the fingers and the toes of hand-arm vibration patients	
Egan	1996	34	Hand vibration causes a generalised increase in sympathetic tone in the heart and extremities.	
Mansour	2000	39	Workers using pneumatic drills risk vibration-induced pigment deposition in the trabecular meshwork and vitreous liquefaction	
Suhr	2007	12	In conclusion, the results support the contention that mechanical stimuli differentially influence factors involved in the induction of angiogenesis.	
Otsuki	2008	10	WBV acutely decreases arterial stiffness	
Bertschinger	2008	1	Clinical case study of spontaneous vitreous haemorrhage following WBV exercise	
Gillan	2010	1	Clinical case study of spontaneous vitreous haemorrhage following WBV exercise	
House	2011	191	Workers assessed for HAVS frequently have cold-induced vasospasm of their feet	
Studies using animals to establish underlying mechanisms for responses				
Rubanyi	1986	In vitro testing	The release of EDRF can be stimulated by shear stress in isolated blood vessel sections	
Pohl	1986	Animal study (dogs)	Endothelial cells act as mediators of flow-dependent dilation	
Wennmalm	1991	Animal study (rabbits)	increase in blood flow facilitation of the formation of the endothelial mediators, EDRF, prostacyclin and endothelin	
Lamontagne	1992	Animal	EDRF formation may result from the high shear stress imposed on	

the endothelial lining by the periodic diameter reduction and from

the direct deformation of the endothelium.

study

(rabbits)

#### 2.3 Reporting issues in current literature

An additional important area to be considered is the manner in which information from vibration exercise research is both received and recorded. Previously published literature reviews have illustrated the inconsistencies of current publications with respect to the lack of structure and standardisation (Lorenzen et al. 2009; Mikhael et al. 2010; Lau et al. 2011). The lack of standardisation creates two major issues. Firstly it makes systemic reviewing difficult and subsequent meta-analysis impossible. Secondly, it creates difficulties in undertaking safe and progressive research. For example, previous publications have been noted to either not report an amplitude <sup>81</sup> or include inconsistencies in the units within an article e.g. 3 cm amplitude in the abstract and 3 mm amplitude in the methods <sup>82</sup>. While it can be assumed this was simply a typing error that survived editing it presents a risk that researchers unfamiliar with human vibration, or researchers who are simply less than vigilant, could base their practises or research on the information presented in the abstract. This would result in participants receiving ten times the intended magnitude of vibration!

Despite recent publications detailing correct reporting strategies <sup>78</sup> and even papers providing a checklist for reporters to use e.g. Rauch et al. (2010) for WBV <sup>84</sup>, for diagnostic accuracy tests, there are still papers using different terminology such as 'peak to peak amplitude' <sup>85–88</sup>. Signal amplitude refers to the height of the signal waveform from the

baseline. The peak to peak distance is the *displacement* (see Figure 8) not an amplitude <sup>83,89</sup>. While as a reporting issue this may seem to be a minor issue. In practise if a non-vigilant researcher attempts to recreate the study there is potential for subjects to be exposed to double the amplitude, therefore increasing the exposure risk and of course producing data that is not applicable. Another issue to be considered is that not all



Figure 8. Vibration graph parameters.

equipment produces vibration via the same technique – or even in the same direction. Thoroughness when reporting details of studies such as the type of device, the sex of the participants, and the location of the participants on the equipment relevant to the centre
vibration would make a difference to standardisation in the literature. A final issue to be considered is the varying types of footwear used. It has been shown that different footwear influences the transmission of vibration (Marin et al. 2009). Although details of footwear are beginning to be included within the scope of the methods reported, the only form of effective standardisation to date is the removal of shoes from participants. While this approach is scientifically effective, it is not a transferable result for practitioners as recreational users will typically be wearing shoes. This lack of standardisation, combined with the plethora of areas investigated (see Table 6 for examples of typical areas within the scope of vibration research), has resulted in reviews containing minimal numbers of papers in the review process; for example Mikhael et al. (2010) searched over 50 years of data yet only reviewed 6 papers. Typically within WBV exercise the focus of research focus is on functional outcomes from exposure to WBV, for example potential increases in performance; muscle or bone mass; balance etc.

Subjective	Activity
Absolute thresholds Subjective equality Subjective order Equality of intervals Equality of ratios Rating of stimuli Cross modality judgements Differential thresholds	Vision Hearing Touch Proprioception Vestibular function Psychomotor performance Cognitive performance Vigilance
Physiological	Biodynamic
Skeletal	Body impedance
Muscle	Hand impedance
Nerve	Body transmissibility
Cardiovascular	Head movements
Respiratory	Hand movements
Central nervous system	Organ movements
Endocrine/metabolic	Energy absorbed

Table 6. Research parameters for human response to vibration (from Griffin 1990).

# 2.4 Thesis Aim

The review of the literature illustrates that while the neurophysiological responses to local vibration are established, it has not yet been confirmed if these results translate to WBV vibration exercise. It is also shown that vibration induces significant changes in peripheral vascular function, though it is not clear if this is direct result of vibration exposure or a consequence of increased muscle activity. The overall aim of this PhD is therefore to address the underlying biomechanical and physiological responses in peripheral muscular and vascular function to whole body vibration. By establishing these responses practitioners and researchers in health related professions will be able formulate evidence based exercise regimes and research questions.

# CHAPTER 3: THE EFFECT OF VIBRATION DURING CYCLING ON THE ELECTRICAL ACTIVITY OF LOWER LIMB MUSCLES

# 3.1 Introduction

The increase in popularity of the use of vibration during exercise has led to manufacturers looking for new approaches to utilise vibration during exercise. One approach that has been pursued is the invention of a vibrating bicycle by Power Plate Ld.

#### 3.1.1 Muscle function in cycling

When investigating cycling action the motion of the pedal, starting from its highest point, or 'top dead centre' is divided in to four sections (see Figure 9). As the cyclist moves through these phases the motion is classified as follows:

- 0°, top dead centre, the point at which there is the most mechanical difficulty to move the pedals.
- 25 160°, the power phase, downwards motion both quadriceps and plantar flexors generating the greatest force during the cycle.
- 180°, Bottom dead centre, as in top dead centre, the point at which there is also increased mechanical difficulty to move the pedals.
- 4.  $180 0^{\circ}$ , upward phase, including 270  $0^{\circ}$ , recovery phase, it is mechanically difficult to generate force in the upward phase of the motion <sup>91,92</sup>.



Bottom dead centre Figure 9. Pedal crank positions.

Typically the lower limb extensors, gluteus maximus and vastus medialis/lateralis, tend to be more active during the power stroke; lower limb flexors are more active from top dead centre through to 270°<sup>92</sup>. The ankle plantar flexor activation varies dependent on the muscle insertion, with medial and lateral gastrocnemius being active through 30 - 270° and soleus being active through 45 - 270°; the ankle dorsiflexor, tibialis anterior, is typically active from 45 - 135°. While these typical values can be observed in a variety of studies <sup>92</sup>, it should be noted that in trained cyclists there is a high heterogeneity in EMG patterns <sup>93</sup>. Therefore direct comparisons must be approached with caution.

#### 3.1.2 Addition of vibration to cycling

Based on the rationale that the addition of vibration would provide a greater stimulus to the cardiovascular exercise, Power Plate Ltd have been developing a vibrating stationary cycle. The powerBIKE has a mechanical innovation providing the user with the option of adding vibration to the stationary cycle via a clutch mechanism (see Figure 10). When activated the clutch mechanism pulls the pedal crank to an eccentric position which generates mechanical vibrations at a ratio of 20:1 i.e. 20 vibrations for full cycle of the pedals. As the vibration is generated mechanically the ratio is therefore generated at a fixed rate which therefore increases with increasing cadence. Therefore at 60 rpm the vibration frequency is 20Hz, at 90 rpm the frequency is 30Hz, at 100 rpm it is 33.3 Hz etc. The amplitude of vibration is fixed at 1 mm, resulting in a 2 mm peak-to-peak displacement.



Figure 10. PowerBIKE vibration mechanism, a = stationary cycle without clutch activated, b = clutch activated and vibrations being generated at the pedal crank. Images supplied by Power Plate ltd.

To date the powerBIKE is not yet available for public purchase as it is still under development and requires further validation in order to ascertain the physiological responses of self-generated vibration during cycling. Previous research has been limited to external vibration using stationary cycles directly attached to vibrating platforms in comparison to mechanical vibration generated by the crank <sup>68,94</sup>. The focus of both of these studies was the cardiovascular system in combination with metabolic factors such as lactate production and vascular enzymes, proteins and factors related to vasoconstriction and dilatation. The results of these studies indicate that both global and local responses occur as a result of the introduction of vibration to cycling, with increases in oxygen kinetics and peripheral haematological makers relating to angiogenic regulation <sup>68,94</sup>. To the best of the author's knowledge, to date there are no studies which have considered the effect of mechanical vibration during cycling on the neuromuscular activity of muscles. The aim of this project is to determine the effect of mechanical vibration on neuromuscular activity. The hypothesis for the study is that the vibration mechanism will increase the electromyographic activation patterns of the lower limb muscle groups.

# 3.2 Methods

#### 3.2.1 Participants

Seven male participants volunteered to participate in this study. The mean and standard deviation ( $\pm$  SD) values of the subjects' age, height and mass were 24.6  $\pm$  2.3 years, 1.79  $\pm$  0.04 m and 74  $\pm$  9 kg, and all subjects were right-leg dominant. All procedures had been previously approved by the University's Research Ethics Committee and participants provided informed consent to participate.

### 3.2.2 Study design

The format for this investigation was a randomised repeated measures study design. Each participant performed: 60, 70, 80 and 90 rpm on the powerBIKE with a randomised order of starting with vibration (VIB), or without vibration (NVIB). The bike was set at a fixed resistance (powerBIKE resistance setting 4) for 3 min at each cadence to reach steady state. The mechanical vibration was cadence-related with a ratio of 1:20 (pedal revolutions to mechanical vibrations) being equivalent to 20, 23.3, 26.7 and 30 Hz, 1 mm amplitude vibration respectively.

#### 3.2.3 Data collection and processing

Retro-reflective markers were fixed to the pedal crank in order to define pedal positions of top dead centre (TDC - 0°) and bottom dead centre (BDC - 180°) during the duty cycle (see Figure 9) using 10 infrared retro-reflective cameras (Oqus, Qualysis AB, Sweden) recording at 500 Hz.

Marker motion was tracked and all synchronous data exported in .c3d format for subsequent post processing in Visual3D (C-Motion). Post processing of data was achieved via interpolation with a 3<sup>rd</sup> order polynomial function with a max gap fitting of 50 data points (equivalent to 0.1 s) and a Butterworth low pass filter (4<sup>th</sup> order 6 Hz cut off).

Electromyography (EMG) was used to determine the activities of the major ankle flexors, quadriceps, hamstrings and hip extensors (see Figure 11). Differential bipolar (10 mm centre to centre) surface electrodes (DE-2.3, Delsys Inc. Boston, MA, USA) were placed over the lateral gastrocnemius, anterior tibialis, vastus lateralis, biceps femoris and gluteus maximus in accordance with SENIAM recommendations <sup>95</sup>. A single reference electrode was placed on C7 vertebrae and all leads connected to the electrodes were secured with tape to avoid artefacts from limb movements. Impedance was minimised by shaving and skin cleaning with alcohol swabs. EMG signals were amplified (1 k gain) via a Delsys Bagnoli system (Delsys Inc. Boston, MA, USA) with a bandwidth of 20-450 Hz. EMG activity was

synchronously acquired with the kinematic data at 2000 Hz. EMG data were initially filtered in Visual3D (C-Motion) using a 60 Hz cut-off 4<sup>th</sup> order bidirectional high pass Butterworth filter to remove any D.C. offset. A full rectification was applied before the signal was filtered with a 10 Hz cut-off, 4<sup>th</sup> order bidirectional low pass Butterworth filter. Amplitudes of each muscular activity displayed relative to pedal position were then determined, following the recommendation of Hug and Dorel <sup>92</sup>. In addition, individual muscle EMG activities were summated to give a 'total' lower limb value. The total limb value obtained during cycling at 60 rpm was taken to be a baseline value, increasing cadence and the addition of vibration was then analysed and plotted as a comparison to this baseline value.



Figure 11. Example of full set up for data collection.

#### 3.2.4 Statistical analysis

EMG recordings were filtered, smoothed and mean activities were determined for 12 duty cycles for each cadence. To illustrate the typical values radar plots were generated displaying the mean EMG amplitude of each muscle at each point in the pedal cycle. Due to the small sample size non parametric statistical tests were applied regardless of data distribution. EMG amplitudes were exported to MatLab (MathWorks, USA), Friedman's repeated measures test was applied to data for each muscle to determine differences in EMG amplitude resulting from increasing cadence. Wilcoxon signed rank test was applied to each muscle at each cadence to test for significance between VIB and NVIB. The level of significance from the Wilcoxon test was adjusted for repeated measures using the

Benjamini-Hochberg False Discovery Rate  $^{96}$ . Significance was set at alpha = 0.05 for all tests.

Due to significant differences in EMG amplitude analysis of the power required to turn the pedals at the different settings e.g. vibration on/off and various cadences, was completed using an ergometer calibrator (Lode, Holland). The calibrator performed an isokinetic analysis by physically rotating the pedal crank at specific cadences matching that performed during the investigation. Power measurements (Watts) were obtained at each of the cadences. The cadence, frequency, power and filtered EMG amplitude were input to Microsoft Excel 2010 for calculation of correlations, coefficients of determinations and generation of graph for presentation.

## 3.3 Results

The location of significant differences between mean EMG amplitude during vibration and non-vibration across the tested cadenced is displayed in Table 7.

Both vibration and cadence resulted in significant increases in EMG amplitude of the muscles assessed. The only muscles which did display a significant increase in EMG amplitude during the VIB condition were anterior tibialis and gastrocnemius, all muscles with the exception of the semimembranosus muscle affected by increasing cadence.

Muscle	VIB vs. NVIB	Cadence (significantly different from 60 rpm)		
Anterior Tibialis	NS	90 rpm*	90 rpm*	
Gastrocnemius	NS	90 rpm*	80, 90 rpm*	
Vastus Lateralis	*	80, 90 rpm*	80, 90 rpm*	
Vastus Medialis	*	80, 90 rpm*	80, 90 rpm*	
<b>Biceps Femoris</b>	$\checkmark$	90 rpm ✓	90 rpm*	
Semimembranosus	*	NS	NS	
Gluteus Maximus	*	90 rpm*	90 rpm√	

Table 7. Statistical difference between test conditions, NS = not significant,  $\checkmark$  = p < 0.05, \*=p < 0.01.

Radar plots of selected muscle activities displayed in millivolts against pedal position (clockwise rotation) at a cadence of 90 rpm. The difference between vibration and no vibration conditions are shown below (Figure 12). These data display both the significant increase in the EMG amplitude and the timing of the increases of the main locomotive muscle groups during stationary cycling.



Figure 12. Radar Plots of muscle activity during the duty cycle, vertical axis = mV,. The red line = vibration, blue line = no vibration.

Comparison of total muscle activities between vibration and non-vibration conditions are shown in Figure 13. The non-vibration protocol resulted in a linear relationship with a coefficient of determination indicating that 99.5% of the variation in the percentage increase in EMG results from increasing the cadence. The vibration protocol resulted in a curvilinear relationship was modelled with a second order polynomial trend line yielding a coefficient of determination of 99.9%.





The relative differences between the increases observed as the cadence increased at each time interval, during both vibration and no vibration, were calculated and expressed relative to the cadence dependent vibration frequency are shown in Figure 14 The initial responses increased at a linear rate until a vibration frequency of 27.5 Hz, after which a dramatic increase from the previous linear trend was noted. The relationship between the difference in the percentage increase in total EMG and the increasing frequency was modelled with a second order polynomial trend line. The coefficient of determination for the trend line indicates that 96.3% of the variation in the difference between percentage EMG increases can be explained by the increasing frequency.



Figure 14. Relative increase in mean EMG activity ± SD compared to increasing vibration frequency.

The results of the analysis of the isokinetic power analysis of the powerBIKE crank resistance, i.e. the power in Watts required to turn the pedals at set cadences, with and without vibration are displayed in Figure 15. Both vibration and non-vibration display a linear increase in power with increasing cadence. The calibration was only performed once; therefore it is not possible to calculate variances. The non-vibration power to cadence relationship has a coefficient of determination of 99.8%, the vibration setting coefficient of determination indicated that 98.9%. At all cadences the power required to pedal during vibration is slightly higher than twice that required during non-vibration.





The EMG amplitude obtained at increasing cadences and power levels is displayed in Figure 16. The initial stages of the vibration protocol can be seen to be a continuation of the final stages of the non-vibration protocol. The non-vibration data has a high correlation between power and EMG amplitude, with a coefficient of determination indicating that 99.1% of EMG amplitude is accounted for by increasing power. Although the first three data points in the vibration data set have a strong linear relationship (r= 0.996), the final data points of the vibration data set deviate from the linear trend creating a second order polynomial relationship with a coefficient of determination where 98.1% of the EMG amplitude variation is accounted for by increasing power required to turn the pedal crank.



Figure 16. EMG amplitude  $\pm$  SD relative to power. The red line = vibration, blue line = no vibration.

# 3.4 Discussion

These preliminary data show that the addition of mechanical vibration during cycling produce significant increases in muscle activation of the major lower limb muscles. Studies have suggested that vibration training initiates changes in muscle length with concomitant muscle spindle activation, eliciting the 'tonic vibration reflex' <sup>97</sup> and that during vibration, there is an increase in motor unit recruitment resulting in faster muscle activation <sup>98</sup>. However, before conclusions are drawn there are some key issues which should be

addressed. To date there is no data regarding the vibration at any other location than the crank itself. The nearest contact point for the participants is the pedals, yet there is currently no information about the frequency or amplitude of vibration at this location. The lack of knowledge about the parameters at user interface points i.e. pedal, seat and handlebars, is a major limitation with regards to drawing conclusions about neuromuscular changes resulting from the addition of vibration.

One area which must be addressed is the increased resistance caused by the addition of the mechanical vibration mechanism. As the frequency of the vibration mechanically generated it there is a perfect linear relationship between increasing frequency and increasing cadence. However, Figure 15 clearly shows that the vibration mechanism also results in increased resistance, with a strong linear relationship. Therefore, potentially the vibration itself did not result in any neuromuscular changes. Figure 16 confirms that the increase in EMG amplitude with vibration reflects the higher power required to turn the crank once the vibration mechanism is turned on. The relationship between EMG amplitude and increased cadence, resistance and vibration frequency is curvilinear, yet as the cadence, resistance and vibration frequency is EMG and in EMG cannot be solely attributed to one of the parameters.

The results indicate that at lower pace cycling (60-80 rpm) the increases obtained during cycling with and with vibration are linear and proportional. However, cadences within this range are deemed as a more economical rate reserved for higher demand phases of competition e.g. uphill cycling <sup>99</sup>. The typical preferred cadence of professional cyclists is approximately 90 rpm <sup>99</sup>. Interestingly this value is the only data point which deviates from the linear response observed in the datasets obtained. As there are not enough additional data points in this region it is not possible to establish if either a) this point is an exponential change or an 'elbow point' change in the data set, or b) if increases beyond the current range will be linear or curvilinear.

Figure 14 displays the relative increase in EMG compared to increasing frequency and clearly indicates the rapid change in EMG activity around 27 Hz vibration. Unfortunately to best of the authors knowledge there is no other studies considering a range of vibration frequencies during cycling for comparison. Potentially 27 Hz could be a key frequency for obtaining neuromuscular changes. However, as previously stated the influence of cadence

and power cannot be distinguished from increasing frequency, therefore further studies are required to confirm this hypothesis.

While the data obtained within the scope of this study cannot confirm or deny a vibration induced influence in the neuromuscular activity of the locomotor muscles. Given the additional data obtained during the scope of this study further experiments could be designed to establish the true influence of the addition of mechanical vibration during cycling.

# 3.5 Summary

This study confirms the hypothesis that the vibration mechanism of the powerBIKE increases the myoelectrical activity of the main locomotor muscles. The total muscle activity data appears to initially show a linear increase, before a non-linear increase in activity at 90 rpm (equal to 30 Hz vibration frequency). The underlying cause for the increase cannot be established as the key parameters assessed are proportionally related.

The unavoidable mechanical relationship between power, cadence and vibration frequency is therefore a major limitation on any future research considering more than one cadence. As the powerBIKE is still currently under development, feedback has been provided to Power Plate Itd who has advised that changes will be made to the design of the prototype tested in this investigation. Based on the now obsolete status of the powerBIKE prototype further research within the scope of this PhD will focus solely on the whole body vibration platform.

