# The efficacy of manual versus local vibratory massage in promoting recovery from post-exercise muscle damage - a systematic review

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### DECLARATION

I, Silungile S Ntshangase, student number 204500491, declare that the work on which this project is based is original and my own work (except where acknowledgements indicate to the contrary) and that neither the whole work nor part thereof has been, is presently or is to be submitted for another degree at this or any other university.

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#### ABSTRACT

**Background.** It is well established that unaccustomed activity/exercise with a large eccentric component causes the development of muscle damage which results in soreness and a decline in the functional capacity of muscle. Although this usually subsides after 3-4 days of relative inactivity, it temporarily impedes the level of performance in sports by preventing training and leading to greater susceptibility to injuries. Manual massage (MM) is a therapeutic modality that has been utilised in management of this condition for centuries with most of its accepted benefits being based on anecdotal reports and its functional benefits remaining contentious. Vibration therapy (VT) has on the other hand, recently gained popularity and replaced more time consuming manual massage. Its effectiveness is however also still in question as there is not enough clear scientific evidence regarding its efficacy in overcoming the consequences of exercise-induced muscle damage (EIMD) when compared to those of MM.

Aims. To conduct a systematic review examining the efficacy of MM as compared to the local vibration therapy (LVT) modalities on recovery from EIMD and to determine its efficacy in attenuating the negative effects of EIMD on measures of joint flexibility, muscle strength/power output, muscle soreness/DOMS, systemic markers of inflammation and blood lactate concentration and / or markers of fatigue.

**Method.** Following extensive computerised literature searches carried out using PubMed/MEDLINE, ResearchGate, EBSCOhost, Google Scholar and Science Direct, and a comprehensive literature review, randomized controlled trials and counter balance trials focusing on the beneficial effects of MM and LVT, were located. Criteria required for inclusion of trials in a systematic review were determined. After screening of the 63 initial studies located, articles that did not meet the inclusion criteria were excluded. The findings in terms of the effects of MM and LVT in terms of measures of joint flexibility, muscle strength/power output, muscle soreness/DOMS, systemic markers of inflammation and blood lactate concentration and /or markers of fatigue, were presented in tabular format, differentiating between the MM and LVT. A binary outcome summary for the trials in each category in which similar methodology was used, was created. Fisher's Exact test was conducted to establish whether the difference between MM and LVT for each outcome measure was statistically significant or not. Finally, the findings of the systematic review were compared to those of Imityaz *et al.* (2014). **Results:** No trials reported a positive effect of MM on joint flexibility, while 50% (n=2) showed a beneficial effect of LVT. Of the 11 trials located investigating the effects of MM on strength, 4 (36%) revealed an attenuation of force deficit, while 50% (n=2) of the 4 trials on the effects of LVT showed a positive effects. DOMS was attenuated following EIMD in 75% (n=9) of the trials following MM and 100% (n=4) following LVT. Blood creatine kinase concentration was reduced in 50% of trials following MM (n=2) and LVT (n=1). No reduction in blood lactate concentration or markers of fatigue was shown following MM or LVT. Fisher's Exact test showed no significant difference in the efficacy of MM and LVT in attenuating the effects of EIMD (p>0.05).

**CONCLUSION:** A systematic review of the literature confirmed that MM is no more effective in controlling functional declines and physiological response to EIMD than LVT. However, most studies had limitations and methodological flaws and frequently reported conflicting results. The number of randomized controlled studies qualifying for review was also small (n=28).

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## LIST OF ABBREVIATIONS

ACTH	adrenocorticotrophin hormone
α	alpha
β	beta
BF	biceps femoris
BP	blood pressure
$Ca^{2+}$	calcium ions
CCT	clinical controlled trial
CON	concentric
CG	control group
CGRP	calcitonin gene-related peptide
CORT	cortisol
СК	creatine kinase
CNS	central nervous system
СРК	creative phosphokinase
CRP	c-reactive protein
DEG	degree
DOMS	delayed onset muscle soreness
DSTM	dynamic soft tissue mobilization
EIMD	exercise-induced massage damage
ELISA	enzyme-linked immunosorbent assay
EMG	electromyography
GTO	golgi tendon organ
h	Hours
$\mathrm{H}^+$	hydrogen ions
HR	heart rate
IL	Interleukin
ISO	isometric
IMVC	isometric maximum voluntary contraction
MVC	maximum voluntary contraction