# CHAPTER 4: THE EFFECT OF WHOLE BODY VIBRATION ON LOWER LIMB TISSUE OXYGENATION

#### 4.1 Introduction

Recent years have seen an increasing popularity of whole body vibration (WBV) platforms as an exercise modality and have been the focus of much scientific research. Recent reviews indicate that WBV is effective in increasing reflex and muscle activity in athletes, older adults and those with compromised health <sup>10</sup>, increasing muscle power and reducing pain<sup>8</sup>. To function effectively muscles need a supply of oxygen at the appropriate pressure and quantity <sup>100</sup>. Typically, the volume of oxygen held within the blood of a healthy adult is 20.1 mL of oxygen per 100 mL of blood <sup>101</sup>. To date there have been few studies investigating the effect of WBV on tissue blood flow and oxygenation parameters. Nakamura et al.<sup>102</sup> were one of the first research groups to report that vibration exercise has different blood flow responses to occupational vibration. The observation that blood flow was increased to the digits of the hand was attributed to a vasodilatation response of the peripheral blood vessels to vibration. Laser Doppler studies have shown that the application of both local vibration <sup>103</sup> and WBV during isometric weight-bearing exercise <sup>71</sup> significantly increased skin blood flow without subsequent vasoconstriction during the recovery period of 10 minutes. Kerschan-Schindl et al. <sup>70</sup> reported a 100% increase of blood flow in the popliteal artery (from 6.5 to 13 cm $\cdot$ s<sup>-1</sup>), corresponding to Lythgo et al. <sup>72</sup> who found an increased mean blood cell velocity in the femoral artery following WBV. Previously Hazell et al. <sup>104</sup> reported no difference in the femoral artery from WBV in addition to Button et al. <sup>105</sup> who found local vibration did not affect blood flow. Yamada et al. <sup>106</sup> considered the influence of vibration on blood flow to the vastus lateralis during isometric squats, with results indicating vibration induced greater depletion of oxygenated haemoglobin (Hb) and myoglobin (Mb) during squats and increased Hb/Mb after completion of the squats. Conversely, Cardinale et al. <sup>73</sup> investigated the effects of vibration during a static squat on vastus lateralis and medial gastrocnemius oxygenation, however no statistically significant results were found. Though it should be noted that Yamada utilised an oscillating platform and Cardinale utilised a synchronous vibration platform. To date there have been limited studies considering the effect of vibration platforms on muscle oxygenation. More recently Coza et al. <sup>74</sup> investigated gastrocnemius muscle oxygenation during heel raise exercise in arteriolar occluded (AO) conditions with respect to performance and recovery, both of which are dependent on blood flow. The results indicated that vibration increased oxygen utilisation during arterial occlusion and increased the recovery rate for both occluded and non-occluded conditions.

## 4.1.2 Measurement of tissue oxygenation

Techniques for measuring tissue oxygenation originated in the 1950's where electrodes were placed in to tissues of interest using glass pipettes, though it has been reported that technical aspects of the approach was somewhat difficult <sup>107</sup>. In the current scientific environment there is a variety of techniques to measure tissue oxygenation. As the designs of electrodes have improved it is possible to obtain measurements via direct insertion of catheters for oxygenation and partial pressures <sup>108</sup>, arterial-venous samples can provide location specific or difference measurements of Hb saturation <sup>109</sup> and blood samples or diverted flow can be analysed for metabolites (such as lactate, pyruvate or phosphocreatine) by NADH fluorescence meters <sup>109</sup>. All of these techniques require invasive procedures to either implant sensors or to obtain blood samples. To circumvent this issue scientists have employed Near Infra-Red Spectroscopy (NIRS) to measure blood flow and oxygenation. Quantification of NIRS parameters is achieved via the application of a differential path-length factor (DPF) with the Lambert-Beer law <sup>110</sup>. NIRS has been shown to provide valid, non-invasive measurements regarding tissue oxygenation parameters <sup>111–114</sup> which are highly correlated with those of strain gauge plethysmography <sup>115</sup> yet have the advantage of obtaining results in a single process without the participant experiencing discomfort <sup>116</sup>.

# 4.1.3 Near infra-red spectroscopy

NIRS units function via an emitter and receiver which are placed on specific regions/muscles of interest and provide information on combined arteriolar, capillary and venular Hb concentrations <sup>117</sup>. The physiological parameters are calculated via changes

detected in the wavelengths of the emitted light signals (see Figure 17) after light absorption by local blood and tissues e.g. skin and skeletal muscle.



Figure 17. Diagram of NIRS emitter and receiver, adapted from Lima and Baker <sup>118</sup>.

Hb and Mb absorption spectra overlap and as such are indistinguishable with NIRS<sup>112</sup>, however, it has previously been reported that 90% of the NIRS signal is influenced by Hb and the remaining 10 % to Mb<sup>119</sup>. Hb absorption at wavelengths of 760–800 nm is highly correlated with saturation of oxygen and changes in local tissue perfusion yet only has a marginal influence of skin blood flow<sup>120,121</sup>. The level of absorption for oxygenated and deoxygenated Hb is equal at a wavelength of 800 nm, though at 760 nm the majority of absorption is in deoxygenated Hb allowing prediction of changing oxygen saturation during monitoring<sup>120</sup> (see Figure 18).



Figure 18. Representative changes in the wavelength of the emitted light (a). b Displays the corresponding change in oxygenation saturation. Figure taken from Liu et al.<sup>121</sup>.

The levels of signal changes obtained are dependent on both oxygen delivery and rate of use <sup>122,123</sup>. The ratio of oxygenated Hb to total Hb provides an index of tissue saturation known as the Tissue Oxygenation Index (TOI), which is expressed as a percentage <sup>111,124</sup>. The final parameter associated with NIRS signals is the Normalised Tissue Haemoglobin Index (nTHI) which is a measure of the total Hb in the tissue and is therefore highly influenced by changes in blood flow <sup>74</sup>.

The aim of this study was to investigate the influence of WBV on skeletal muscle tissue oxygenation parameters during simple dynamic movements i.e. heel raises, in order to establish fundamental physiological changes that occur. The hypothesis of this study was that acute increases in blood flow during dynamic exercise with WBV vibration will protect peripheral blood volume levels and that resulting differences are detectable by NIRSderived muscle oxygenation parameters.

## 4.2 Methods

#### 4.2.1 Participants

The format for this investigation was a randomised cross over study design. This study was carried out in accordance with University Ethics Guidelines and the ethical standards of the Declaration of Helsinki. All participants gave informed consent and received familiarisation of the procedure before data collection. Twenty physically active subjects (14 male, 6 female, age 29  $\pm$  10.4 years, height 1.75  $\pm$  0.09 m, weight 76.2  $\pm$  17.2 kg), with no recent history of lower limb musculoskeletal disorders or peripheral vascular problems were selected for inclusion in the study.

## 4.2.2 Study design

All heel raise exercises were performed on a Power Plate pro6 (Power Plate Ltd) whole body vibrating platform (40 Hz 1.9 mm vertical displacement), with either NVIB or VIB being utilised in ten alternating sets of 15 heel raises each. The initial set for each participant was randomised (VIB or NVIB). The exercises were completed using a metronome operating at 1 Hz to ensure all exercises were completed at the same pace. The participants' were instructed to move at a pace of 0.5 Hz i.e. one second up on to toes to maximum heel raise and one second down to complete flat foot and to ensure each repetition was a full heel raise i.e. as far up onto their toes as possible. Participants' were also instructed to keep a light bend on their knees, equivalent to approximately 3-5°. During straight leg heel raise activity although the soleus muscle contributes to the movement, the prime activity comes from the gastrocnemius which is mechanically better positioned to generate full power while the knee is extended compared to when flexed <sup>125,126</sup>. To reduce confounding factors from differing levels of cushioning in shoes participants were asked to remove shoes and socks during testing.

# 4.2.3 Data collection and processing

Tissue oxygenation parameters were obtained using a NIRO 300 (Hamamatsu Photonics, Japan), the emitter and recording sensor were placed on the right lateral gastrocnemius with the central distance between the emitter and detector 1/3 of the distance between the head of the Fibula and the Calcaneus. While there are no specific guidelines on placement of the location of NIRS sensors, the guidelines for placement of EMG sensors<sup>95</sup> typically occur over the widest part of the muscle belly, therefore are also suitable for NIRS sensors. A constant distance of 4 cm was maintained between the emitter and the detector. Analogue output to via a USB AD board allowed synchronous oxygenation and motion data capture. One retro-reflective marker was placed on the right lateral Malleolus and tracked for 60 seconds at 20 Hz to determine ankle motion (Oqus3, Qualysis AB, Sweden).

Marker motion was tracked and all synchronous data exported in .c3d format for subsequent analysis in Visual3D (C-Motion). Motion data was filtered (6Hz, 4<sup>th</sup> order low pass Butterworth filter), maximal and minimal vertical displacements were defined from which vertical ankle displacements were determined as well as total exercise time. Voltage calibration was used to convert oxygenation signals to appropriate values; these data were then smoothed using a 0.2 Hz 4<sup>th</sup> order low pass Butterworth filter. All signals were baseline corrected relative to the first 5 seconds of data prior to initiation of the exercise. Maximal or minimum values during the exercise period were used to determine absolute

concentration changes for deoxyhaemoglobin ( $\Delta$ HHb), oxyhaemoglobin ( $\Delta$ O2Hb), total haemoglobin ( $\Delta$ cHb), and tissue oxygenation index (TOI) and normalised tissue haemoglobin index (nTHI). The magnitude of change in signal parameters was assessed via the slope of the TOI and the nTHI graphs as these figures are representative of all signal parameters. The slope for the TOI during the normalised exercise period was calculated via the following equation:

Equation 1: TOI slope = 
$$\frac{\Delta TOI}{\Delta time}$$

The pattern of change for nTHI was different from TOI, with an initial decrease returning to baseline after completion of the exercises. Therefore the slope for the nTHI graph was calculated by the following equation:

Equation 2: 
$$nTHI$$
  $slope = \frac{(THI_{end} - THI_{minimum})}{\Delta time}$ 

# 4.2.4 Statistical analysis

Mean values of the five VIB and five NVIB repetitions were determined for each participant and group mean data are presented as means ± SEs. Data were checked for normality (Shapiro-Wilk test) and between-conditions analysed using a Paired-Samples T-Test. Cross correlation analysis was completed to identify any relationship between movement and the cyclical changes observed in some of the oxygenation parameters. Gender differences were assessed using one way ANOVA. Statistical significance was set at alpha = 0.05 for all tests. PASW Statistics 18 software (IBM Corporation, USA) was used for statistical analysis.

# 4.3 Results

No significant differences were observed in vertical ankle displacements (NVIB:  $9.7 \pm 0.4$  cm, VIB  $9.2 \pm 0.02$  cm) or in the time taken to complete each set of exercises (no vibration 29.4 ± 0.2 s, vibration: 29.3 ± 0.3 s). Peak changes in NIRS muscle oxygenation parameters during heel raise exercises between NVIB and VIB conditions are shown in Table 8.

Table 8. NIRS tissue oxygenation parameter changes (mean ± SE).

Parameter	NVIB	VIB
Δ HHb (μM)	13.7 ± 1.4	14. 1± 1.5
Δ O2Hb (μM)	-18.1 ± 1.3	-14.4 ± 1.4 ✓
Δ cHb (μM)	-8.1 ± 1.1	-3. 7± 1.0 ✓
nTHI (a.u.)	-3.3 ± 0.5	-1. 6± 0.5 ✓
TOI (%)	-18 ± 1.0	-16.9 ± 1.0
TOI Slope (% s <sup>-1</sup> )	-0.7 ± 0.05	-0.6 ± 0.05

✓ = significant differences between no vibration and vibration conditions (P<0.001).

Aggregate time series data representing increasing  $\Delta$  HHb and decreasing  $\Delta$  O2Hb profiles are shown in Figure 19 and Figure 20 respectively, which together are characteristic of tissue hypoxia. NVIB and VIB conditions produced very similar  $\Delta$  HHb profiles with no significant difference in absolute concentration changes. However, the  $\Delta$  O2Hb profiles showed a higher O2Hb depletion during exercise during NVIB. During VIB O2Hb depletion was significantly reduced relative to NVIB (P < 0.001).



Figure 19. Δ HHb profile (mean and SE). Solid line indicates no vibration and dotted line vibration conditions. Vertical dashed lines indicate start and end of the exercise protocol.



Figure 20.  $\Delta$  O2Hb profile (mean and SE). Solid line indicates no vibration and dotted line vibration conditions. Vertical dashed lines indicate start and end of the exercise protocol.

Aggregate time series data representing  $\Delta$  cHb and nTHI profiles are shown in Figure 21 and Figure 22 respectively. During exercise in both NVIB and VIB conditions the time series data show similar patterns with an initial rapid decrease in response to the onset of exercise, a slow but linear recovery during the exercise, followed by a rapid increase in both  $\Delta$ cHb and nTHI ending in levels above the pre-exercise baseline. The decrease in blood volume (as indicated by  $\Delta$ cHb and  $\Delta$ nTHI) during VIB was less than half of that seen during NVIB (P < 0.001).



Solid line indicates no vibration and dotted line vibration conditions. Vertical dashed lines indicate start and end of the exercise protocol.

In addition to the significant difference in the level of signal reduction, the nTHI also displayed a significant difference in the response of the signal during exercise (see Figure 22). In NVIB condition the reduction in signal was consistent, however during the VIB condition the signal displayed a trend towards returning to baseline levels.



Figure 22. nTHI profiles (mean and SE).

Solid line indicates no vibration and dotted line vibration conditions. The blue arrows represent the nTHI slope Vertical dashed lines indicate start and end of the exercise protocol.

The raw nTHI data displayed an inverse synchronous cyclic pattern with a high correlation to ankle motion (Figure 23). The highest level of cross-correlation coefficient (r = 0.473) occurred with a lag difference of only 0.6 s.



Figure 23. Comparison of nTHI and ankle vertical displacements. The black line represents vertical ankle displacements; the blue represents changes in nTHI. Orange arrows indicate synchronicity.

Aggregate time series data representing TOI profiles are shown in Figure 24. Similar profiles were observed in both NVIB and VIB conditions with an initial linear decrease in tissue oxygenation from the onset of exercise and a corresponding linear increase following cessation of exercise. A higher level of tissue desaturation was observed in NVIB compared to the VIB, however the absolute changes from baseline to minimal TOI values did not reach significance nor did the TOI slope of the initial linear rate of change in TOI.



Figure 24. TOI profiles (mean and SE). Solid line indicates no vibration and dotted line vibration conditions. The blue arrow represents the TOI slope. Vertical dashed lines indicate start and end of the exercise protocol.

When investigating gender difference one way ANOVA analysis only highlighted differences in  $\Delta$  HHb and nTHI slope (see Table 9), with females producing significantly less HHb during both NVIB and VIB conditions in comparison to male participants.

Parameter	Gender	NVIB		VIB	
Vertical displacement	male	0.1 ±	0.01	0.1 ±	0.003
	female	0.1 ±	0.003	0.1 ±	0.003
Duration	male	29.4 ±	0.3	29.3 ±	0.4
	female	29.6 ±	0.1	29.5 ±	0.1
Δ O2Hb	male	19.5 ±	1.6	15.7 ±	1.7
	female	14.4 ±	1.3	11.1 ±	1.2
ΔHHb	male	15.7 ±	1.6	16.1 ±	1.7 🖌
	female	8.9 ±	0.9 *	8.9 ±	0.5
Δ CHb	male	-8.1 ±	1.2	-2.9 ±	0.9
	female	-8.0 ±	2.4	-5.8 ±	2.5
ΔΤΟΙ	male	19. ±	1.4	17.7 ±	1.3
	female	15.6 ±	1.3	15.0 ±	1.3
ΔTHI	male	-3.1 ±	0.7	-1.1 ±	0.7
	female	-4.5 ±	0.8	-2.9 ±	0.6
TOI slope	male	-0.7 ±	0.07	-0.7 ±	0.06
	female	-0.5 ±	0.04	-0.5 ±	0.06
nTHI slope	male	0.2 ±	0.02 *	0.2 ±	0.03
	female	0.1 ±	0.02	0.1 ±	0.03

 Table 9. Gender comparison for all parameters (mean ± SE),
 \* = significant difference between genders.

#### 4.4 Discussion

The results obtained indicate that there were significant differences in tissue oxygenation resulting from the addition of whole body vibration to heel raise exercise. Whilst NIRS does not precisely measure blood flow, changes in Hb levels are indicative of changes in blood volume in the area assessed <sup>112,127,128</sup>. Variation in Hb levels changes can also be an indication of oxygen delivery and utilisation in non-occluded conditions <sup>129</sup>.

The changes in cHb observed in this study suggest an initial decrease in blood volume/flow, potentially a consequence of 'start up costs' of exercise, followed by an increase in blood volume/flow. The differences observed in cHb, nTHI and O2Hb suggest that the addition of vibration during exercise reduces the depletion of these measures. Since the nTHI is based on an assessment of an unknown path length the actual tissue volume assessed is not known, therefore the units obtained are arbitrary units. However, the relative level of changes are an indication of changes in blood volume or blood flow, with potential to

discriminate between arterial (decreased nTHI) or venous (increased nTHI) occlusions <sup>130</sup>. During exercise decreased levels of available oxygenated Hb in conjunction with increased levels of deoxygenated Hb are indicative of local muscular hypoxia; therefore any intervention which alters these parameters has potential to influence local muscle hypoxia. The pattern of change observed in nTHI is very similar to that reported by Coza et al. <sup>74</sup>, who investigated the effect of vibration in arterially occluded and non-occluded blood flow in the lower limb. The results indicated changes in nTHI parameters, which were attributed to increased blood flow.

The absolute decrease in in TOI was less than that observed in the study of Yamada et al. <sup>106</sup>, though different muscles were analysed (lateral gastrocnemius vs. vastus lateralis). However, the pattern of changes in TOI was again very similar. These results also reflect previous research which has shown the rate of blood flow in the popliteal artery doubles during vibration exercise <sup>70</sup>. The increase in blood flow potentially explains the lower levels of depletion in oxygenated Hb found in this study. Influx of blood to exercising muscles brings additional Hb and, in the absence of occlusion, will influence the recorded levels of oxygenated Hb.

This increase in blood flow and volume will also influence tissue saturation and therefore TOI <sup>131</sup> which may explain the lack of a significant difference observed between the exercise conditions. The similar  $\Delta$  HHb profiles observed during exercise with and without vibration suggest that the mechanical and metabolic costs of each protocol were essentially the same. The only parameter noted to have a significant difference was  $\Delta$  HHb between male and female participants. It could be suggested that female subjects were subjected to less mechanical work (due to lower body masses), or that gender differences in subcutaneous adipose tissue thickness affected NIRS signals <sup>132</sup>. However, further work in this area is required to determine the relative impact of this on each of the NIRS signals.

Interestingly the inverse relationship between changes in the levels of nTHI and position of the ankle suggests that systematic drops in HB occur at the point in time when the ankle is at its highest point i.e. at the peak of muscle contraction. This decrease could either be indicative of maximum use of Hb at the point when the muscle is working hardest, or reduced blood flow due to the muscle working isometrically for a brief period at the point of maximum heel raise. It should also be considered that a potential consequence of vibration exercise is a shift in the type of fibres being utilised to perform the exercise from

type II fibres to type I fibres resulting in greater increases in HHb levels which was not observed. In order to address these questions the exercise protocol could be repeated whilst recording electromyographic activity to provide a greater insight into the possibility of muscle fibre activity.

Previous studies have investigated the role of the gastrocnemius contraction as a muscle pump in relation to blood flow and venous return. The validity of muscular contractions influencing venous return remains a controversial issue with many authors reporting no influence and suggestions that increased blood flow is more likely to be a result of increased vasodilatation <sup>133–136</sup>.

It should also be considered that a more distal response, such as vasospasm in the feet, could result in a resistance to blood flow from the calves. Recently Thompson et al. <sup>137</sup> reported a case of occupational exposure resulting in vasoconstriction in the feet but not the hands; diagnostic testing indicated normal ankle brachial indices but reduced digital plethysmographic waveforms in the toes when exposed to cold. However, it should be noted that the case study was based on a miner with 18 years of exposure. While this potential mechanism has not been fully investigated and to the author's knowledge never investigated in response to WBV exercise, this type of local response could explain the results obtained.