LVT	local vibration therapy
LDH	lactate dehydrogenase
LFF	low frequency fatigue
MTG	massage therapy group
MTrP	myofascial trigger points
MM	manual massage
MMG	mechanomyography
MVIC	maximum voluntary isokinetic contraction
Min	Minutes
ΝΓκΒ	nuclear factor kappa B
PPT	pressure pain threshold
PGC-1a	peroxisome proliferator- activated receptor
	gamma coactivator 1-alpha
PCNA	proliferating cell nuclear antigen
RCT	randomised controlled trials
ROM	range of movement
RBE	repeated bout effect
REPS	Repetitions
1RM	one repetition maximum
SAV	sinusoidal vertical vibration
SES	stretch explosive strength
S & R test	sit and reach test
SR	sarcoplasmic recticulum
TNF-α	tumor necrosis factor alpha
UAC	upper arm circumference
VES	vibration explosive strength
VF	vibration with flexibility test
VL	vastus lateralis
VT	vibration therapy
VTG	vibration therapy group
VAS	visual analog scale
WBVT	whole body vibration therapy

## CHAPTER ONE Introduction

#### **1.** Background to the study

When skeletal muscles are exposed to exercise with a large eccentric component, as in resistance training or downhill running bouts, muscle fibers undergo trauma which, when excessive, causes muscle damage or injury (Charge and Rudinicki, 2004; Friden *et al.*, 1992). Particularly following unaccustomed eccentric exercise, the athlete experiences delayed onset muscle soreness (DOMS). This begins to develop during the first 24hours (h) after exercise (Hilbert *et al.*, 2003) and peaks at 48-72h, depending on the severity of the exercise induced muscle damage (EIMD) (Tiidus, 1997; Armstrong, 1984).

It is well accepted that skeletal muscles have the ability to regenerate after injury (Kawiak *et al.*, 2006) and the regeneration process has been found to be similar in most types of muscle injuries (Baoge *et al.*, 2012). Although there is no existing treatment that is accepted as a universally used tool (Ernst, 1998), massage is commonly used in the athletic community despite little scientific evidence confirming its effectiveness (Stamford, 1985). As an alternative treatment it has been described as effective and safe in reversing or controlling moderate inflammation, improving blood flow, reducing DOMS (Crane *et al.*, 2012) and has remained popular in sport with many athletes requesting massage treatment following heavy training sessions and during/after competitive events (Hilbert *et al.*, 2004). Mackenzie (2000) estimates that up to 45% of current physiotherapy treatment in sport consists of massage. Much money and time has been invested by athletic teams to provide sports massage for their participants trusting that it will reduce exercise-induced functional muscle stiffness and strength losses, muscle soreness and enhance recovery (Tiidus, 1997).

Manual massage (MM) is well accepted to be one of the earliest forms of physical therapy known to man and has been used by many different cultures for more than 3000 years (Callaghan, 1993). But it was only in the 19<sup>th</sup> century that the research started investigating the efficacy of this form of massage which varied from light effleurage (stroking) to deep kneading (petrissage), frictions and tapotement, in enhancing post injury recovery (Callaghan, 1993). The popularity of MM as a treatment modality has, however, declined recently with (i) the development of the pharmaceutical industry, (ii) new machines supplanting older forms of physical therapy and (iii) the dehumanisation prevalent in the therapist-to-patient relationship (Callaghan, 1993). One example of modern technological advances is the current use of mechanical vibratory massage as a mode of physical therapy which has grown substantially in the last two decades (Summer and Pletcher, 2014).

Although it has been used in physiotherapy for pain management and by athletes to supplement their training, vibration therapy (VT) is one of the interventions that has not been studied extensively (Lau and Nosaka, 2011). It was first used by Jean-Martin Charcot, a French neurologist in patients with Parkisons' Disease (Vegar and Imtiyaz, 2012) and Nasarov was the first to use VT in sport believing that it helped to improve athletes' performance (Nazarov, 1987). According to Vegar and Imityaz (2012), VT helps with the synchronization of motor unit activity by preventing sarcomere disruption and also improves muscular strength, power development and kinaesthetic awareness.

Callaghan (1993) argues that MM is too time-consuming a technique for physiotherapists and highlights that research reports found in English publications are contradictory concerning how long the technique is to be performed in order to give a positive result, whereas localised VT (LVT) applied with the use of electrically or battery powered hand-held devices, is found to be faster and more uniform. It has been suggested that the physiotherapist must rather supervise and perform passive stretches and warm-ups in getting athletes ready for competing than spending extended time massaging athletes (Callaghan, 1993).

Due to the growing market for the sale of mechanical vibratory equipment for this modern massage modality, the question must therefore be asked whether these are as or more effective than MM and whether they can be used as a substitute for MM by therapists in the treatment of EIMD or sport injuries.