A final explanation to consider is that of a global response to vibration. Previous investigations of the extremities have indicated that local vibration to the hand has resulted in changes in circulatory disturbances of the foot <sup>53,138</sup>. These changes have been attributed to a central sympathetic vasoconstrictor reflex elicited by vibration <sup>53</sup>. These findings partially confirm the earlier work of Greenstein and Kester <sup>54</sup> who investigated the effect of unilateral hand vibration with and without a nerve block. They found that in the majority of cases a bilateral response suggestive of a sympathetic vasoconstrictor reflex in the absence of a digital nerve block was obtained. When the nerve block was administered the response was absent and vasodilatation was noted. However, it should also be noted that in some subjects vasodilatation was noted without the nerve block. The authors concluded that acute vibration may elicit both a vasoconstrictor reflex and an active local vasodilatation, with both mechanisms competing against each other. In 2002 Schweigert <sup>139</sup> conducted a systemic review to establish if competing hypothesis regarding the underlying mechanisms

for circulatory disturbances of the extremities in response to vibration could be developed into an established theory. Unfortunately the studies were found to have significant validity flaws such as lack of independent variables, selection and survivor bias, the assessment of confounding exposures and the lack of blinding of investigators. The ultimate conclusions were that there is some evidence for lower limb vascular symptoms (cold induced vasospasm) to be associated with Hand Arm Vibration Syndrome (HAVS) but not in workers exposed to vibration without HAVS. The effect on local vascular structures has also been investigated by assessing pulse wave velocity and blood pressure at the ankles as an estimate of peripheral arterial stiffness. While it is difficult to distinguish between peripheral and central arterial stiffness, it was suggested that by obtaining measurements at the ankles peripheral influence would be greater <sup>67</sup>. Results obtained indicated that arterial stiffness reduced approximately 20 minutes after WBV exercise and lasted for up to 40 min. The authors proposed that this may reflect vasodilatation via vascular endothelial function. This is supported by evidence that vibration during cycling resulted in an increase in the release of vascular endothelial growth factor <sup>68</sup> which could result from increased shear stress in blood vessels, as mathematically modelled by Yue et al. 63-64.

#### 4.5 Summary

The results obtained indicate that the addition of vibration to heel raise exercise did not increase the metabolic cost of completing the exercise for the lateral gastrocnemius muscle. However, the addition of vibration during exercise does decrease the reduction in local muscle oxygenation parameters, potentially indicating less reduction in tissue blood volume and/or increased blood flow, this pattern of responses is indicative of reducing exercise induced tissue hypoxia. Nonetheless, it is important not to over interpret these results. To date it has not been fully established if the observed changes are a direct result of increased blood flow to the leg, or a consequence of a vasoconstriction response in the feet creating blood pooling effect in the legs. Without confirming the influence of WBV on blood flow in the feet the hypothesis that WBV protects depletion in peripheral blood volume cannot be confirmed.

Assessing the level of lower limb muscular activity can also be achieved by measuring the volume and frequency of electrical activity of the musculature during exercise. By repeating

the protocol adopted during this study and measuring electrical activity greater insight into the influence of vibration on the lower limb could be achieved. Further studies should be undertaken to investigate these potential explanations prior to conclusions being formed and exercise/rehabilitation recommendations being issued.

# CHAPTER 5: THE INFLUENCE OF WHOLE BODY VIBRATION ON THE CENTRAL AND PERIPHERAL CARDIOVASCULAR SYSTEM

# 5.1 Introduction

It is well documented that occupational vibration at high frequencies, above 100 Hz, is potentially hazardous to health<sup>89</sup>. Risks include; structural damage to blood vessels, reduction in blood supply, venous insufficiency, nerve damage leading to paraesthesia and in severe cases arthritis and Vibration White Finger Syndrome <sup>140,141</sup>. It has, however, been suggested that at low amplitude (0-5 mm), low frequency (5-50 Hz ) WBV using specifically designed exercise equipment, may have the potential for enhancing exercise <sup>142</sup>. Typically WBV investigations have been used for short durations, with cumulative exposure of up to 15 min, to investigate influences on the central cardiovascular system. These studies have reported statistically, though not physiologically, significant increases in oxygen consumption and heart rate <sup>143</sup>, significant increase in both systolic and diastolic blood pressure (BP) <sup>144</sup>, significant increases in diastolic blood pressure only <sup>145</sup> and no significant changes in blood pressure <sup>67,146</sup>. Despite the conflicting results from central cardiovascular system investigations, other studies have reported additional cardiovascular benefits, even in the absence of significant changes in heart rate and blood flow, in the form of the attenuation of the increases in leg and abdominal aortic arterial stiffness following acute WBV and reduced blood pressure following repeated WBV exercise sessions 67,145,147,148. Although to date there is no conclusive evidence, it has been speculated that changes in arterial stiffness are a direct result of WBV induced oscillations creating mechanical stimuli on blood vessels combined with changes in vascular endothelium function <sup>67</sup>. However, the reported effects of WBV on peripheral blood flow velocity (BFV) are inconclusive, with some studies indicating an increase in BFV <sup>71,97,149</sup> and others reporting decreases or no change after vibration <sup>104,105</sup>. Peripheral blood flow has also been shown to significantly increase in response to increase in temperature <sup>150</sup>, which has been shown to increase in response to WBV <sup>151</sup>. The relationship between peripheral temperature and peripheral blood flow has been attributed to 'thermoregulatory peripheral vasoconstriction' <sup>150</sup>. Vasoconstriction, or a reduction in the cross sectional area of a blood vessel, is a physiological mechanism to control the rate and volume of blood flow. The opposite of

vasoconstriction is vasodilatation <sup>152</sup>. An increase in the cross sectional area of a blood vessel decreases the physical resistance and leads to an increase in blood flow, a decrease in cross sectional area increases resistance, decreases blood flow and potentially results in blood flow being fully or partially redirected to other blood vessels <sup>153</sup>. This redistribution of blood is one possible explanation of an underlying mechanism for the reduction in depletion of blood observed in the investigation detailed in Chapter 4. The potential for vibration to induce vasoconstriction is not a positive prospect; this in itself could become a risk factor for patients with peripheral vascular conditions. Therefore the area requires further research to establish the influence of WBV on peripheral blood flow.

# 5.1.1 Measurement of blood flow

When measuring blood flow velocity the technique utilised is typically achieved with either strain gauge plethysmography, Ultrasound Doppler, Laser Doppler, LED sensors such as NIRS units or photoplethysmography (PPG). Strain gauge plethysmography is typically applied to the forearm or calf of participants. Blood flow to the limb is controlled via an inflatable cuff. Inflation pressures of 50mmHg are sufficient to occlude the blood flow in veins but not the arteries <sup>154</sup>. A strain gauge approximately 5-10% smaller in diameter than the forearm/calf at its widest point is placed around the area to be assessed. The strain gauge is filled with a conductive fluid e.g. mercury and attached to electrodes to enable an electrical current to be passed through. As blood flows to the area an increase in diameter changes the resistance of the strain gauge allowing calculation of the blood flow to the area <sup>155</sup>. The results from strain gauge plethysmography have been used to validate NIRS, with results being highly correlated <sup>115</sup>, though it has been reported to have the advantage of obtaining results in a single process with the participant experiencing discomfort <sup>116</sup>.

#### 5.1.2 Laser Doppler measurements

Ultrasound Doppler is available in two forms:

#### 1. Transcutaneous Ultrasound Doppler

Doppler ultrasound relies on the principle defined by Johann Doppler, in which the frequency of a waveform is dependent on the velocity of movement between the source and observer of the sound or light generated. With regards to ultrasound, the

sound signal reflected from the red blood cells which will change depending on the blood flow velocity and the angle between the emitted signal and the blood flow (see Figure 25) <sup>156</sup>.

#### 2. Intravascular Doppler

The process can also be applied invasively the insertion of a microprobe into blood vessels unavailable or difficult to record from via transcutaneous ultrasound was tested. The results provide measurements using a 0.3 mm probe inserted via a 5F catheter typically used in angiographic procedures <sup>157</sup>. The results correlated well with transcutaneous measurements where comparisons were possible.



Figure 25. Representation of Laser Doppler measurement of blood flow velocity. As the velocity of the blood flow increases, the frequency of the reflected ultrasound signal increases. Using the change in frequency and the angle of reflection (Θ) the blood flow velocity is calculated.

Laser Doppler measures on singular blood vessels are highly accurate. However, when multiple blood vessels are located in close vicinity of each other the signal can be influenced by multiple reflections of the different blood vessels. In this instance multiple emitters and detectors within the ultrasound probe can be used to employ a pulsed wave analysis where the backscattered signal is analysed at a time relative the emitted signal <sup>158</sup>. Technically this approach is not utilising the Doppler effect, the detected change is in the

position of the backscattered signal, not a shift in the frequency of the signal, though the probes are generally still referred to as Doppler probes <sup>158</sup>.

Laser Doppler measurements provides can provide measurements including: blood flow velocity, in both forward and reverse flow, the ratio between forward and reverse flow in relation to the duration of a cardiac cycle (pulsatility index), and the ratio between the forward flow at the start and end of each cardiac cycle (resistance index). Resistance index can indicate changes in peripheral resistance via vasodilatation or vasoconstriction of capillaries in distal muscles <sup>70</sup>.

NIRS units are also based on an optical sensor system as reviewed in Chapter 4. An additional optical system used for estimation blood flow velocity in the superficial or dermal blood vessels is known as photoplethysmography (PPG) or venous photoplethysmography (VPPG). PPG is a low cost technique that focuses on venous function in the micro-vascular bed <sup>159</sup> and works in a similar way to NIRS, where a light signal is emitted through the skin and changes in wavelength allow calculation of parameters such as blood flow velocity, depletion of blood in micro-vascular and levels of oxygenation <sup>120</sup>.

The aim of this investigation was to identify the influence of WBV on the central or peripheral cardiovascular system during quiet standing WBV whole body vibration exposure. In order to address this question, the effects of acute WBV participant's heart rate, blood pressure, skin temperature in the lower leg and foot and BFV in the foot were investigated.

The hypothesis for the study was that the vasospastic responses in the feet resulting from vibration exposure created peripheral resistance resulting in blood pooling in the lower limb.

#### 5.2 Methods

#### 5.2.1 Participants

This study was carried out in accordance with University Ethics Guidelines and the ethical standards of the Declaration of Helsinki. All participants gave written informed consent. Twenty participants (12 male 8 female, age 24  $\pm$  3 years, height 1.74  $\pm$  0.09 m, weight 66  $\pm$  10 kg, calf girth 0.36  $\pm$  0.03 m) with no recent history of illness or lower limb musculoskeletal disorders, peripheral vascular problems or contraindications to vibration exposure were recruited for the study.

#### 5.2.2 Study design

The format for this investigation was a randomised repeated measures study design. Participant's cardiovascular parameters including: peripheral skin temperature, peripheral venous function, BFV in the dorsalis pedis artery, blood pressure, heart rate were assessed on two separate sessions, with at least 24 hours rest between data collection. Subjects avoided caffeine, alcohol and exercise for 24 hours before both test days in order to prevent any excitatory influences on the cardiovascular system. The study was divided into two separate phases. Test one (T1) consisted of the collection of measurements for blood pressure, micro-vascular blood volume depletion and temperature, whereas test two (T2) determined the BFV and heart rate. Participants experienced 5 min of vibration (1 min on 1 min off), on both testing days, as shown in Figure 26. Following vibration an additional 4 min of recovery data collection was completed.



Figure 26. Chapter 5 data collection procedure.

#### 5.2.3 Data collection and processing

During T1 participants sat with no shoes or socks for 10 min to acclimatise to ambient room temperature. Thermocouple temperature probes (Grant instruments, Cambridge UK) were then attached to the skin over two muscles with blood supplied by the anterior tibial artery; tibialis anterior (TA), peroneus longus (PL) and two distal muscle in the foot; extensor hallucis brevis (EHB) which has blood supplied from the lateral tarsal artery, an extension of the anterior tibial artery and the adductor hallucis (AH) muscle (see Figure 27).



Figure 27. Position of thermocouple sensors.

These muscles were selected due to their blood being supplied proximally connected to the dorsalis pedis artery. The rationale for the selections of these locations being that should vasoconstriction occur, blood would be shunted to proximal muscles creating changes on temperature. Finally the abductor hallucis muscle (AH), which is further distal on the foot and supplied by the medial plantar artery, was measured to monitor changes in the extremity.

Peripheral venous function was assessed via venous photoplethysmography (VPPG) using a Rheo Dopplex II photoplethysmograph (Huntleigh Diagnostics, Cardiff, UK) with the sensor placed 0.1 m above the medial malleolus. VPPG parameters included venous drainage (VD) and half-amplitude time (HAT), a reported measure of venous refilling time. Systolic and diastolic blood pressures (BP) were measured using an Omron M3 automatic blood pressure monitor (Omron, Kyoto, Japan).
The venous function data collection protocol required participants to sit motionless while VPPG signal stability was obtained; the participants then moved their foot in and out of dorsiflexion in time with a computer generated audio and visual signal which was used to promote consistent movements resulting in 10 'foot taps'. This process was repeated 3 times consecutively, following the manufacturer's guidelines and is postulated to effectively lower the levels of local venous blood.

After the movements were completed the participants again sat still while the recovery patterns were measured. The participants where then placed in a standing position on a Power Plate pro6 whole body vibration platform (Power Plate Ltd) for a period of 5 min to allow stabilization of orthostatic pressure, in order to obtain baseline measurements, skin temperature, and blood pressure.

Once the values were recorded, 5 sets of 60 s of vibration (40 Hz 1.9 mm vertical displacement) with 60 s recovery were introduced. Skin temperatures were carried out in each set after 30 s of vibration. Blood pressure was obtained, at the mid-point of the rest periods, and temperature was recorded 30 s in to the rest period. This process was repeated throughout the intervention process. It was not possible to collect venous function data during the vibration section of the protocol as the vibration interfered with the signal acquisition. Once the final 60 s of vibration were completed the participant was immediately seated, and blood pressure, venous function and skin temperature was reassessed.

In T2 the participants followed the same two acclimatisation periods (sitting and standing); however during the standing acclimatisation baseline heart rate was recorded using a Polar Heart Rate monitor (Polar FT1, Warwick UK). For normalisation, heart rates were expressed as a percentage of the individuals predicted heart rate maximum (HRmax) estimated by Equation 3<sup>160</sup>.

# **Equation 3:** *HRmax* = 207 - (0.7 x age)

Once the standing acclimatisation was completed baseline blood flow measurements were taken from the dorsalis pedis artery using a Rheo Dopplex II with an 8 MHz laser Doppler probe. The location of the dorsalis pedis artery was identified medial to the navicular bone as detailed in <sup>161</sup>, BFV and resistance index were simultaneously obtained. Blood flow measurements were taken during the rest periods between vibration exposures and every minute during the recovery phase.

# 5.2.4 Statistical analysis

All data was collated in Microsoft Excel and exported to MatLab (MathWorks, USA), where Lilliefors test for normal distribution was used to identify whether parametric or nonparametric analysis should be implemented.

Data that was normally distributed is displayed as means  $\pm$  1 standard deviation (SD), data that was not normally distributed is displayed as median  $\pm$  1 median absolute deviation (MAD). For Grouped data pooled variance (PV<sub>SD</sub> or PV<sub>MAD</sub>) is used to display the level of variance across the group using the Equation 4.

Equation 4: *Pooled Variance* 
$$(PV) = \sqrt{\frac{\sum_{i=1}^{k} (n_i - 1)\sigma_i^2}{\sum_{i=1}^{k} (n_i - 1)}}$$

Where n = the number of results in a group/repeated measure and  $\sigma$  represents variance (SD or MAD depending on data distribution).

For testing between conditions, where data was normally distributed a repeated measures ANOVA was applied and the Friedman's analysis of variance test was used for data that was not normally distributed. Where significant differences were found Wilcoxon matched pairs analysis was completed on each stage compared to baseline, with P values adjusted using the false discovery rate method <sup>96</sup>. To assess the impact of variance on significant results effect sizes were calculated using matched pairs biserial correlation coefficient previously detailed <sup>162</sup>, with each stage being compared to the baseline values. Significance was set at alpha = 0.05 for all tests.

Equation 5: matched pairs biserial correlation coefficient =  $\frac{4\left|T - \left(\frac{R_+ + R_-}{2}\right)\right|}{N(N+1)}$ 

Where  $R_+$  and  $R_-$  are the positive and negative ranks respectively, T is the smaller of the two values and N = the number of pairs of scores. The results are classified as small =  $\leq 0.2$ , medium  $0.3 \geq 0.7$  and large =  $\geq 0.8$ .

# 5.3 Results

Initially there were twenty participants in the study; however, only seventeen were available to complete both testing procedures. The following results are therefore based on data obtained from these participants. Heart rate data was the only normally distributed data set, all other variables were found to have non-Gaussian distributions.

The results for peripheral temperature did not display significant differences throughout the duration of testing. There were no significant differences in the level of change in skin temperature at any of the locations recorded. Median skin temperatures  $\pm$  1 PV<sub>MAD</sub> were: TA = 33.7  $\pm$  0.03°, PL = 33.3  $\pm$  0.04°, EHB = 30.4  $\pm$  0.04° and AH = 29.3  $\pm$  0.06°.

The results obtained for blood pressure and venous function are displayed in Table 10. The blood pressure displayed some variance between subjects, as indicated by the pooled variance. However the within subject differences did not change, as indicated by a lack of significant differences in median values for standard blood pressure values at any stage in of the intervention.

The results for venous function indicate that the volume of blood displaced displayed large variance between subjects, as indicated by the pooled variance. However, as with blood pressure, the within subject changes did not change, as indicated by a lack of significant differences in median values.

Table 10. Blood pressure and venous function results presented as medians + 1 PV<sub>MAD</sub>.

	Blood Pressure (mmHg)		Venous Function	
	Diastolic	Systolic	VD (au)	HAT (s)
Baseline	122 ± 9	77 ± 7	30 ± 9	18±5
Vibration	121±8	75 ± 7	N/A	N/A
Recovery	123 ± 12	72 ± 9	29 ± 9	15 ± 5

The results obtained for the mean heart rate normalised as a percentage of predicted HRmax are displayed in Figure 28. Despite a slight increased variance after the second period of vibration both the mean %HRmax was highly consistent throughout the intervention with no significant differences occurring at any time point. The mean heart rate and %HRmax being 81.6  $\pm$  14.6 beats min<sup>-1</sup> and 42.9  $\pm$  6.3% respectively.



Figure 28. Heart rate as mean percentage of predicted maximum ± 1 SD.

The results obtained for BFV in the dorsalis pedis artery are displayed as a smoothed scatterplot in Figure 29. BFV was significantly different from baseline values following the first three vibration exposures only; the effect sizes for these differences were medium, large and large respectively. There was no significant difference between the resistance indexes of the BFV. The median blood flow velocities  $\pm 1 \text{ PV}_{MAD}$  were  $0.5 \pm 0.2$ ,  $1.0 \pm 0.2$  and  $0.5 \pm 0.2 \text{ cm} \cdot \text{s}^{-1}$  during baseline, vibration and recovery stages respectively. The resistance indices of the BFV were  $1 \pm 0.06$ ,  $0.99 \pm 0.02$  and  $0.99 \pm 0.01$  during baseline, vibration and recovery stages respectively.



Figure 29. Median blood flow velocity of dorsalis pedis artery ± 1 MAD. Horizontal dotted line represents the velocity at baseline, \* indicates significant differences from baseline.

#### 5.4 Discussion

The current study was designed with the absence of movement, allowing any significant changes to be solely attributed to the introduction of vibration. Previous studies using WBV have shown changes in systolic blood pressure <sup>144,145</sup> and increases in heart rate <sup>143</sup> in response to vibration. However, comparison of results cannot be directly made as the studies referenced were involved muscle contractions, both static and dynamic and longer durations of exposure.

The primary findings of this study were the significant changes that were observed in the BFV of the dorsalis pedis artery. BFV is influenced by both central and peripheral factors such as: heart rate, blood pressure, muscle temperature and cross sectional area of local vasculature <sup>150,153</sup>. The data show no significant changes in the central cardiovascular parameters of heart rate or blood pressure. There were also no significant differences observed at any of the four locations where skin temperature was measured, though typical temperature on the foot were lower than that observed in the calf. The difference in temperature is likely explained by the reduced density of local muscle tissue in the foot compared to the leg. The lack of change in temperature is in contrast to previous reported values <sup>104</sup> where measured skin temperatures 0.025 m superior to the lateral malleolus of the left ankle during static squats reported a maximum increase of 2°. However, it should be noted that this was following a much longer period of 15 min of WBV exposure than the 5 min used in the current study. While differences in skin temperature did not display significant changes throughout the protocol, it cannot be assumed that this indicates no change in muscle temperature. Cochrane *et al.*<sup>151</sup> compared the effects dynamic squats during WBV, cycling and soaking in a hot bath and showed that intramuscular temperatures rose by 2° for all conditions, with no concomitant change in skin or core temperatures.

The results obtained for peripheral venous function using VPPG were not significant. However, during data collection immediately after vibration exposures the VPPG system required 90 - 120 s before sufficient signal stability was obtained to allow measurement. Previously venous function investigations using strain gauge plethysmography indicated improved venous drainage <sup>163</sup>. Therefore it could be suggested that if changes in venous function were occurring immediately post recovery, the fluctuating results interfered with VPPG sensors. Based on this hypothesis significant changes were potentially missed. However, if this were the case the significant changes were transient and therefore unlikely to have a significant physiological effect on healthy participants. In addition, the fact that the BFV resistance index did not change infers that there was no reduction in peripheral resistance via widening of capillaries in distal muscles <sup>70</sup>.

Analysis of the peripheral BFV was completed with each repeated measure being compared to the baseline value. The first three values obtained following the vibration exposures, were significantly increased from the baseline value with the greatest effect sizes seen after

the second and third exposures. Of all measures obtained the data following the second exposure displayed the largest variance, equivalent to approximately 50% of the median value obtained. However, this ratio is equivalent to values for baseline femoral artery BFV reported in Lythgo et al.<sup>72</sup>, who reported increases of over 300%. Hazell et al.<sup>104</sup> also considered femoral blood flow, during a minute on minute off protocol totalling 15 min, resulting in the mean femoral blood flow doubling, though with particularly high variance. Kerchan-Schindl *et al.*<sup>70</sup> investigated the effect of WBV on popliteal artery blood flow over 9 min, the results displayed doubling of BFV from 6.5 to 13.0 cm·s<sup>-1</sup>. Lohman *et al.* <sup>71</sup> examined calf skin blood flow over three minutes of vibration, significant changes were only observed following direct skin vibration, as opposed to standing on the platform performing semi squats. The Lohman et al. concluded these changes were a consequence of mechanical friction upon cutaneous cells, potentially causing greater nitric oxide circulation resulting from the pulsatile stresses. These physiological responses could explain the occurrence of erythema previously reported <sup>70,144,164</sup>. Erythema is an additional example of reported changes in peripheral venous function following the introduction of vibration. However, participants often quickly adapt and after two or three sessions of WBV no longer display signs of erythema<sup>144</sup>. In the current study no participants showed signs of erythema. Rittweger et al.<sup>144</sup> used laser Doppler techniques to measure skin blood flow on the calf and foot, but did not state exactly where on the foot the measurements were taken. The reported increases were 1.8 - 2.8 times that of baseline values. However, as these were arbitrary units and cutaneous not arterial measurements, the absolute values detailed were not directly comparable to results in the current study. It should also be noted that squats were performed until exhaustion rather than quiet standing adopted in the current study.

Considering the results obtained in the current study did not display significant changes in heart rate, blood pressure or resistance index, it can be inferred that changes in BFV can be attributed to changes in vascular cross sectional area prior to the distal capillaries or the influence of muscular contractions proximal to the extremities. However, further research needs to be completed in order to confirm this hypothesis. Considering only the first 3 of the 5 vibration exposures resulted in significant differences in peripheral BFV and no recovery BFV measurements resulted in significant differences, further studies should also be completed with greater volumes of vibration exposure.

#### 5.5 Summary

The results from this investigation indicate that the peripheral cardiovascular system is more sensitive to vibration than the central cardiovascular system. However, the exact physiological processes which control vibration induced vasoconstriction have yet to be established and it is likely to be a combination of the cardiovascular system and both central and peripheral neural mechanisms <sup>165,166</sup>. While the lack of changes in resistance index and VPPG suggest that there were no micro-vascular changes influencing the BFV, peripheral macro-vascular, i.e. blood vessels > 150  $\mu$ m in diameter <sup>167</sup>, changes are not accounted for. It should be noted that the increase in popliteal artery blood flow velocity reported by Kirchan-Schindl <sup>70</sup> was accompanied by an increase in popliteal artery diameter.

Overall the results of this investigation do not support disprove the hypothesis that WBV results in vasospastic responses in the feet. While direct measures of macro and microvasculature diameter were not made, vasospastic responses would have resulted in increased resistance index and decreased BFV. Therefore the underlying mechanism of the reduced depletion in blood volume observed in Chapter 4 is unlikely to have been caused by vasospastic responses in the feet. However, as the BFV in the dorsalis pedis artery was increased, it is likely that the BFV in the proximal blood vessel was also increased and influencing blood volume in the lower limb. The results obtained are of particular interest to exercise and rehabilitation practitioners and researchers; though it is advised that further studies should also be completed in participants with circulatory dysfunction before any form of clinical applications are adopted.

# CHAPTER 6: EFFECT OF WHOLE BODY VIBRATION DURING STATIC SQUATS ON THE MYOELETRICAL PROPERTIES OF THE VASTUS LATERALIS

# 6.1 Introduction

The investigations reported in Chapters 4 and 5 indicate that WBV influences muscle tissue oxygenation parameters and extremity blood flow. Previous publications have indicated that WBV increases blood flow <sup>70</sup>, however, if isometric contractions are preventing the perfusion of blood into muscles via increased intramuscular pressure <sup>168,169</sup> it can therefore be hypothesised that the muscular contractions can reduce the perfusion of local blood in to the active muscles. It has previously been reported that changes in parameters of tissue oxygenation affect EMG <sup>170</sup>, via investigation into the changes in NIRS and synchronous EMG resulting from WBV during isometric squats. A significant correlation was found between the decrease of the mean power of the frequency (MNF) and the decreased tissue oxygenation recorded synchronously in human vastus lateralis muscles.

#### 6.1.1 Parameters of electromyography

Analysis of myoelectrical signals is typically completed in either the frequency or the time domain. The time domain allows calculation of temporal parameters such as the onset, duration and offset of the myoelectrical signal. The time domain also allows measurement of the volume of the activity i.e. the signal amplitude and the amount of power of the within signal i.e. the root mean square (RMS). The frequency domain indicates how a signal can be decomposed into a range of its constituent frequencies. Within EMG research these frequency ranges are often summarised by two measures of central tendency, the mean (MNF) and median (MDF) frequencies. To date research on the effect of vibration on EMG frequency has been equivocal, with reports of the MNF decreasing in lower limb muscles <sup>171</sup>, increasing MDF of the vastus lateralis during squats <sup>144</sup> and increasing MDF of vastus lateralis and rectus femoris during knee extensions following vibration <sup>128</sup>. Reports of muscle fatigue are typically reported as either the mean or median value of the total frequency spectrum.

The equations used to calculate MNF and MDF are as follows:

Equation 6: 
$$MNF = \frac{\sum_{j=1}^{M} f_j P_j}{\sum_{j=1}^{M} P_j}$$

Where  $f_j$  represents the frequency of the spectrum j points along the x axis,  $P_j$  represents the EMG power at point j and M represents the maximum value on the x axis. The MNF is representative of the volume of power from the frequency spectrum in a given period of time e.g. one second.

Equation 7: MDF = 
$$\sum_{j=1}^{MDF} P_j = \sum_{j=MDF}^{M} P_j = \frac{1}{2} \sum_{j=1}^{M} P_j$$

The MDF represents half of the total power in the frequency spectrum. The power in the spectrum is therefore divided in to equal amounts covering the range of frequencies below and above the MDF. Both MNF and MDF highly correlated to independent measures of fatigue <sup>172</sup> generally the MNF is slightly higher than MDF <sup>173</sup>. The MDF is also less affected by random noise and therefore muscle fatigue has a greater influence on MDF than MNF <sup>173</sup>. MDF is also indicated to be superior to the MNF when considering changes in muscle fibre conduction velocity (CV) <sup>174</sup>. Calculating CV is achieved via cross correlation analysis of EMG signals obtained from electrodes placed along the length of a muscle fibre <sup>175</sup>. The cross correlation algorithm calculates the lag in the signal which is used on conjunction with the known distance between the recording electrodes to calculate the conduction velocity. As the basis for the calculation is the signal lag and the known distance between the electrodes there are two key factors when undertaking CV analysis. The first is to ensure that during set up the electrodes are aligned with the muscle fibre orientation, this can be achieved via imaging equipment or array electrodes which detect the direction of signals along muscle fibres and secondly ensuring there is an exact known distance between recording electrodes, Figure 30 displays representative EMG signals used for calculation of CV.



Figure 30. Representative EMG signal for calculation of muscle fibre conduction velocity across array of EMG sensors.

# 6.1.2 Cardiovascular influences on electromyography

The influence of blood flow on CV has previously been investigated in ischemic muscles, where the authors indicated that maintenance of blood flow for removal of by-products of muscular contractions, such as lactic acid, is vital in order for CV in the gastrocnemius and soleus to remain stable <sup>176</sup>. The relationship between CV and the frequency content of EMG has also been studied for many years. Previously studies focused on animal musculature <sup>176</sup> progressing to more recent studies based on human participants <sup>177</sup>. Throughout this period studies have consistently indicated a relationship between CV and frequency content. In an early attempt to ascertain the exact relationship between CV and frequency content the influence of local warming of muscle, in the absence of exertion, against fatigue inducing contractions was compared <sup>178</sup>. The results indicated both approaches were capable of inducing change in frequency content; however contractile action had a much greater influence on CV. The authors inferred this to indicate that there must be factors other than CV influencing the frequency content of myoelectrical activity. In 1987 Zwarts *et al.* <sup>179</sup> investigated the relationship between CV and frequency changes in short duration contractions followed by recovery with and without induced ischemia. The results

indicated different recovery patterns for CV and MDF. MDF recovered fully in all subjects but CV only partially recovered. The authors therefore concluded that blood flow influences CV but not MDF. The changes in CV of vastus lateralis during different contraction types (isometric vs. dynamic) has also been considered <sup>180</sup>, where isometric contractions resulted in significant decreases in CV whilst dynamic contractions did result in any significant change. Both contractions resulted in significant increases in amplitude and decreases in MDF, though not significantly different from each contraction type. Based on these findings the authors concluded that metabolic state has a greater influence on CV than on MDF or amplitude and that changes in MDF cannot be explained by CV alone. Based on these findings it can be proposed that the combination of myoelectrical signal frequency and CV analysis will provide the option of identifying influences on contractile function.

The results from Chapter 4 indicates that WBV influences tissue oxygenation of the lateral gastrocnemius, unfortunately pilot studies indicated that obtaining the conduction velocity of the lateral gastrocnemius did not yield reproducible results, potentially due to the changing orientation of gastrocnemius muscle fibres during contraction. The vastus lateralis is a key locomotor muscle, similar in fibre type ratio to that of the lateral gastrocnemius <sup>181</sup>, which has been previously studied for CV, as such the focus of this study was on the vastus lateralis. The aim of this study was to investigate the influence of WBV on myoelectrical activity during isometric squat exercises to provide an insight into the physiological consequences on muscular contractile activity of adding vibration to exercise. The hypothesis for the study is that decreased depletion of oxygenated haemoglobin will protect CV, but not MDF.

#### 7.2 Methods

## 7.2.1 Participants

This study was carried out in accordance with University Ethics Guidelines and the ethical standards of the Declaration of Helsinki with all participants providing informed consent. Twelve male participants ( $25.4 \pm 4.0$  years,  $1.81 \pm 0.1$  m,  $82.2 \pm 10.7$  kg) with no recent history of illness or lower limb musculoskeletal disorders volunteered for the study.

#### 7.2.2 Study design

The format for this investigation was a randomised cross over study design. All exercises were performed on a Power Plate pro6 (Power Plate International Ltd) whole body vibrating platform. Six alternating sets of 50 s unloaded isometric partial squats were performed with either no vibration (NVIB) or whole body vibration (VIB; 40 Hz 1.9 mm vertical displacement). The initial squat condition (NVIB vs. VIB) was randomised for each participant.

#### 7.2.3 Data collection and processing

Myoelectrical activity was acquired from the vastus lateralis with an EMG-USB system (OTbioelettronica, Torino, Italy) using an 8 sensor EMG surface array with an interelectrode distance of 5 mm. To identify the optimal location for array placement, a reference line was first drawn on the surface of the thigh aligned between the anterior superior iliac spine and the top of lateral pole of the patella. A second line representing muscle fibre orientation (FO) was drawn from the top of the lateral pole of the patella  $20^{\circ}$ relative to the reference line. This process has been defined by and thoroughly reported by Beck et al. <sup>182</sup> who used anatomical measures of muscle fascicle orientation to establish the technique. Prior to placement of the EMG array the region between the innervation zone and the distal tendon was determined. According to Rainoldi et al. 183 the innervation zone can be found on average at a distance of 94 mm from the patella. Therefore, the skin was shaved at a distance of approximately 100 mm along the FO line and cleaned with alcohol. Each participant was then asked to perform a sub- maximal muscle contraction whilst a 16 electrode silver bar electrodes (5 mm x 1 mm, inter electrode distance of 5 mm) was used to identify the innervation zone along the FO line. The innervation point was identified as the electrode position where signal reversal was observed (see Figure 31). Once the innervation zone was identified an appropriate area distal to this point was marked for recording vastus lateralis conduction velocities. The participants then undertook 5 min of stationary cycling at 70 rpm (50 W) as a pretesting warm up. Once this was completed the predetermined recording area was re-cleaned using alcohol after which the 8 electrode array was attached using double sided adhesive foam and 30 µl of hypoallergenic conductive gel pipetted into each electrode well to improve signal quality. Pilot testing indicated that the vibration platform caused significant noise contamination of the EMG signal. Therefore data was acquired from the first and last 5 seconds of isometric partial squats. During the VIB squats, 30 seconds of vibration was introduced after the initial data collection with the squat being maintained after vibration to allow for a second set of data collection. An additional 5 seconds before and after vibration was incorporated in to the protocol to allow the vibration platform to fully start and stop without influencing signal acquisition. EMG conduction velocities and amplitudes were calculated during sequential 0.5 s epochs using OTBioLab software (OTbioelettronica, Torino, Italy). Muscle CVs were determined using cross correlation analysis of high resolution alignment of sampled waveforms <sup>184</sup> from 3 of the 8 channels of the acquired data array.



Figure 31. Representative EMG signals used to identify the location of the innervation zone. The signals acquired via the electrodes central to the array detect signals smaller in amplitudes prior to the electrodes at each end of the array. The central electrodes also identify the point of signal reversal.

#### 7.2.4 Statistical analysis

The CV, amplitude and raw data signals were exported to MATLAB (MathWorks, USA) for additional analysis. The raw signals of the three channels used to calculate CV and amplitude were analysed using a custom written script to identify the MDF of each channel using FFT analysis. All results were tested for normal distribution using Lilliefors test, then significant differences were assessed using paired t-Test if the data was normally distributed or Wilcoxon signed ranks tests if the data was not normally distributed. For all statistical analysis significance was set at alpha = 0.05. Results of EMG parameters are presented as mean values and 1 SEM. Where significant differences were found Cohen's *d* effect size (Equation 8) was calculated using the following formula:

Equation 8: *Cohen's* 
$$d = \frac{(\overline{x}_1 - \overline{x}_2)}{\sigma_{pooled}}$$

Where  $\bar{x}$  represents the group mean, *i* the sample number, *k* the maximum number of samples and  $\sigma_{pooled}$  represents the pooled variance (Equation 9).

Equation 9: *Pooled Variance* = 
$$\sqrt{\frac{\sum_{i}^{k}(n_{i}-1)\sigma_{i}^{2}}{\sum_{i}^{k}(n_{i}-1)}}$$

Effect size was then adjusted for upward bias with Hedges g (Equation 10) using the following formula:

Equation 10: 
$$g = Cohen's d x \left(1 - \frac{3}{4(n_1 + n_2) - 9}\right)$$

Where *n* represents the number of samples in the group.

#### 7.3 Results

The signal amplitude data was not normally distributed; therefore the results were analysed using Wilcoxon signed rank test. As myoelectrical signal amplitude is typically variable amongst test days and test participants the signal amplitude was only analysed as the differences between the start and the end of the squats. During squats with vibration the amplitude increased by an average of  $1.3 \pm 0.6 \mu$ V, during squats without vibration the signal amplitude increased by  $2.0 \pm 1.4 \mu$ V these results are displayed in Figure 32. These increases were not found to be significant.



Figure 32. Difference in amplitude at the start and end of squats. Outliers, identified as being greater than the interquartile range from the median, are plotted as red crosses.

The MDF were found to be normally distributed; therefore the results were analysed using paired t-Tests. During NVIB squats the MDF at the start of the squats was 76.3  $\pm$  4.6 Hz and at the end of the squats the MDF was 76.4  $\pm$  4.5 Hz. This difference was not found to be significant. During VIB squats the MDF at the start of the squats was 77.6  $\pm$  4.8 Hz and 73.9  $\pm$  4.5 Hz at the end of the squats. This difference was found to be significant, although the effect size was found to be small (g = 0.2).

The rate of decline (final frequency – initial frequency) in MDF is displayed in Figure 33, this result was also found to be significant. Finally effect size difference in decline in MDF was calculated and found to be large (g = 0.8).



Figure 33. Difference in median frequency at the start and end of squats with and without vibration. Outliers, identified as being greater than the interquartile range from the median, are plotted as red crosses.

The CV results were normally distributed; therefore the results were analysed using paired t-Tests. During the NVIB condition the mean CV at the start of the squat was  $3.56 \pm 0.48$  ms<sup>-1</sup> and  $3.58 \pm 0.51$  ms<sup>-1</sup> at the end of the squat. These results were not found to be significantly different. During VIB the mean CV at the start of the squat was  $3.56 \pm 0.6$  ms<sup>-1</sup> and  $3.4 \pm 0.4$  ms<sup>-1</sup> at the end of the squat. This difference was also not significant. The amount of difference between the CV at the start and end of squats with and without vibration is displayed in Figure 34 which shows median change values, these differences were again not significant.



Figure 34. Difference in muscle fibre conduction velocity at the start and end of squats. Outliers, identified as being greater than the interquartile range from the median, are plotted as red crosses.

#### 7.4 Discussion

This study investigated the effect of static partial squats on myoelectrical activity of the vastus lateralis. Partial squats were employed studied <sup>185</sup>, comparing deep, parallel or partial squats. There was no significant difference between biceps femoris, vastus medialis or vastus lateralis EMG activity between the squat depths analysed. The EMG array placement reflects that previously reported <sup>182</sup>, with indications that specific and consistent placement of the electrode is required if the signals are to be compared across subjects. The recommended location of the sensor matches that previously reported <sup>186</sup>, where CV is most stable when obtained between the innervation zone and the myotendinous junction.

This region was also reported to produce the highest values for MDF, though the rate of change was not affected by sensor location. While previous studies such as that of <sup>182</sup>, have used the EMG array to analyse knee extension, static squats were selected for this study. This decision was based on two factors. Firstly it is impractical to add whole body vibration to knee extension as it is not practical to place resistance equipment on to the vibration platform and secondly it has previously been shown that squats produce both greater amounts of myoelectrical activity and greater reduction in MDF in the vastus medialis and lateralis compared to knee extension <sup>187</sup>. Shift in MDF has long been used as an indicator of fatigue during isometric contractions <sup>188</sup>. Previous investigation <sup>180</sup> considered the effect of both static and dynamic contractions on both MDF and CV of the vastus lateralis during knee extension; their results indicated that isometric extensions elicited greater changes in both MDF and CV, with no change at all in CV during dynamic exercise.

Over all the results in the current study indicate that the only significant differences obtained was the decrease in MDF. The fact that the MDF did not change in the NVIB condition suggests that the warm up was appropriate, as previously it has been shown that active warm up increases the MDF of exercising muscles and this effect relates to increases CV <sup>189</sup>. An important point to note is that while there was a significant decrease in MDF this is unlikely to be indicative of a physiologically fatigued state. There are two reasons which indicate this to be true. Firstly the reduction in MDF was 4.6%, previous studies inducing fatigue and measuring MDF have found the reductions to be much greater e.g. 10% decrease <sup>190</sup> 22.4% <sup>180</sup> and between 12-26% <sup>191</sup>. Therefore while the reduction in MDF observed is suggestive of the onset of physiological fatigue it is unlikely that the participants actually reached a fatigued state via this protocol. Secondly the effect size for the change in the vibration group was 0.2, which according to <sup>192</sup> is a small effect size. When comparing the amount of change in MDF between VIB and NVIB conditions the effect size was 0.8, indicating a large difference between the two conditions.

No significant differences were obtained in the CV although it could be argued that the level of contraction was potentially not high enough to induce a change. It has been suggested that a linear relationship between CV and MDF during isometric contractions at 40% of MVC exists <sup>179</sup>. However, further investigations on the effect of varying intensities of muscular contractions (as percentage of MVC) on changes in CV of both biceps brachii and

vastus lateralis identified thresholds of 20-30% MVC to increase CV, whilst 40% of MVC was identified to induce a reduction in CV. It was also reported that at all intensities CV is sensitive to changes in blood flow <sup>168</sup>. In contrast <sup>193</sup> investigated isometric contractions of the biceps brachii at 60% and 70% of MVC. In all participants the mean frequency and the CV were reported to vary linearly, both decreasing during sustained muscle contractions. This led the authors to conclude that frequency shifts occurring during fatiguing muscle contractions are a consequence of reduce CV. Additionally, previous research <sup>178</sup> considered two different influences on myoelectrical properties of the adductor pollicis muscle. Firstly a 60 second maximal voluntary contraction (MVC) was employed to induce fatigue and secondly muscle temperature was changed in the absence of fatigue. Both protocols induced similar changes in signal frequency. However, fatigue was found to have a much greater influence on CV than temperature. The baseline CV and amount of change is also dependent on muscle fibre type ratios. Participants with greater volume of fast fibres will have greater CV than participants with majority slow fibres <sup>194,195</sup>. This observation has been investigated via electrical stimulation whilst monitoring the level of intracellular free calcium <sup>196</sup>. It was found that a single electrical stimulation of fast twitch fibres would elicit an action potential and a rise in intracellular concentration approximately three times faster than slow twitch fibres. Based on these findings it was concluded that in fast twitch fibres calcium may be delivered at a faster rate to contractile proteins. This is likely to be the explanation for variances in baseline CV and MDF measurements. However, this is unlikely to influence the results obtained as the data analysis focussed on the change in parameters between the start and the end of the squats. Muscle fibre type can influence resistance to change on myoelectrical parameters. Finally, it should be noted that when comparing participants with varying ratios of fast and slow twitch muscle fibres it can take 25-30 knee extension repetitions before differences are observed <sup>191</sup>. The combination of these results refutes the hypothesis that WBV results in contractions generating intramuscular pressure preventing the perfusion of blood in to muscle tissue. The hypothesis of this study was based on results obtained in Chapter 4, where studies of the lateral gastrocnemius yielded significant reductions in depletion of oxygenated haemoglobin. Previous studies utilising NIRS on the vastus lateralis during isometric squats have yielded conflicting results with reports of decreased total haemoglobin <sup>106</sup> and no significant differences in total haemoglobin or in tissue

oxygenation index <sup>73</sup>. However, without considering the individual oxygenated and deoxygenated haemoglobin levels direct comparisons cannot be made.

# 7.5 Summary

In summary the results obtained indicates that acute exposure to WBV does not affect CV or amplitude of myoelectrical signals. The frequency content of the signal is statistically significantly reduced during squats with WBV; however this is unlikely to represent a physiologically fatigued state. These results, in conjunction with the findings of Chapter 4, provide further evidence for the hypothesis that WBV protects the CV but not the median frequency during partial isometric squats. However, as there is conflictions in the reported responses of tissues oxygenation parameters of the vastus lateralis during isometric vibration exercise <sup>73,106</sup>. The value of this information is that it indicates that WBV increases the muscular contractile activity during static squats without inducing fatigue. This finding is potentially beneficial for practitioners in rehabilitation, health and exercise who may incorporate vibration in to exercise protocols.

# CHAPTER 7: THE EFFECT OF WHOLE BODY VIBRATION DURING DYNAMIC MOVEMENTS ON THE MYOELECTRICAL ACTIVITY OF LOWER LIMB MUSCLES

# 7.1 Introduction

Recent meta-analyses have established the benefits of WBV for improvement of muscle strength <sup>5</sup> and power <sup>6</sup>, building on previous reviews which have illustrated the benefits in lower limb muscular performance and balance <sup>98</sup>. However, some of the fundamental biomechanical and physiological changes have still not been fully investigated to explain the underlying mechanisms of these changes. The results from Chapter 4 indicate that simple heel raise exercises completed with WBV influence tissue oxygenation parameters. The results from Chapter 5 suggest these changes are not a consequence of vibration induced vasoconstriction in the feet; therefore whether muscle contraction intensity or duration is changing must be questioned. The reduction of MDF observed in the results of Chapter 6 is an indication of increased muscle activity and potentially an early indication of the onset of fatigue. However, these results were based on a different muscle. To appropriately investigate the fundamental physiological and biomechanical changes in response to WBV the study completed in Chapter 4 must be repeated, whilst investigating additional parameters.

# 7.1.1 Assessing muscle function with electromyography

To address issues of this nature EMG provides a tool which is typically used to quantify the level of activation of muscles, the timing of the electrical activity driving muscular contractions, the force/EMG signal relationship and the use of the EMG signal as a fatigue index <sup>197</sup>. A fatigue index is typically where the MDF is tracked throughout contraction periods and compared to the initial MDF value <sup>197</sup>. Recently Pererira et al. <sup>198</sup> reported that heel raise activity not only displayed changes in the frequency of muscle activity as participants approached fatigue, but resulted in phase specific changes in the amplitude of the EMG signal as the participants approached fatigue i.e. increases in amplitude during the heel raising phase and reductions in amplitude during the lowering phase. The reduction in

MDF has previously been validated as an indicator of calf muscle fatigue during both heel raise exercises <sup>199</sup> and uphill running on a treadmill <sup>200</sup>.

#### 7.1.2 Assessing muscular response to vibration with electromyography

The effect of WBV on the soleus, gastrocnemius and vastus lateralis EMG was previously investigated during a four minute dynamic workout <sup>171</sup>. EMG signals were sampled at four set points during the exercise regime in one second intervals at which the participants was not moving. The results indicated that during WBV the MNF decreased in all muscles and the root mean square (RMS) increased in both soleus and gastrocnemius, but did not change in the vastus lateralis. In contrast static squat positions of high and low depth of squat and one legged squats have been shown to significantly increase in the RMS of the EMG signal of the rectus femoris, vastus lateralis, vastus medialis, and gastrocnemius in response to vibration <sup>201</sup>. The difference between static and dynamic squats during WBV was later considered by Hazel et al. 202. Results indicated amplitude increases of 4.8% of MVC in the vastus lateralis and 1.0% in the biceps femoris during static squats and 6.2% in the vastus lateralis 1.2% in the biceps femoris during dynamic squats <sup>202</sup>. A similar comparative study was later completed by Abercromby et al.<sup>12</sup> where results indicated equal or higher EMG amplitudes of vastus lateralis, biceps femoris gastrocnemius and tibialis anterior muscles during static squats with WBV compared to dynamic squats with WBV. The effect of quite standing during WBV with only slight knee bends, approximately  $15^{\circ}$ , was investigated by Pollock *et al.*<sup>203</sup>. The influence of both WBV amplitude (2.5 – 5.5 mm) and frequency (5, 10, 15, 20, 25 and 30 Hz) were investigated. Results indicated increases in EMG amplitude of soleus, lateral gastrocnemius, anterior tibialis, rectus femoris and biceps femoris ranging from 5 to 50 and from 5 to 20% MVC for amplitude and frequency changes respectively. The increases in EMG amplitude were most marked in the lower leg, with reasonably linear increases for soleus, gastrocnemius and anterior tibialis (r = 0.505, 0.446 and 0.596 respectively, P < 0.001). The rectus femoris and biceps femoris also increased linearly, yet with reduced linear correlation (r = 0.294 and 0.388 respectively, P < 0.05). Increased WBV amplitude always resulted in higher EMG amplitude, though the difference was not always significant. Increases in lower limb musculature has been attributed to vibration induced stretch reflexes, following comparison of EMG results obtained during both WBV and a custom built rig which introduced dorsiflexion movements <sup>204</sup>. Comparative analysis of EMG signals of soleus, gastrocnemius medialis and rectus femoris muscles suggested changes resulting during WBV area a result of stretch reflexes within the muscle. The theory of vibration induced stretch reflexes has also been investigated via the use of ultrasound to measure the temporal association between EMG activity and muscle contractile tissue displacement during quiet standing with only a slight knee bend <sup>19</sup>. The results indicate significant increases in the contractile length of the medial gastrocnemius muscle in response to WBV.

The review of current literature indicates that WBV results in increased EMG amplitude of the lower body during dynamic and isometric squatting exercises. However, to the best of the author's knowledge the influence of dynamic movements at the ankle in the presence of WBV has not yet been investigated. Heel raise exercise utilises the triceps surae muscles (gastrocnemius and soleus) which are easily accessible for surface EMG analysis. Though it should be noted that previous reports have indicated that the medial and lateral gastrocnemius muscles have different skin-fold thicknesses, with the lateral gastrocnemius being closer to that of the soleus muscle <sup>205</sup>. The medial gastrocnemius has also been shown to be more sensitive to changes in knee angles than the lateral gastrocnemius <sup>206</sup>.

The aim of investigating this hypothesis is to investigate the effect of WBV on EMG during simple dynamic human movements, such as heel raise exercise. Therefore, the hypothesis of this study is that vibration will increase the EMG activity of the lateral gastrocnemius and the soles muscle during heel raise exercise.

## 7.2 Methods

#### 7.2.1 Participants

Ten healthy male subjects (age  $27 \pm 5$  years, height  $1.78 \pm 0.04$  m, weight  $75.75 \pm 11.9$  kg), with no recent history of lower limb musculoskeletal disorders were selected for inclusion in the study, took part in this study and provided informed consent in accordance with University ethics guidelines.

#### 7.2.2 Study design and protocol

The format for this investigation was a randomised cross over study design. All heel raise exercises were performed on a Power Plate pro6 (Power Plate International Ltd) whole body vibrating platform (40 Hz 1.9 mm vertical displacement), with either NVIB or VIB being utilised in 6 alternating sets of 15 seconds during which heel raises were performed. The initial set for each participant was randomised (i.e. VIB or NVIB). The exercises were completed using a metronome operating at 1 Hz to ensure all exercises were completed at the same pace. The subjects were instructed to move at a pace of 0.5 Hz i.e. one second up on to toes to maximum heel raise i.e. as far up onto their toes as possible. Subjects were also instructed to keep a light bend on their knees to prevent excessive transmission to their heads. During straight leg heel raise activity the soleus muscle contributes, but a greater contribution comes from the gastrocnemius which is in a mechanically better position to generate full power compared to whilst the knee is bent <sup>125,126</sup>.

#### 7.2.3 Data collection and processing

Differential bipolar (10 mm centre to centre) surface electrodes (DE-2.3, Delsys Inc. Boston, MA, USA) were placed over the right lateral gastrocnemius and soleus muscles in accordance with SENIAM recommendations <sup>95</sup>. A single reference electrode was placed on C7 vertebrae and all leads connected to the electrodes were secured with tape to avoid artefacts from limb movements. Impedance was minimised by shaving and skin cleaning with alcohol swabs. EMG signals were amplified (1 k gain) via a Delsys Bagnoli system (Delsys Inc. Boston, MA, USA) with a bandwidth of 20-450 Hz. EMG activity was synchronously acquired with the kinematic data at 2000 Hz. Prior to undertaking any exercise MVCs were obtained in a seated position with the knee against a fixed resistance. Ankle motion was captured from a 16 mm retroreflective marker located on the right lateral malleolus at 500 Hz using 10 infrared retro-reflective cameras (Oqus, Qualysis AB, Sweden). Prior to capture a 46 m<sup>3</sup> volume was calibrated with a mean residual error of 1.6

mm. Marker motion was tracked and all synchronous data exported in .c3d format for subsequent post processing in Visual3D (C-Motion). Maximal and minimal vertical displacements were defined from which vertical ankle displacements were derived to define when the ankle was at the bottom or top of its movement cycles, the vertical displacement of the ankle and total exercise time.

EMG data were initially filtered using a 60 Hz cut-off 4th order bidirectional high pass Butterworth filter to remove any D.C. offset. A full rectification was applied before the signal was filtered with a 2 Hz cut-off, 2nd order bidirectional low pass Butterworth filter. Peak amplitudes, normalised to the MVC, of each muscle and timings for peak activity relative to movement onset were then determined.

Data sequences of 0.6 s centred on the peak EMG activity, 0.3 s before and 0.3 s after peak activity, were identified and exported for analysis in the frequency domain using an 'in-house' LabView virtual instrument (National Instruments Corporation). A Hanning window was applied to the data prior to fast Fourier transformation. Power spectra of the vibration EMG data were used to identify the dominant frequency due to the vibration 'noise'. A 4<sup>th</sup> order Chebyshev band stop filter was applied with low and high cut-off frequencies 2 Hz below and 2 Hz above the first vibration frequency as well as its 2<sup>nd</sup> and 3<sup>rd</sup> harmonics. The same process was then applied to the no vibration data and mean power frequencies calculated as the frequency centroid of the spectrum.

#### 7.2.4 Statistical analysis

EMG amplitude, timing and frequency data were exported to MATLAB (The MathWorks Inc., Natick, MA) and tested for normality using Lilliefors test, mean and standard error of the mean values were also calculated for each variable with a normal distribution. Data normally distributed was then tested for significant differences using paired t-Tests and non-normal distributions were analysed using Wilcoxon signed rank tests. Statistical significance was set at alpha = 0.05 for all tests.

#### 7.3 Results

No significant differences were observed in vertical displacement of the ankle (NVIB: 0.082  $\pm$  0.02 m, vibration: 0.079  $\pm$  0.002 m) or in the time taken to complete each heel raise (NVIB 1.96  $\pm$  0.02 s, VIB: 1.96  $\pm$  0.02 s) p > 0.05.

The mean, normalised amplitude of the lateral gastrocnemius did not display a significant change between VIB and NVIB conditions; however the soleus EMG amplitude was significantly increased during VIB heel raises. Figure 35 displays the mean EMG amplitude with the error bars representing the standard error of the mean. The sample distribution was not normal therefore Wilcoxon signed rank tests were applied to both soleus and gastrocnemius EMG amplitude data. The results for gastrocnemius VIB vs. NVIB conditions were not significant (p > 0.05), the results for soleus VIB vs. NVIB conditions were highly significant (p < 0.01).



The black bars represent VIB data, grey bars represent NVIB data. \* = significant difference.

A small increase in the median frequency was seen in both the lateral gastrocnemius (105  $\pm$  7 to 111  $\pm$  7 Hz) and in the soleus muscle (102  $\pm$  7.5 to 105  $\pm$  7.5 Hz) during the VIB conditions. The data distribution was normal therefore changes were analysed with paired t-Tests, results were not significant (p > 0.05). EMG activity during the movement cycle i.e. from when the heel was down through the heel raise back to the point when the heel was down is displayed in Figure 36 and Figure 37. The error bars represent the standard error of the mean. Each movement was paced via a metronome providing audible signals to aid participants a consistent movement rate of 0.5 Hz (average time for time for heel up and heel down movement during NVIB 1.96  $\pm$  0.02 s and VIB: 1.96  $\pm$  0.02 s). The introduction of VIB caused a small reduction in the time taken for participants to reach the peak EMG activity of the lateral gastrocnemius during the movement, from 75.3% of the time to the peak of the movement i.e. half the total movement, during NVIB to 72.8% with VIB. This difference was not found to be significant.



Figure 36. Gastrocnemius EMG.

Solid line indicates vibration and dotted line no vibration conditions. Vertical dashed lines indicate the point of maximum heel height. The black lines represent VIB data, grey lines represent NVIB data.



Figure 37. Soleus EMG.

Solid line indicates vibration and dotted line no vibration conditions. Vertical dashed lines indicate the point of maximum heel height. The black lines represent VIB data, grey lines represent NVIB data.

The soleus EMG activity also reached peak at an earlier time during the movement cycle (57.5% during VIB compared to 59.4% during NVIB) although the difference was again not significant. Interestingly the soleus EMG peak activity was consistently earlier than that of the peak gastrocnemius EMG activity. The data distribution was normal, therefore the difference between gastrocnemius peak EMG activity and soleus peak EMG activity was assessed via t-Tests yielding significant differences during VIB (p<0.05) and highly significant differences during NVIB (p<0.01). Figure 37 also reiterates that there is a significant difference in EMG amplitude in the soleus muscle during VIB and NVIB heel rises which was not found in the lateral gastrocnemius.

#### 7.4 Discussion

When considering the normalised amplitude of the EMG signal there are two points of interest. Firstly the amplitude of the signal for the lateral gastrocnemius actually decreased with VIB. It could be suggested that the standard error of the mean could account for the differences observed and potentially there was not a true change at all. However, there was a comparable standard error for the both conditions and both conditions had normal data distributions; therefore this explanation is somewhat unlikely. Secondly, while there was no significant difference between the amplitude of the gastrocnemius EMG signal during VIB and NVIB conditions, a significant increase in soleus EMG amplitude during VIB was noted. Based on these results it could be suggested that the increased output from the soleus muscle accounts for a greater proportion of the workload. As the physical workload, i.e. mass and amount of movement were not changed, the outcome of the increased workload of the soleus muscles could be reduced workload for the gastrocnemius muscles. If this is the case, it is likely that the lack of additional resistance during the exercise resulted in the gastrocnemius muscles simply not reaching the threshold needed for increased activity. Structurally the gastrocnemius and soleus muscles have been shown to have different fibre type distributions. The proportion of type I fibres in the soleus, medial gastrocnemius, and lateral gastrocnemius has been reported to be 75.2, 58.5 and 52.4% respectively <sup>207,208</sup>. These physiological differences are the most likely explanation for the different responses observed. It is also worth noting that the MVC technique selected for this study is unlikely to obtain a true MVC for the gastrocnemius muscles due to the amount of knee flexion, however as the focus of the data analysis was based levels of change within EMG normalised to a predetermined level - not absolute signal power; the fact that a maximal signal was not obtained should not be an issue. This is presuming that knee position affects EMG, though recently Hébert-Losier et al.<sup>209</sup> suggested that changes in knee angle of up to 45° only results in 4-5% changes in the level of EMG activity. It should also be noted that amending the position of the feet during heel-raise exercises will prompt varying degrees of medial gastrocnemius and lateral gastrocnemius activation <sup>210</sup>. Therefore although the exact position of the feet was not standardised in the protocol of this study, the participants were asked not to change the position of their feet once testing had begun.

The frequencies obtained agree with that previously reported in heel raise activity <sup>211–213</sup>. However, the total range of frequencies obtained was smaller. The smaller range of frequencies, combined with the lack of significant changes in the median frequency, indicate that neither muscle reached a fatigued state. This is not particularly surprising due to the low intensity of the testing protocol. The analogous reduction in median frequency for both muscles resulting from heel raise activity is also in line with that reported previously values <sup>211</sup>. Potentially, the slight drift in the median frequency observed in both the lateral gastrocnemius and the soleus could be attributed to fatigue of fast twitch but not slow twitch fibres. Slow twitch fibres are not only more suited to repeated exertions, but less likely to exhibit frequency changes during exertion <sup>214</sup>. It has also been shown that individual slow twitch fibres contain greater capillary density than fast twitch fibres in both soleus and gastrocnemius muscles<sup>215</sup>, providing greater support for repeated exertions. The slightly higher median frequency range for the lateral gastrocnemius compared to the soleus muscle could be explained by a greater proportion of fast twitch fibres within the gastrocnemius <sup>216</sup> compared to that of the soleus muscle. The lack of significant differences in results obtained from the lateral gastrocnemius is in contrast to that obtained via NIRS in Chapter 4, potentially suggesting that greater differences would have been obtained if the NIRS sensor was placed upon the soleus muscle. However further studies would need to be completed to confirm this hypothesis. Despite these speculations, the total reduction in frequency was not significant at the exercise intensity of this protocol indicating muscle fatigue was not present.

The timing of the peak activity during the heel raises suggests that the early part of the movement contains a combination of soleus and gastrocnemius muscle activity, after which the level of soleus muscle activity declines and the gastrocnemius muscle is the main contributor to the exercise. However, before firm conclusions are formed there are some additional factors that must be considered. One key area to be considered is the effect of electromechanical delay (EMD). The exact value of EMD was not measured within this study, however previously values have been reported as ranging from 41.9 to 77 ms in the upper limb <sup>217</sup>, if these values are applied to the data obtained in the current experiment that would equate to average EMD's of 5.9% of the time taken to for the ankle to reach peak height. Though Grosset *et al.* <sup>218</sup> reported lower limb EMD to be much lower, in the region of 8.3 ms on average depending on the type of training recently training undertaken

(plyometric or endurance training), based on these numbers the timing of the peak activity would only be out by 0.8% on average. Nordez et al. <sup>219</sup>also estimated a lower duration of EMD at 11.64 ms (providing an EMD of 1.16% to the current data). Nordez also reported that half of the EMD is due to the force propagation along the passive part of the series elastic component (i.e. tendon and aponeurosis). Therefore EMD is greatly influenced by the mechanical properties of the tendon and aponeurosis, which are known to be variable among subjects <sup>220</sup> and biomechanical factors such as ankle angle, participant flexibility, musculotendinous tension throughout the movement and musculotendinous stiffness <sup>221,222</sup>. A final area to consider with regards to EMD is the effect of fatigue. It has been shown that EMD is modified during a fatiguing task <sup>223</sup>. Therefore while fatigue was not achieved in the current protocol, the exact point at which EMD occurs during dynamic movement has not yet been identified. Ultimately based on the available information, particularly that of lower limb assessment, it is likely that if EMD's were present they were not significant enough to change the overall results. Particularly as the muscles considered share a common tendon in the Achilles.

Within the muscle groups there were not significant differences in the timings of peak activity. Subtle differences in the gastrocnemius muscles activity can be observed in figure 3, which is likely the cause of the different levels of significant difference between the peak soleus and gastrocnemius muscle activity during vibration and non-vibration comparisons. An additional point of interest is that during the last 5% of the movement i.e. the 'heel down' time, the level of muscular activity increased. This is likely due to pre-activation in anticipation of the next movement. Despite the potential deviations from reported values by influences such as EMD or pre-activation, the results obtained indicate that the soleus muscle reaches peak activity shortly after half of the heel rise, whereas the gastrocnemius muscle reaches peak activity at approximately three quarters of the time to maximum heel rise. The timings of these peaks are not significantly affected by WBV.

## 7.5 Summary

WBV in the absence of additional resistance results in significant changes in healthy populations. While the changes observed in the scope of this study were not the result of a fatigue inducing protocol, as indicated by the lack of change in EMG signal frequency, the ratio of plantar flexor muscle amplitude did change. Although there were no changes in the level of gastrocnemius muscle activity, greater levels of soleus muscle activity was found during whole body vibration. This is in contrast to the hypothesis that increases would be observed in both gastrocnemius and soleus muscles. The current study also identified the different phases of muscle activity during heel raise. Initially the movement is a combination of soleus and gastrocnemius muscle activity reduces whilst gastrocnemius continues to increase to a maximum at approximately three quarters of the time to maximum heel height. These findings help to provide a fundamental understanding of the action of the plantar flexor muscles during heel raise exercise and the influence of WBV on the lateral gastrocnemius and soleus muscles.

# CHAPTER 8: EFFECTS OF VIBRATION ON DYNAMIC AND STABILISER MUSCLE ACTIVITIES DURING THE PRESS UP

# 8.1 Introduction

To date there have been many studies considering the effect of whole body vibration (WBV) on the lower body, as reviewed by Rehn <sup>98</sup>, however yet there have been few investigations on the effects of WBV platforms on the upper body.

#### 8.1.1 Upper body vibration effects on EMG

Hazell et al. <sup>202</sup> considered the effect of WBV during static and dynamic squats and bicep curls on EMG of both the upper and lower body, with results indicating significant increases in signal RMS, yet this was not a consequence of direct vibration of the upper body. In a similar study Marin et al.<sup>224</sup> also reported significant increases in EMG activity of the biceps of older adults following isometric squats and bicep contractions via straps attached to a WBV platform. Gómez-Cabello et al. <sup>225</sup> investigated the effects of 11 week dynamic squats with WBV and found significant increases in both lower and upper body strength. McBride et al. <sup>226</sup> investigated the effect of a direct upper body vibration, via a vibrating dumbbell, on subsequent isometric contractions. The results indicated reduced EMG amplitude with an increased EMG frequency following vibration. Mischi and Cardinale<sup>227</sup> also investigated the influence of vibration during isometric upper limb exercise; reporting an increase in EMG RMS during vibration. However, subsequent muscle function was not analysed, making comparison of results difficult. Tripp et al. <sup>228</sup> considered the influence of a vibrating dumbbell at varying frequencies on elbow joint position sense; the results indicated both increased accuracy and decreased variability. While the investigations reported provide some insight into the influence of vibration on upper limb function, they do not provide an insight into multi-muscular or 'compound' exercises.

# 8.1.2 Press up exercise analysis

The press up is a commonly applied exercise to target the upper body for improvements of muscular strength, power and endurance <sup>229</sup>. The upper body musculature utilised during press ups can be split in to two groups. The dynamic muscles: pectoralis major, triceps and anterior deltoid <sup>229–231</sup> and the stabiliser muscles: latissimus dorsi, biceps, posterior deltoid,

trapezius (upper, middle and lower) and the serratus anterior <sup>229,230,232–235</sup>. Investigations of press up exercises have utilised EMG as a tool to examine press ups variations, such as: unstable surfaces e.g. hands placed on Swiss balls or basketball(s), altered hand and/or leg positions and altered hand and/or leg height <sup>231,234–236</sup>. However, care must be taken when altering the position or style of press up exercises. Modelling of compression forces in the lumbar spine during the various forms of press up have indicated that press up varieties such as amending hand position or speed of movement increases lumbar spine compressive forces by 20-37%, use of unstable surfaces creates increases of 25-55% and changing completing dynamic 'jumping' style press ups increases lumbar spine forces by 58-238% <sup>231</sup>. Therefore variations in press up exercises which do not rely on altering the standard position are potentially beneficial. In addition to standardising the position of the participant care must be taken to address technical aspects of the data collection process itself. The placement of the electrodes is a particularly important technical factor in study protocols. If electrodes are placed above the neuromuscular innervation zone the signal amplitude will be minimised <sup>237,238</sup>, if surface electrodes are placed too close to the tendon during analysis of dynamic motion the myotendinous movement under the surface of the skin can also relate in significant signal reduction <sup>239</sup>. While these studies are typically completed using surface electrodes, it is worth noting that muscle contractions and length changes have also been shown to move intramuscular electrodes <sup>240</sup>. The relative changes between electrode and muscle position is a particular concern since up to 17% of peak to peak amplitude of detected EMG signal has been shown to result from 'cross-talk' from neighbouring muscles <sup>241</sup>. Studies of optimal surface electrode positions have been published in order to provide evidence based practice to avoid these issues <sup>95</sup>. To the best of the author's knowledge, to date there is no published research on the effect of vibration during press ups. The aim of this study is therefore to consider the effect of using WBV platforms on press up exercise performance (posture) and muscular activity (EMG). The hypothesis of the investigation is that press ups with vibration will increase the muscular activation of dynamic and stabiliser muscles detected by EMG.

#### 8.2 Methods

#### 8.2.1 Participants

Nine healthy male participants (21.1  $\pm$  3.4 years, 1.68  $\pm$  0.03 m, 72.3  $\pm$  9.5 kg), experienced in recreational training and without any history of recent illness or injury volunteered for the study and provided informed consent in accordance with University Ethics Committee and the Declaration of Helsinki.

#### 8.2.2 Study design

Three press up conditions were tested on the WBV platform in a randomised cross over study design: press up in the absence of vibration (NVIB), low amplitude and frequency vertical vibration (30L; 1.2 mm, 30 Hz) high amplitude and frequency vertical vibration (40H; 1.9 mm, 40 Hz). Press ups were completed in a randomised order at a self-selected pace by participants for a period of 15 s with 3 min rest between each set. Participants were assessed for press up ability and familiarisation at least 24 hours before data collection. All press ups were completed in one testing session and all press ups were completed on the platform with feet resting on a bench of equal height to the platform.

#### 8.2.3 Data collection and processing

Prior to testing participants completed 4 min arm crank ergometery (Lode Angio, Groningen The Netherlands) as a warm up activity. Press ups were performed with the hands consistently placed at a distance 1.2 times the distance between the acromioclavicular joints. Press ups on a Power Plate Pro 6 whole body vibration platform (Power Plate Ltd). During press ups on the Power Plate the participant's feet were placed on a small bench to replicate the press up position of that on the floor. 3D motion capture was acquired using 16 mm retroreflective markers on the spine at C7 and S1, and acromion processes bilaterally. The sampling frequency of the motion capture was 500 Hz using 10 Oqus cameras (Qualisys, AB, Sweden). Maximal vertical displacements of the C7 and S1 were used to define cervical and pelvic vertical movements, the displacements between
the C7 and S1 markers were used as a measure of postural changes i.e. kyphotic or lordotic postures. Synchronous to motion capture data surface EMG (Delsys Bagnoli system, Boston, USA) was obtained via differential bipolar Ag-AgCl electrodes placed on the triceps (TR), pectoralis major (PM), serratus anterior (SA) and lower trapezius (LT) muscles in accordance with SENIAM recommendations <sup>95</sup>. Signal impedance and noise artefacts were minimised by skin preparation and taping of wires to prevent excessive movement during press ups. EMG signals were amplified (1 k gain) with a bandwidth of 20-450 Hz and a sampling frequency of 2000 Hz. A single reference electrode was placed on the skin level with the C7 vertebrae. All synchronous data was acquired using Qualisys track manager software (Qualisys, AB, Sweden) and exported in .c3d format for post processing in Visual3D<sup>tm</sup> (C-Motion, Inc. Germantown, USA). EMG signals were corrected for zero offset by subtracting the signal mean, fully rectified and smoothed using a 6<sup>th</sup> order Butterworth low pass filter with a cut-off frequency of 2 Hz. For direct comparison of averaged results between subjects, signals were time normalised from the start to the end of each press up based on the vertical position of the C7 marker. The mean signal amplitude for both the concentric and eccentric phase was calculated using the vertical displacement of C7 marker to identify the timing of each phase of the movement. The time normalised signal and mean values were then exported to MatLab (MathWorks, USA) for statistical analysis.

#### 8.2.4 Statistical analysis

The Anderson-Darling test showed that the kinematic data was not normally distributed, the mean EMG data, with the exception of the lower trapezius during the concentric phase and pectoralis major for the eccentric phase were all normally distributed. Therefore, median values were reported in place of means for the kinematic data <sup>242</sup>, with statistical dispersion represented as <u>+</u> one median absolute deviation (MAD) for kinematic data and standard error of the means (SEM) were used for mean EMG data. Friedman's test was used to test non-normally distributed conditions and repeated measures ANOVA was used for normally distributed data. The locations of significant differences were identified with Tukey's honest significant difference test to account for repeated measures analysis. In all analyses significance was set at alpha = 0.05.

For data that was normally distributed the effect size was calculated using Omega<sup>2</sup> ( $\hat{\omega}^2$ ) using Equation 11.

Equation 11: 
$$\hat{\omega}^2 = \frac{(k-1)(F-1)}{(k-1)(F-1)+kn}$$

Where F is the ANOVA F-value, N represents the number of participants and *k* represents the number of repeated measures.

For data that was not normally distributed the effect size was calculated using the coefficient of concordance (*W*) using Equation 12.

Equation 12: 
$$W = \frac{X^2}{N(k-1)}$$

Where  $X^2$  represent the Friedman's test statistic, N represents the number of participants and k represents the number of repeated measures.

## 8.3 Results

The results obtained for all kinematic data during each condition are presented in Table 11. Comparison between the 3 conditions showed no significant differences in the duration of the press ups, vertical displacement of the upper thorax (C7) or pelvis (S1) or spinal posture (C7-S1 distance).

Variable	NVIB	30L	40H
Duration (s)	1.76 <u>+</u> 0.38	1.69 <u>+</u> 0.34	1.76 <u>+</u> 0.38
C7 vertical displacement (m)	0.37 <u>+</u> 0.05	0.35 <u>+</u> 0.07	0.34 <u>+</u> 0.04
S1 vertical displacement (m)	0.24 <u>+</u> 0.02	0.24 <u>+</u> 0.03	0.24 <u>+</u> 0.02
Spinal posture (m)	0.51 <u>+</u> 0.03	0.51 <u>+</u> 0.03	0.52 <u>+</u> 0.03

The time normalised EMG amplitude for all muscles analysed is displayed in Figure 38. The pattern of muscle activation i.e. the EMG amplitude over time was consistent in SA and PM. For all muscles the activation pattern for 30L was similar to that seen in NVIB. However, the activation patterns for both LT and PM during 40H VIB displays differences from NVIB. The LT and PM muscle activation during 40H displays a new peak emerging in the eccentric phase occurring at approximately halfway through the eccentric phase. The concentric phase of LT 40H is similar to 30L. In addition to the eccentric phase increase of PM during 40H VIB, the concentric phase also displays a new peak, again occurring around halfway through the phase.



Figure 38. Time normalised EMG amplitude for each muscle during press up exercises. The red line = NVIB, the green line = 30L and the blue line = 40H. The error bars represent SEM.

None of the muscles analysed displayed significant increases in mean EMG amplitude in response to 30L VIB in the eccentric or the concentric phase of the exercise. During the eccentric phase the TR muscles displayed a significant increase from NVIB but not from 30L. PM was the only muscle in the eccentric phase with data that was not normally distributed, therefore was analysed with Friedman's test rather than repeated measures ANOVA. The effect size for all muscles was small (0.2), except TR which was medium (0.3).

The mean amplitude of the EMG signals during the concentric phase of the press up exercises displayed significant increases from NVIB in response to 40H VIB in all muscles except the lower trapezius, where 40H resulted in a slight decrease in amplitude. LT was the only muscle with data that was not normally distributed and therefore analysed with Friedman's test rather than repeated measures ANOVA. The mean EMG amplitudes for the eccentric and concentric phases are displayed in Figure 39. The effect size for all muscles was small (0.2), except TR which was medium (0.4).



Figure 39. Eccentric phase mean EMG amplitude (a) and Concentric phase mean EMG amplitude (b). The black bars = NVIB, the dark grey bars = 30L and the light grey bars = 40H.0 The error bars = SEM, \* = significantly different from NVIB (p < 0.05).

## 8.4 Discussion

The lack of significant difference between the timing to complete press ups, vertical displacements of cervical spine (C7) and pelvis (S1) and the distance between C7 and S1 indicate that the participants performed each press up in the same pace, range of motion and did not change posture i.e. develop kyphotic or lordotic postures during the press ups in any condition. This consistency of movements is important for two reasons, firstly vibration has previously been shown to reduce postural stability <sup>243</sup> and secondly the consistency in movement removes the variable of changing exercise performance when considering changes in EMG. Care was taken during the initial set up of NVIB, 30L and 40H to ensure all participants were positioned with hands and feet level, as changing the

relative position of height and hands and feet has been shown to influence EMG activity of shoulder musculature, particularly that of the serratus anterior <sup>234,244</sup>. In addition, hand position was standardised as the distance equivalent to that between the acromioclavicular distance with avoidance of movement in a caudad and cephalad direction, as this has been shown to influence the EMG activity of the shoulder and upper arms agonist muscles <sup>232,236</sup>. During a standard press up it has previously been reported that the forces experienced are typically around two thirds of body weight <sup>236,245</sup>. Although it has been shown that narrow base press ups, i.e. with the hands closer together, have an elbow torque which is 71% of maximal torque, compared to wide base press ups which are only 29% <sup>246</sup> which likely explains the reported increase in EMG activity during press ups with altered hand positions <sup>232,236</sup>. Considering the adherence to specific participant positional set up, combined with the lack of significant differences in kinematic parameters, it can be assumed the only variable influencing muscle performance was the introduction of vibration. WBV platforms have been shown to increase EMG activity of the lower body <sup>247–249</sup> with results indicating that increases occur in line with that obtained during increasing resistance of comparable exercises <sup>250</sup>. Utilising muscle contractions typical of the activity to be analysed i.e. without the intervention condition, has also been shown to have the advantage of providing a form of representative activation of the position for the movement to be analysed <sup>236</sup>, therefore providing comparable muscle activity for analysis. The results in Figure 38 and Figure 39 provide a sense of the level of increase in activity resulting from changing the vibration experienced during press up exercises. The greatest increases observed in EMG amplitude were observed in TR during 40H VIB, with increases apparent during both the eccentric and concentric phases. Interestingly Marin et al.<sup>251</sup> found that elbow extension during WBV is enhanced with high magnitude vibration (50Hz, 2.5 mm) but not low vibration (30 Hz, 1.2 mm), suggesting that triceps muscles require a certain threshold of vibration to enhance performance. The pattern of EMG activity for PM displayed a significant change in mean activation during 40H VIB only during the concentric phase. However, it should be noted that the data distribution of the mean EMG amplitude data during the eccentric phase was not normally distributed and therefore the analysis via non-parametric methods did not have the same power as that used to analyse the other muscles during the eccentric phase. The low and medium effect sizes obtained for the results are a further indication that tests with reduced power i.e. non-parametric hypothesis tests, are less likely to able to confirm

statistical significance. The eccentric phase of PM displayed a change in the pattern of activation, with a new peak becoming apparent at approximately halfway through the eccentric phase (see Figure 38). An increase in LT activation was also noted at this approximate time point, suggesting potential for increased muscular demand at this point of the movement. To the best of the author's knowledge there is no data on transmission of vibration through the human body in this position; yet it has previously been shown that when standing with increased knee flexion reduces vertical transmission of vibration <sup>7</sup>. It is therefore a reasonable assumption that whilst the elbows are flexed there is increased demand for damping of vibration transmission via the shoulder girdle, though further studies should be completed to confirm this. While patterns of EMG activity in LT did occur it should be noted that the increases in mean amplitude were not significant. The LT is the furthest analysed muscle from the WBV platform surface and is the only muscle to not display any significant increases in mean EMG amplitude. This observation can potentially be explained by the postulation that increased distance from the platform decreases muscle activity <sup>252</sup> indicating the role of the muscle and the location of the muscle are the key factors in the influence of vibration on muscle activity. This theory is given further credence by previously reported values of EMG during standard press ups to fatigue, where the SA displayed changes 3.5 times the level of the lower trapezius <sup>253</sup>. These differences in activation level are also potentially explained by the fact that the SA has previously been reported to be both the prime mover of the scapula <sup>254</sup> and a stabiliser muscle, particularly in prevention of conditions such as scapula winging <sup>255</sup>.

One final area worthy of note is that it has previously been reported that large differences have been observed between skilled and unskilled performers of press up exercises <sup>231</sup>. Though unfortunately while the timing of EMG activity was also reported, a single pass Butterworth filter was applied to the signals. Single pass filters cause phase distortions resulting in time lags within the signal <sup>256</sup> and are therefore not comparable to the data obtained within this investigation.

# 8.5 Summary

The introduction of vibration to press up exercises does not result in kinematic changes to exercise performance. Duration, posture and range of motion did not change in any tested condition indicating the actual exercise performance did not change in any condition. In contrast EMG activity displayed highly significant differences, particularly in the shoulder stabiliser muscles which displayed increases of 3.5 times the activity in a standard press up. In addition the pattern of EMG activity in both pectoralis major, serratus anterior and lower traps displayed significant changes. These results suggest that vibration has significant influence on both dynamic and stabilising muscles of the shoulder; these changes are potentially influenced by the changing the force moment arm during the movement. However, further studies should be completed on the transmission of vibration through the upper body and the potential safety implications of transmission of vibration in press up exercises.

# CHAPTER 9: CAN BENCH PRESS EXERCISES BE USED TO QUANTIFY CHANGES IN EMG DURING PRESS UPS ON A VIBRATING PLATFORM?

# 9.1 Introduction

The results from Chapter 8 indicate significant increases in muscle activity, particularly in the pectoralis major, following the addition of vibration during press ups, yet this does not provide information on the potential force changes resulting from increased vibration. To date research on the effects of vibration on the upper body force or power are both limited and varied.

#### 9.1.1 Effects of vibration on upper body muscular strength and power

The varying approaches adopted for investigation of the effect of vibration on the upper body include the use of custom rigs applying vibration to a cable used for elbow flexion exercises <sup>257,258</sup>, direct vibration of biceps tendon prior to elbow flexion <sup>259,260</sup> and use of vibrating dumbbells <sup>151,227,261</sup>. The application of custom rigs resulted in significant increases in acute power <sup>257</sup> and contraction force <sup>258</sup>. Results from indirect vibration using a vibrating dumbbell are inconsistent with different studies showing increases in EMG activity and force <sup>227</sup>, increases in power but not EMG activity <sup>97</sup> and no increases in power <sup>151</sup>. Direct musculotendinous vibration studies have found no increase in either upper limb power, joint moment or angular velocity during bicep curls <sup>259,260</sup>. The influence of standing WBV on upper body function has also been investigated. Grip strength has been shown to be non-significant <sup>262–264</sup>, though the effect of WBV on EMG during elbow flexion has produced both significant <sup>224</sup> and non-significant <sup>202</sup> changes. Although these investigations begin to provide a picture of the potential effective use of vibration exercise in upper body exercise; the application in complete exercise regimes is somewhat limited to isolation exercises e.g. bicep curls. One of the most common upper body compound exercises is the press up, which is used to improve upper body strength, power and muscular endurance <sup>229</sup>, yet to the best of the author's knowledge there is only one peer reviewed investigation in to the influence of vibration on the press up. Marin et al <sup>265</sup> considered the effect of vibration exposure during press up exercises on subsequent bench press performance, although the results did not indicate improved performance. This research therefore suggests that vibration results in changes in both EMG parameters and force/power output. While the volume of myoelectrical activity is known to vary with the intensity of muscular contraction, predicting force from EMG amplitude must be approached with caution <sup>256,266</sup>.

# 9.1.2 EMG and force

The relationship between EMG amplitude and force has been studied for over 60 years, yet there are conflicting results with regards to whether this relationship is linear or non-linear (see Table 12).

Authors	Linear	Non- Linear	Muscles	Comments
(Lippold, 1952) <sup>267</sup>	✓ ✓		Gastrocnemius Soleus	
(Close J et al., 1960) <sup>268</sup>	$\checkmark$		Soleus	
(Zuniga and Simons, 1969) 269		$\checkmark$	Biceps	
(Milner-Brown and Stein, 1975) <sup>270</sup>	$\checkmark$		First dorsal interosseous	Force adjusted on a logarithmic scale
(Komi and Vitasalo, 1976) <sup>271</sup>		$\checkmark$	Rectus femoris	
(Miller and Seireg, 1977) <sup>272</sup>	$\checkmark$		Vastus medialis	
(Bigland-Ritchie, 1981) <sup>273</sup>	√ √ √	√ √ √	Soleus First dorsal interosseous Adductor pollicis Biceps Triceps BR	Non-linear graphs display a linear response above 50% MVC
(Lawrence and De Luca, 1983) <sup>274</sup>	✓	√ √	First dorsal interosseous Biceps Deltoid	
(Alkner et al., 2000) <sup>275</sup>	√ √	✓	Vastus lateralis Rectus femoris Biceps femoris	
(Madeleine et al., 2001) <sup>276</sup>		$\checkmark$	First dorsal interosseous	
(Praagman et al., 2003) <sup>277</sup>	✓ ✓		Biceps Brachioradialis	Also linearly related to NIRS

Table 12. The relationship between force and EMG amplitude.

It is clear from Table 12 that there are conflicting results in scientific literature. One of the primary reasons for these differences results from the conclusions drawn from the results. For example, Bigland-Ritchie *et al.* <sup>273</sup> chose to report muscle responses as non-linear when the response is only linear after a certain threshold of activation is reached, whereas Miller and Seireg <sup>272</sup> opted to report a similar responses as linear. Milner-Brown and Stein <sup>270</sup>

applied a logarithmic scale to the force value in order to obtain a linear response, which suggests that raw force values would have yielded a non-linear relationship between EMG and force. In addition there are also technical and physiological aspects of study design which can influence the difference in the analysis of EMG to force. An example of technical influences of study design was introduced in Chapter 8 with regards to placement of electrodes during protocol set up. Another example of a technical influence on test results is the type of electrodes utilised in the study. It has previously been shown that during muscular contractions at less than 50% of MVC the EMG signal amplitude from surface electrodes will be lower than that obtained from intramuscular electrodes, however, at contractions above 50% of MVC the amplitude from surface EMG progressively increases <sup>278</sup>. In conjunction to technical aspects of EMG analysis the physiological influences on myoelectrical signals must be considered. The first key physiological factor to be considered is the contraction type. Concentric contractions are weaker than both isometric and eccentric contractions (see Figure 40 (a)) and as such typically require greater levels of myoelectrical activity to produce a given force. Eccentric contractions are typically the strongest of the muscular contractions and therefore require the least amount of myoelectrical activity to achieve the required force levels <sup>279–281</sup>. The second physiological factor to be considered is the muscle fibre length. A fundamental aspect of concentric and eccentric contractions is a change is muscle length. It has been suggested that joint position influences EMG amplitude <sup>278</sup>. However, studies of MVC and evoked contractions of the biceps have resulted in constant levels of EMG regardless of joint position <sup>282</sup>. Further studies have produced results suggesting muscle groups can differ in their responses, with biceps and brachioradialis EMG being influenced by joint angle, yet triceps EMG was not <sup>283</sup>. Isometric contractions of the long head of the biceps femoris at increasing muscle length have shown an inverse relationship between EMG amplitude and joint torque, with EMG decreasing and torque increasing as muscle lengthened <sup>284</sup>. The ability of muscle to generate greater forces at greater lengths is attributed to the combined influence of passive and active tensions within the muscle  $^{268}$ , as displayed in Figure 40 (b).



Figure 40. Effect of velocity (a, adapted from Hall <sup>285</sup>) and muscle length (b, adapted from Close et al. <sup>268</sup>) on force.

The disparities in reports of the influence of muscle length can potentially be explained by the influence of the force moment arm. Nourbakhsh and Kukulka<sup>286</sup> investigated the influence of changing moment arm and muscle length of the triceps surae musculature. The results indicated when the moment arm was changed in the presence of a constant muscle length the EMG amplitude resulting from plantar flexion torque was significantly increased. No significant differences where observed with stable moment arm and increasing muscle length. Mohamed et al. suggested a similar hypothesis for decreased EMG activity in knee flexor muscles at extended knee angles (semitendinosus, short head of biceps femoris, sartorius and gracilis), where reduced EMG amplitude is speculated to be a consequence of a reduced moment arm of the muscles in extended knee positions. While the authors report 'common responses of decreased EMG activity' a lack of consistency in results prevented significant differences being reported <sup>287</sup>. Coupled with changes in muscle length is the velocity at which the changes occur. Gerdle et al. 288 used an isokinetic dynamometer to investigate the influence of contraction velocity on the EMG amplitude and MNF. The results indicated that neither parameter is dependent on the angular velocity of the contraction. Potvin et al. <sup>289</sup> investigated the influence of different contraction velocities of the biceps brachii whilst holding a 7 Kg load. Concentric contractions at higher velocities yielded increases in EMG amplitude, conversely eccentric contractions increased EMG amplitude during slower velocities. To the best of the author's knowledge there does not appear to be enough evidence to deduce whether the differences in findings are a consequence of different muscle groups or contractions types. A final physiological area to be considered is that when collecting EMG data the muscle, or muscle groups, being considered may not represent all of the muscular activity responsible for the action being completed <sup>279,290</sup>. All movement is achieved by the combined effort of groups of muscles, therefore the fact that some muscles which are not being recorded with EMG may contribute to the movement. While direct measurement of EMG during press ups on a vibration platform does not present an issue, measurement of force between a participant and a WBV platform during vibration is difficult. The aim of this study is therefore to investigate if the level of change in EMG can be used for comparison to exercises using the same agonist muscles i.e. bench press, to estimate a comparative change in force output. The hypothesis is that vibration will directly augment the level of EMG during press up exercises.

### 9.2 Methods

#### 9.2.1 Subjects

Fourteen healthy male participants (25.6  $\pm$  3.9 years, 1.8  $\pm$  0.1 m, 73.0  $\pm$  3.9 kg) without any history of recent illness of injury volunteered for the study and provided informed consent in accordance with University Ethics Committee and the Declaration of Helsinki.

#### 9.2.3 Study design

Participants attended two testing sessions separated by at least 48 hours to provide participants time to recover from exertions during test session. During both sessions participants completed 4 min arm crank ergometery at 40 W (Lode Angio, Groningen; The Netherlands) as a warm up activity. The first test session consisted of a one rep maximum (1RM) bench press test completed following the NSCA protocol <sup>126</sup>. During the second visit bench press exercises were completed at 20, 32, 44, 56 and 70% of 1RM to provide data for the generation of calibration curves and three press up conditions on a Power Plate Pro 6 WBV platform (Power Plate Ltd). Press up conditions were: press up in the absence of vibration (NVIB), low amplitude and frequency vibration (30L; 1.2 mm, 30 Hz) high amplitude and frequency vibration (40H; 1.9 mm, 40 Hz). All exercises were performed in a randomised order at a self-selected pace by participants for a period of 15 s with 3 min rest

between each set. Both press ups and bench press exercises were performed with the hands consistently placed at 1.2 times the distance between the acromioclavicular joints. During press ups on the Power Plate the participant's feet were placed on a small bench to replicate the press up position of that on the floor i.e. hands and feet at equal height.

#### 9.2.3 Data collection and processing

3D motion capture during the bench press and press up exercises was acquired using 16 mm retro-reflective markers on each end of the barbell and the spine at C7 respectively. The sampling frequency of the motion capture was 200 Hz using 10 Oqus infrared cameras (Qualisys, AB, Sweden). Synchronous to motion capture, surface EMG (Delsys Bagnoli system, Boston, USA) was obtained via differential bipolar Ag-AgCl electrodes placed on the anterior deltoid (AD), pectoralis major sternal (PMS) and clavicular (PMC) portions and triceps (TR) muscles in accordance with SENIAM recommendations <sup>95</sup>. Signal impedance and noise artefacts were minimised by skin preparation and securing of wires to prevent excessive movement during press ups. EMG signals were amplified (1 k gain) with a bandwidth of 20-450 Hz and a sampling frequency of 2000 Hz. A single reference electrode was placed on the skin level with the C7 vertebrae. All synchronous data was tracked using Qualisys track manager software (Qualisys, AB, Sweden) and exported in .c3d format for post processing in Visual3D (C-Motion, Inc. Germantown, USA).

EMG signals were corrected for zero offset by subtracting the signal mean, fully rectified and smoothed using a 8th order Butterworth low pass filter with a cut-off frequency of 4 Hz.

The process for producing time normalised graphs of EMG amplitude during press ups detailed in Chapter 8 was repeated to allow direct comparison of the data generated, furthermore the EMG data obtained during bench press with 56% of 1RM was filtered and plotted for direct comparison of EMG amplitude and timing of activity. For direct comparison of average, time normalised results between subjects, signals were divided into concentric and eccentric phases for each press up based on the vertical position of the C7 or barbell markers and exported to MatLab (MathWorks, USA) for statistical analysis. The total EMG activity during each phase of the bench press movement was calculated by summing the individual amplitudes of muscle activity.

#### 9.2.4 Statistical analysis

A regression analysis utilising individual and total muscle activity was completed in order to generate calibration graphs for comparison to total EMG activity during press up exercises (representative trace in Figure 42). The level of variance in muscle activity explained by variance in the increasing mass (1RM%) was quantified via the coefficient of determination of the regression line (R<sup>2</sup>). To ensure that press up durations between Chapter 8 and the current study were not significantly different the duration of the eccentric component was also tested. The Anderson-Darling test for data distribution was applied to the data, with results indicating a normal distribution. A repeated measures ANOVA test with Tukey's HSD post hoc was applied to identify the level and location of any significant differences between test conditions and two sample t-Test was used to identify any differences between the mean eccentric duration of the press ups in chapters 8 and the current study. Finally the omega squared effect size  $(\hat{\omega}^2)$  of differences was calculated using the approach detailed in Kinnear and Gray <sup>162</sup>. All values presented are means ± 1 SD. The level of significance for statistical tests was set at alpha = 0.05.

#### 9.3 Results

All participants completed the protocol; however, the EMG activity of four participants was outside the range of the calibration graph and as such was not suitable for regression analysis. The ratio of 1RM to body mass (1RM/BM) of the 10 suitable participants was 1.23  $\pm$  0.23 s, compared to the ratio of the remaining four unsuitable participants which was 0.86  $\pm$  0.19. There was no significant difference in the displacements of the C7 marker during press ups or the displacement of the barbell during bench press exercises indicating consistent range of motion for all exercises completed. There was no significant difference in the duration of the press ups. However, there were highly significant differences between the durations of both the concentric phase and total movement time of bench press repetitions at 70% of 1RM compared to all other bench press up and bench press exercises the time normalised EMG activity is displayed in Figure 41. An intensity of 56% of 1RM was selected from the

range of bench press conditions as the EMG amplitude was representative of the press up condition, 56% was also the highest exercise intensity level with the same velocity of movement as the press up exercises. The TR and PM muscles are analysed in both Chapters 8 and the current study, with the electrode placement for TR and PMS being identical, therefore allowing direct comparison between muscle activities. The activations patterns of all muscles analysed in the current study did not change in timings or location of peaks for either eccentric or concentric phases of the press up exercises with regards to the total press up duration. However, the eccentric phase in Chapter 8 occurred at approximately 48% of the total press up duration, whereas in the current study the eccentric phase lasted for approximately 55% of the total press up duration, this difference is highly significant (p < 0.001).



Figure 41. Time normalised EMG amplitude for each muscle during press up exercises. A = anterior deltoid, b = triceps, c = sternal portion of pectoralis major, d = clavicular portion of the pectoralis major. The red line = NVIB, the green line = 30L and the blue line = 40H. The error bars represent SEM.



Figure 42. Representative EMG calibration graph for triceps EMG activity. O = concentric phase, X = eccentric phase,  $\Phi$  = NVIB,  $\blacksquare$  = 30L,  $\blacklozenge$  = 40H.

The predicted percentages of bench press 1RM during the concentric phase of the press up as calculated from the calibration graphs are displayed in Table 13. Significant differences (p < 0.001) were obtained for both the sternal and clavicular portions of the pectoralis major muscle at 40H VIB compared to NVIB, but not the anterior deltoid or the triceps muscles, although the overall  $\hat{\omega}^2$  was small, at 0.3 and 0.4 respectively.

	NVIB (%)	30L (%)	40H (%)	R <sup>2</sup>
AD	56.8 ± 10.2	55.2 ± 17.3	61.6 ± 16.6	0.96 ± 0.02
PMS	51.0 ± 15	52.1 ± 17.4	$60.0 \pm 14.2^*$	0.95 ± 0.1
РМС	55.7 ± 8.6	62.4 ± 9.8	85.8 ± 19.5 <sup>*</sup>	0.97 ± 0.2
TR	60.8 ± 13.7	59.4 ± 10.6	65.8 ± 14.6	0.94 ± 0.07

Table 13. Predicted percentage of 1RM based on EMG activity during press up. R2 = the coefficient of determination, \* = significant increase from NVIB (p < 0.05).

The combined mean EMG amplitude for all muscles during the press up exercises is displayed in Figure 43 as a predicted percentage of the 1RM during concentric phase of the bench press exercise. These values are based on the regression equations obtained from the calibration graphs. The mean value for the predicted exertion level during press ups based on the combined EMG activity of all muscles analysed i.e. summed mean activity of all four muscles. The difference between NVIB and 30L was not significant. The difference between 40H and both NVIB and 30L was highly significant (p < 0.01). The effect size for the overall differences was small ( $\hat{\omega}^2 = 0.4$ ).



Figure 43. Predicted percentage of mean EMG amplitude during 1RM. Black bars = Concentric phase (n = 10), grey bars = eccentric phase (n = 7 for NVIB and 30L, 4 for 40H). \* = significant increase from NVIB.

The EMG during the eccentric phase of the press up exercise produced EMG levels outside of the calibration graph range in 8 of the 15 participants for the NVIB and 30L conditions and 11 out of 15 participants for the 40H condition. The mean values were  $60.5 \pm 10$ ,  $62.5 \pm$ 5 and  $66.9 \pm 3\%$  for NVIB, 30L and 40H respectively. The eccentric sample size, (n = 4) was considered too small for reliable hypothesis testing.

#### 9.4 Discussion

One of the key findings from this investigation is that for regression analyses such as the study undertaken here, a minimum strength:1RM ratio of approximately 1 or higher is required to generate a calibration curve. For effective interpolation of outcome values (Y axis) and the predictor values (X axis), must be within the range obtained during the data collection <sup>291</sup>. In the current study four participants did not meet this criterion, therefore reducing the sample size. Although the remaining 10 participants provided data from the concentric phase, some values were close to the upper limit. Potentially extending the calibration curve to 80% of 1RM could have allowed inclusion of greater number of participants. However, considering the lower ratio of 1RM/BM of the excluded participants, the increase in bench press intensity was not advisable as the increased demands would likely exceed the ability of the participants. Previous studies have indicated that the forces experienced during press up exercises are approximately two thirds of body weight <sup>236,245</sup>, which provides a sensible hypothesis for the need for a minimum bench press 1RM when generating a calibration curve. Bench press loads equivalent to that experienced during press ups have been shown to have PM and TR EMG amplitudes that are not significantly different <sup>245</sup>. While both press up and bench press exercises use the same agonist muscles to achieve the desired movement, it should be noted that during the press up the eccentric component has far higher levels of EMG activity than that obtained during the eccentric phase of the bench press. Of all participants tested only four produced EMG data during the eccentric phase of the press up exercises that fell within the predictor values obtained during the bench press exercises. This could be explained by the greater stability of the bench requiring less muscular action to stabilise the body. This issue could be addressed in future studies by changing the predictor exercise to press ups with upper body loading via weight vests. This approach would allow loading to increase the forces required for completion of the exercise without changing the style of the exercise performed.

To the best of the author's knowledge the only potentially comparable study is that of Poston et al <sup>292</sup>, who investigated the influence of a mechanically vibrated barbell on power output during bench press exercises. However, there were key differences between the studies that limit direct comparisons. Firstly, the vibration was introduced via a barbell and press ups were not considered. Secondly, vibration was introduced during isometric

holds, not during dynamic movements. Thirdly, only one set of vibration parameters (30 Hz, 1.1 mm amplitude) was considered and finally the variances in the results were particularly large preventing the detection of significant differences in performance. Ultimately the authors could only speculate that differences were the result of psychological factors.

The level of change occurring during different variants of press ups has been studied more extensively. Results indicate that narrow base hand positions are effective increasing the level of EMG in both the PM and TR, with increases of 2-25% of PM MVC and 10-35% of TR MVC <sup>232,236</sup>. The increase in EMG can potentially be explained by two factors, firstly it has been shown that narrow base press ups increase elbow torque to 71% of maximal torque <sup>246</sup>, secondly narrow base hand positions require the PM muscles to function in shortened muscle length position. Basic muscle mechanics indicate that force output is lower in shortened and lengthened states, therefore requiring higher motor unit function to generate forces equivalent to that in optimal muscle fibre lengthened positions <sup>266</sup>. In addition Freeman et al. <sup>231</sup> investigated a variety of press up exercises including press ups with both hands on a basketball, press ups with hands on 2 basketballs, fast concentric movement and plyometric movements. EMG increases as a percentage of MVC in PM were 8, 20, 26 and 27%, AD 9, 2, 11 and 16% and TR 3, 0, 2 and 20% respectively. While it should be noted these results are displayed on scales different to that of the current study, the increases are of a similar magnitude, based on this it could be hypothesised that press ups with vibration is approximately equivalent to either narrow base or plyometric press ups with regards to increases in EMG. Typically the results obtained indicated that PMC activity was a higher percentage of MVC compared to PMS, this is in line with results obtained when comparing the effect of grip width and hand position of bench press exercises <sup>293</sup> yet contrast to investigations of incline compared to decline bench pressing <sup>294</sup>. However, it should be noted that EMG electrodes placed centrally on the PM, both vertically and horizontally, typically display lower EMG amplitudes than at any other location on the PM <sup>295</sup> and that the regional activity of PM has been shown to be influenced by fine tuning of the motor units by the CNS to match the task required <sup>296</sup>. Considering this variability within EMG of the PM regions and functions care must be taken before conclusions can be drawn. Over-interpretation of results can be avoided by the analysis of muscle function in its combined form i.e. the analysis of total EMG amplitude, as presented in Figure 43. These results lead to the conclusion that when using synchronous vertical vibration for upper

body exercise 30L setting will not increase EMG activity, yet 40H settings will lead to an increase approximately equivalent to increasing the load on a barbell by 10% of 1RM. An additional area worth of note is the spatial and temporal effects of the exercises. The press up exercise durations did not change during the concentric phase or total repetition times. The introduction of vibration also did not change the level of displacement of the C7 marker, representing vertical displacements completed during press up exercises. Therefore the increase in muscular activation observed during 40H vibration cannot be attributed to changing pace or range of movement during press ups. Contrarily, bench press exercises displayed highly significant decreases in movement velocity during repetitions at 70% of 1RM for both the concentric phase and the total movement duration. Considering the actual range of movement duration introduces a fundamental difference between the bench press at 70% of 1RM and press ups with vibration i.e. increasing muscle activation via the introduction of vibration does not reduce movement velocity.

Overall the effect sizes for the significant increases in muscle activations were small, though  $\hat{\omega}^2$  is influenced by two factors which lead to this conclusion. Firstly, at 10-15 % of the mean values, the variance within the predicted values compared to the level of change in 30L and for AD and TR both in 30L and 40H were fairly large, secondly and more importantly, the fact that were no differences between NVIB and 30L reduces the overall effect size dramatically. If the authors had ignored the first repeated measure and simply performed Cohen's effect size between NVIB and 40H the effect size would have been large, despite the level of variance. Consideration of the lack of changes in EMG seen in 30L condition, in both Chapter 8 and the current study, and the overall influence on the effect size, future studies could focus simply on NVIB vs. 40H without the need to consider 30L.

With regards to comparing the muscular activation recorded during the current study and that in the previous Chapter it is worth noting the TR and PMS displayed less activation during the eccentric phase relative to the concentric in the current study compared to that observed in Chapter 8. An anecdotal observation is that the participants of the current study had greater resistance training experience which likely explains the differences in activation patterns.

#### 9.5 Summary

The results obtained suggest that bench press exercises can be used to generate a calibration curve. However, there are minimum strength requirements for this to be effective. In addition, the calibration curve is only effective for the concentric phase of press up exercises. Providing these limitations are accounted for, the results indicate that bench press exercises can be used to generate effective calibration curves for press up exercises can also be confirmed.

The results from this study also confirm the second hypothesis, that appropriate levels of vibration can significantly increase muscle activation of the upper body, therefore vibration can be used to augment muscle function during press up exercises. The increase is approximately equivalent to a load increase of 10% of 1RM during bench press exercises. In contrast to bench press exercises, the increase in muscle activation during press ups with vibration is achieved without influencing the duration or kinematic aspects of the exercise.

# **CHAPTER 10: CONCLUSIONS**

The aim of the research reported in this doctoral thesis was to investigate the fundamental responses of the human body to vibration exercises. This research indicates that WBV during exercise has significant influences on both the muscular and vascular physiological systems of the human body. Throughout the studies there were no significant changes in either spatial or temporal parameters, indicating that WBV did not alter the performance of the exercises analysed.

Analysis of the powerBIKE identified issues with the mechanical design. The vibration mechanism introduced additional resistance and the settings provided no equivalent resistances for NVIB and VIB conditions. However, while it is pertinent to not over interpret results; initial findings indicate that at higher cadences there was a greater increase in muscle activation in the presence of vibration. This suggests that when comparable resistance parameters are available, i.e. power matched VIB and NVIB cadence, meaningful differences in muscle activation could be hypothesised.

Subsequent studies utilised the WBV platforms to investigate physiological and biomechanical responses to vibration during both dynamic and static exercises. The introduction of WBV to heel raise exercises significantly reduced the depletion of oxygenated haemoglobin, total haemoglobin and the normalised tissue haemoglobin index of the lateral gastrocnemius. NIRS cannot indicate if these changes are a consequence of distal vasospastic responses, changes in proximal blood flow or local muscle activity, therefore further studies were designed to address these issues. During quiet standing, there was no indication of vasospastic responses causing blood pooling in the lower limb.

It can be inferred that vascular resistance to blood flow was not altered by the addition of vibration since laser Doppler and photoplethysmographic measurements showed no significant changes in microvascular drainage or resistance index. The blood flow velocity of the dorsalis pedis artery significantly increased, despite no changes in heart rate or blood pressure. The increase in blood flow velocity, without central cardiovascular influence, indicates further investigation of peripheral muscle function was required.

The myoelectrical activity of the lateral gastrocnemius did not increase during heel raises with WBV, but was significantly increased in the soleus muscle. A possible explanation for this is that the soleus muscle has a higher volume of slow twitch fibres, which have lower activation thresholds. The reduced depletion in oxygenated haemoglobin of the lateral gastrocnemius could be explained by an increase in blood flow, as indicated by increased blood flow velocity. However, the peripheral cardiovascular influences on myoelectrical activity could not be inferred and required further investigation. During isometric squats the muscle fibre conduction velocity of the vastus lateralis did not change but the myoelectrical median frequency significantly decreased. This could be a consequence of local oxygenated haemoglobin volume protecting muscle fibre conduction velocity.

The data obtained thus far indicates that WBV has a significant influence on the lower body. However, there is limited comparable scientific data in the literature on the effects of upper body exercise on vibration platforms; therefore, the impact of vibration during press ups was investigated.

There were significant increases in the EMG amplitudes of shoulder muscles in response to higher vibration settings (40 Hz high amplitude), when compared to press ups without vibration. Greater increases were also noted in the EMG amplitude of dynamic muscles compared to stabiliser muscles. In terms of force, the quantification of increased muscle activity required the generation of calibration curves for muscular activity. Combined muscle activity significantly increased during the concentric phase of movement, with 40 Hz high amplitude vibration having an equivalent effect to increasing bench press load by 10% of the participant's one repetition maximum. These results are particularly important as to date there is no published research on the influence of vibration during the performance of press up exercises.

The changes in muscle activation of both lower and upper body studies indicate that vibration has a selective influence on muscle function, with vibration having a varied influence on different muscles. The combination of results within this thesis contributes to the scientific evidence base for both practitioners and researchers in the health related professions. The significant changes that were measured in the muscular and vascular physiological systems of the human body can be used as a basis to influence the design of exercise regimes and inspire future research.

# **CHAPTER 11: FUTURE WORK**

While the studies completed within the scope of this PhD have added to the scientific evidence base for the use of vibration during exercise, there are still many areas for further research. The powerBIKE presents the most difficult challenge for progressive research. While adjustments to the current mechanical design have potential to produce power matched settings, the manufacturer is still developing the prototype design. Until a final version of the powerBIKE is produced, scientifically sound studies cannot be completed, or are pointless with the prototype utilised in chapter 3, if the current design is never reproduced.

WBV platforms present a more valid option for future research. An interesting observation within the current lower body studies was the significant reduction in lateral gastrocnemius NIRS but lack of significant change in EMG amplitude. The increased EMG amplitude in the soleus muscle potentially explains these differences; therefore NIRS of the soleus muscle during heel raise exercises with WBV would provide valuable information on lower leg function during vibration. Logically this also raises the question of whether slow twitch fibre are more susceptible to WBV than fast twitch fibres. The increase in muscular activity is also potentially an explanation for the increase in peripheral blood flow, with muscular contractions increasing blood flow. If future research can support this hypothesis it would provide an explanation for how vibration affects local blood flow without changing central blood flow i.e. heart rate. Considering there are conflicting reports of tissue oxygenation parameters of the vastus lateralis during isometric vibration exercise <sup>73,106</sup>, further studies of the vastus lateralis, particularly with synchronous EMG, will help to strengthen the scientific knowledge of the influence of vibration on blood flow and tissue oxygenation and how this in turn influences EMG.

It should be noted that all studies completed in this PhD were completed in the absence of additional load i.e. bodyweight exercises or static positions. Future work with additional resistances applied during exercise, particularly in press up exercises where research is scarce, would be beneficial.

One final key area for future research is with regards to the transmission of vibration whilst in the press up position. Whilst to date the transmission of WBV in the standing position has been studied, there are no such studies for the upper body. As transmission of vibration is related to the safety of the exercise, accelerometer studies during the press up position are vital for evidence based practice and research. The results from studies of vibration transmission through the upper body in this position would provide important information with regards the potential risks to the head and neck.

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## **APPENDICES**

## **Appendix I Participant information letter**



#### MEDWAY SCHOOL OF SCIENCE

SCHOOL OF LIFE AND SPORT SCIECNCE

# The effects of whole body vibration training on neuromuscular performance in healthy individuals.

Researchers contact details:

Daniel Robbins0208331 7986rd51@gre.ac.ukDr Mark Goss-Sampson0208331 7986gm03@gre.ac.ukCentre for Sports Science = rd Human Performance.University of Greenwich = t Medway,Central Avenue,<br/>Chatham Maritime,<br/>Kent ME4 4TBsupervisor:Supervisor:

Dr Mark Goss-Sampson

Dear participant,

Thank you for volunteering to participate in this study. The overall aim of this study is to investigate the effects of vibration on exercise performance. In order to achieve this aim the immediate effects of vibration on exercise must first be understood. The study you have volunteered for will involve light exercise on a vibrating platform. Prior to starting the exercise there may be some questions you would like answered, therefore this handout has been produced to help you understand the process. If there are any additional questions you would like answering please do not hesitate to contact me on either rd51@gre.ac.uk or 0208331 7986 (please only use the phone number between 9am-4pm).

#### **Confidentiality**

All data and personal information will be stored securely within University of Greenwich premises in accordance with the terms of the Data Protection Act 1998 and the University's own data protection requirements, and will be accessed only by the researchers involved. After completion of the study, all data will be made anonymous (i.e. all personal information associated with your data will be removed). Your data will be anonymous in any written reports, articles, and presentations of the results of the study.

- 1. What sort of exercises will I be expected to do?
  - a. The exercises will be lower limb exercises, examples include: squats (static and/or dynamic), heel and/or toe raises, lunges. All exercises will be explained and demonstrated prior to testing.
- 2. Will I be exposed to vibration for the whole session?
  - a. No. The total time estimation includes set up, rest times and removal of any test equipment
- 3. How long will testing last?
  - a. The whole testing session will not be longer than one hour, depending on the study you may be asked to come back for an additional session.
- 4. What test equipment will be used?
  - a. Depending on the testing you are undertaking we may be looking at the amount of oxygen in your muscles, the electrical activity of your muscles or your position while you exercise. It is assumed that vibration will cause changes in each of these factors so we will place sensors on the surface of your skin (there are no needles or anything that will cause pain) to detect these changes. We may need to prepare the skin at the site of the sensors (cleaning or potentially shaving the area), however we will not break the skin and only use suitable and clean equipment to do this.
- 5. Do I need to do anything before testing?
  - a. It is better if you do not exercise up to 24hours before testing and do not eat immediately before testing. It is also preferable you do not use skin creams on your legs up to 24 hours before testing. Apart from that there are no special requirements.
- 6. Are there any risks associated with testing?
  - a. If you have had an intraocular lens replacement you are at risk of dislocation
  - b. We are looking to test people without any injuries, if you have any injuries you should state what the injury is prior to testing. It is likely you will not be suitable for testing if you have lower limb or spinal injuries.
  - c. Pre-test questionnaires will help establish if you are suitable.
- 7. Will I get feedback about my test results?
  - a. Data will not be immediately available as analysis and comparison to other data will be required. If you are interested a summary will be available at a later date.

#### **Important**

You are free to take part or not in this study. You can withdraw from your participation at any time without any reason given or consequences.

#### Once again, thank you for volunteering!

#### Q+A:

Appendix II Example Consent form

## UNIVERSITY of GREENWICH

#### **RESEARCH ETHICS COMMITTEE**

#### **CONSENT FORM**

SCHO	OOL/DEPARTMENT	
Title o	of Study	
Invest	tigator's name	
	<b>completed by the subject/patient/volunteer/informant/interviewee/parent/guardian</b> te as necessary)	
1. 2. 3. 4. 5. 6.	<ul> <li>Have you read the information sheet about this study?</li> <li>Have you had an opportunity to ask questions and discuss this study?</li> <li>Have you received satisfactory answers to all your questions?</li> <li>Have you received enough information about this study?</li> <li>Which researcher/investigator have to spoken to about this study?</li> <li>Do you understand that you are free to withdraw from this study?</li> <li>at any time</li> <li>without giving a reason for withdrawing</li> </ul>	YES/NO YES/NO YES/NO YES/NO
	without affecting your future with the University/studies/medical or nursing care	YES/NO YES/NO
7.	Do you agree to take part in this study?	YES/NO
Signed		Date
Name	e in block letters	
Signa	Date	

#### Please note

- For persons under 18 years of age the consent of the parent(s) or guardian(s) must be obtained or an explanation given to the Research Ethics Committee and the assent of the child/young person should be obtained to the degree possible dependent on the age of the child/young person.
- In some studies witnessed consent may be appropriate

The consent form **must** be signed by the actual investigator concerned with the project after having spoken to the subject to explain the project and after having answered his or her questions about the project.

#### **Appendix III Example MATLAB scripts**

## III(a) Loading Data from Excel and Testing for Normal Distribution

#### Limitations

This script will only work with a dataset of four columns with the same column/vector/variable headings as used in the file selected below.

#### **Clear Memory and Load EMG Data**

```
close all, clear all,clc
ds = dataset('xlsfile', 'timings for matlab.xls');
```

#### Performing Lilliefors Test for Normality and Displaying Results

```
GVnorm = lillietest(ds.GV);
         if GVnorm == 0
                  disp ('The distribution of Gastroc EMG during vibration is normal')
         else
                  disp ('The distribution of Gastroc EMG during vibration is not normal')
         end
GNVnorm = lillietest(ds.GNV);
        if GVnorm == 0
                  disp ('The distribution of Gastroc EMG during no vibration is normal')
         else
                  disp ('The distribution of Gastroc EMG during no vibration is not normal')
         end
SVnorm = lillietest(ds.SV);
         if SVnorm == 0
                  disp ('The distribution of Soleus EMG during vibration is normal')
         else
                  disp ('The distribution of Soleus EMG during vibration is not normal')
         end
SNVnorm = lillietest(ds.SNV);
         if SNVnorm == 0
                  disp ('The distribution of Soleus EMG during no vibration is normal')
         else
                  disp ('The distribution of Soleus EMG during no vibration is not normal')
```

#### end

#### **Results of distribution analysis**

The distribution of Gastroc EMG during vibration is normal The distribution of Gastroc EMG during no vibration is normal The distribution of Soleus EMG during vibration is normal The distribution of Soleus EMG during no vibration is normal

## Performing Paired hypothesis tests on data

As the data is from normally distributed data then parametric hypothesis tests will be used. The data is from a cross-over study with single session data collection therefore paired t-Tests will be used. The direction of any data changes is not currently known so two-tailed tests are required.

```
[h p ci] = ttest(ds.GV,ds.GNV,0.05,'both');
        if p>0.05
                 disp('p > 0.05, The difference for gastroc vib vs. no vib is not significant');
        elseif (p<0.05) & (p>0.01)
                 disp('p<0.05, The difference for gastroc vib vs. no vib is significant');
        else p<0.01
                 disp('p<0.01, The difference for gastroc vib vs. no vib is highly significant')
        end
[h p] = ttest(ds.SV,ds.SNV,0.05,'both');
        if p>0.05
                 disp('p > 0.05, The difference for soleus vib vs. no vib is not significant');
        elseif (p<0.05) && (p>0.01)
                 disp('p<0.05, The difference for soleus vib vs. no vib is significant');
        else p<0.01
                 disp('p<0.01, The difference for soleus vib vs. no vib is highly significant')
        end
[h p] = ttest(ds.GV,ds.SV,0.05,'both');
        if p>0.05
                 disp('p > 0.05, The difference for gastroc vib vs. soleus vib is not significant');
        elseif (p<0.05) && (p>0.01)
                 disp('p<0.05, The difference for gastroc vib vs. soleus vib is significant');
        else p<0.01
                 disp('p<0.01, The difference for gastroc vib vs. soleus vib is highly significant')
        end
[h p] = ttest(ds.GNV,ds.SNV,0.05,'both');
        if p>0.05
                 disp('p > 0.05, The difference for gastroc no-vib vs. soleus no-vib is not significant');
        elseif (p<0.05) && (p>0.01)
                 disp('p<0.05, The difference for gastroc no-vib vs. soleus no-vib is significant');
        else
                 disp('p<0.01, The difference for gastroc no-vib vs. soleus no-vib is highly significant')
        end
```

#### **Results of Hypothesis Tests**

P > 0.05, The difference for gastroc vib vs. no vib is not significant
P > 0.05, The difference for soleus vib vs. no vib is not significant
P < 0.05, The difference for gastroc vib vs. soleus vib is significant</li>
P < 0.01, The difference for gastroc no-vib vs. soleus no-vib is highly significant</li>

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## III(b) Pooled Variance

### Limitations

This code calculates pooled variance on an array with normal. If data is not normally distributed the code should be amended to use mad(x,1) in place of std(x).

#### Load Data and Define Matrix

```
ds = dataset('xlsfile', 'TAtemp.xls');
a= [ds.Baseline ds.PostVib1 ds.PostVib2 ds.PostVib3 ds.PostVib4...
ds.PostVib5 ds.Recovery1 ds.Recovery2 ds.Recovery3 ds.Recovery4 ds.Recovery5];
```

#### **Calculate Parameters**

```
n=length(a(:,1));
stdev=std(a);
stdev=stdev.^2;
```

## **Pooling Variance**

top=sum(top);

```
for j=1:length(a(1,:))
            bottom(j)=(length(a(:,j))-1);
end
```

```
bottom=sum(bottom);
```

k=top/bottom;

```
PooledVariance = sqrt(k);
```

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## III(c) Matched-pairs Rank Biserial Correlation Coefficient

#### Limitations

This is the effect size used with non-parametric matched pair's datasets. If the data is not matched pairs Glass rank biserial correlation coefficient should be used. The results are potentially useful after a Wilcoxon analysis for generating effect sizes following Wilcoxon or Friedman tests. The process is detailed in Kinnear and Gray (2010) PASW 17 Statistics made simple

#### Loading the Raw Data

x=xlsread('data'); low = x(:,1); high = x(:,2);

### **Establishing and Ranking Differences**

dif=low-high; abDIF = abs(dif); rank = tiedrank(abDIF);

Note, tiedranks only ranks the dataset, it does not account for the fact that non-parametric analysis requires differences amongst the datasets being analysed to be ranked.

#### Adjusting rank

```
This section adjusts ranks to remove data points with equal values
for i=1:length(abDIF)
       if abDIF(i)==0
              countZEROS(i)=1;
       else
              countZEROS(i)=0;
       end
end
count=sum(countZEROS);
rank=rank-count;
for i=1:length(rank)
       if rank(i)< 0
              rank(i) = 0;
       else
               rank(i) = rank(i);
       end
end
```

#### **Defining Negative and Positive Ranks**

```
for i=1:length(dif)
    if dif(i)< 0
        rank(i) = rank(i)*(-1);
    else
        rank(i) = rank(i);
    end
end</pre>
```

#### Summing the Negative and Positive Ranks

```
PRtotal=abs(sum(posRANK));
```

```
NRtotal=abs(sum(negRANK));
```

## **Calculating the Effect Size**

n=length(dif);

```
eSIZE=(4*abs((min([PRtotal NRtotal]))-((PRtotal+NRtotal)/2)))/(n*(n+1));
```

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## III(d) Benjamini Hochberg False Discovery Rate (FDR) for Adjusting P-values from Multiple Tests

While the FDR process has been used for data analysis within the PhD, this example MATLAB script is based on data from the textbook *Westfall et al., 1999 Multiple Comparisons and Multiple Tests Using SAS. P 34.* The rationale for this is that it allows anyone unfamiliar with the process to easily confirm the validity of the MatLab script/output. I have replicated the results from the textbook (p35). The initial rawP dataset below is the data from the textbook.

#### Limitations

While the FDR has maximum power out of the post hoc test options (see: Koen J. F. Verhoeven, Katy L. Simonsen and Lauren M. McIntyre, 2005, Implementing false discovery rate control: increasing your power. OIKOS 108: 643-647,), it should also be noted that the FDR is the least conservative of the post hoc tests. This is acknowledged in Westfall *et al.* and should be considered if the process is applied to high impact data e.g. pharmaceutical intervention studies.

#### Importing the data

rawP = [0.0001 0.0058 0.0132 0.0289 0.0498 0.0911 0.2012 0.5718 .8912 0.9011]';

#### **Establishing parameters**

n=length(rawP); [sorted order] = sort(rawP); adjustedP(n)=max(rawP)';

As BH is a step down procedure the maximum value must first be established. This allows a point the algorithm can work from. This also has the advantage of pre-allocating the vector the for loop will work through.

#### Adjusting the P Value

#### **Displaying the Results**

The array is converted into an array to help display the output; potentially this could have been completed earlier when sorting results.

compare=sortrows([order sorted adjustedP']);

I am using 'sortrows' here to % help the output be as easy to read as much as possible. 'sortrows' has the advantage of keeping the relationship between the rows within in an array.

results = dataset({compare 'HypothesisNum', 'Raw\_P', 'Adjusted\_P'})

results =

HypothesisNum	Raw_P	Adjusted_P
1	0.0001	0.001
2	0.0058	0.029
3	0.0132	0.044
4	0.0289	0.07225
5	0.0498	0.0996
6	0.0911	0.15183
7	0.2012	0.28743
8	0.5718	0.71475
9	0.8912	0.9011
10	0.9011	0.9011

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## Appendix IV NSCA One rep max protocol

This protocol is taken from:

Baechle, T and Earle, R. 2008, Essentials of Strength training and Conditioning.

- 1. Instruct the athlete to warm up with a light resistance that easily allows 10-10 repetitions
- 2. Provide a 1 minute rest period
- 3. Estimate a warm up load that will allow the athlete to complete three to five repetitions by adding
  - a. 10 to 20 pounds (4-9 Kg) or 5% to 10% for upper body exercise or
  - b. 30 to 40 pounds (14-18 Kg) or 10-20% for lower body exercise
- 4. Provide a two minute rest period
- 5. Estimate a conservative, near maximal load that will allow the athlete to complete two to three repetitions by adding
  - a. 10 to 20 pounds (4-9 Kg) or 5% to 10% for upper body exercise or
  - b. 30 to 40 pounds (14-18 Kg) or 10-20% for lower body exercise
- 6. Provide a two to four minute rest period
- 7. Make a load increase:
  - a. 10 to 20 pounds (4-9 Kg) or 5% to 10% for upper body exercise or
  - b. 30 to 40 pounds (14-18 Kg) or 10-20% for lower body exercise
- 8. Instruct the athlete to attempt a one repetition maximum (1RM)
- If the athlete was successful, provide a two to four minute rest period then go back to step
- 10. If the athlete failed, provide a two to four minute rest period, then decrease the load by subtracting:
  - a. 5 to 10 pounds (2-4 Kg) or 5% to 10% for upper body exercise or
  - b. 15 to 20 pounds (7-9 Kg) or 10-20% for lower body exercise

AND then go back to step 8

Continue increasing or decreasing the load until the athlete can no longer complete one repetition with proper technique. Ideally the athlete's 1RM will be measured within three to five training sets.