As the body of literature available remains relatively small and a lack of consensus exists regarding many of the hypothesised functional benefits of MM and VT a systematic review with possible subsequent meta-analyses was deemed appropriate in order to thoroughly examine the research and literature to date with the objective to guide practice and identify gaps in the literature providing directions for future research in the field of the efficacy of various massage modalities and their practical application.

#### 1.2 Aim and Objectives

The broad aim of this study is to systematically review the early and more recent scientific literature on the comparative effects of traditionally used MM as opposed to the more modern electrically powered local vibratory therapy modalities on recovery from EIMD. This will provide an indication of whether LVT is as effective as manually executed massage in the treatment of EIMD.

The specific objectives of this study focussed on specific outcome measures including the following physical measures of muscle function and biochemical/immunological parameters:

- measures of joint flexibility
- measures of muscle strength/power output

- muscle soreness/DOMS
- systemic markers of inflammation
- blood lactate concentrations or markers of muscle fatigue

#### **1.3 Hypotheses**

According to the knowledge of the writer, lack of consensus exists regarding the relative efficacy of MM and LVT in accelerating recovery following EIMD. The following null-hypotheses were thus set prior to the study:

1.3.1 There is no difference between the relative effects of MM and LVT on post-exercise joint flexibility

1.3.2 There is no difference between the relative effects of MM and LVT on post-exercise muscle strength or power

1.3.3 There is no difference between the relative effects of MM and LVT on post-exercise DOMS and muscle soreness

1.3.4 There is no difference between the relative effects of MM and LVT on post-exercise systemic markers of muscle inflammation

1.3.5 There is no difference between the relative effects of MM and LVT on post-exercise and blood lactate concentrations or markers of muscle fatigue.

#### 1.4 Scope of the Study

Various computerised literature searches were conducted using ResearchGate, Cochrane Library, EBSCOhost, Science Direct and PubMed/MEDLINE. Only human randomised controlled trials (RCT) on MM or VT implemented following strenuous exercise involving a large eccentric component, were selected. A total of 28 studies which met all inclusion criteria and focussed on one or more of a set of five pre-selected outcome variables, were included in a systematic review.

#### CHAPTER TWO

#### **Review of the Related Literature**

Although massage has been used by physiotherapists in the treatment of EIMD, little is known of the histological effects of different types of massage on recovery from eccentrically induced muscle damage and its effect on muscle function. This review of the related literature will examine the current state of the knowledge regarding (i) EIMD and (ii) the efficacy of MM and VT in reducing the impairments in muscle function, muscle soreness and systemic markers of an inflammatory response to this muscle damage. It will focus on both animal and human experimental models and will conclude by highlighting gaps in the literature and areas for future research.

#### 2.1 Ultrastructural changes following eccentric contraction

#### 2.1.1 Concentric versus eccentric contraction:

As is well described in standard human physiology text books, the contractile unit of the skeletal muscle consists of myofibres possessing longitudinal myofibrils which lie parallel to each other and are made up of a large number of primarily thin, actin filaments and thick myosin filaments **Figure 2.1**, which run from one z disk to the next, comprising sarcomeres (Guyton and Hall, 2011).



Figure 2.1 Microstructure of the human skeletal muscle. Adapted from Powers and Howley (2005).

An anchoring plane for the thin actin filaments is provided by Z disks lying in the middle of the light actin-containing inotrophic (I) band which is pulled towards the centre of each sarcomere as the muscle shortens during concentric muscle contraction (Armstrong, 1990).

During eccentric contraction, the muscle however contracts while it is lengthened. This is demonstrated in **Figure 2.2** in which a participant lowers his forearm and lengthens the biceps muscle while the weight in his hands is lowered.



Figure 2.2 Eccentric muscle contraction in the biceps muscle

As the muscle lengthens, non-contractile proteins including titin, desmin, talin, vinculin and dystrophin keep the sarcomeres attached to the cell membrane (Schwane and Armstrong, 1983) and work together to maintain the integrity of the muscle. When they fail to achieve this, severe disruption of the microstructure of the muscle, including torn actin and myosin filaments and disruption of the anisotrophic (A) & I bands, central nuclei, swollen mitochondria and displaced organelles, can result (Newham *et al.*, 1983, Friden *et al.*, 1983, Gibala *et al.*, 1995). As is shown in **Figure 2.3**, this can include single and half disrupted sarcomeres with streaming and widening of Z disks.





microstructure of the skeletal muscle following eccentric contraction (Reprinted with permission from Feasson *et al.*, 2002).

#### 2.1.2 The presence and function of the non-contractile proteins

Regarding the cytoskeletal elements which are important in maintain Z-disk structure and membrane integrity, titin (**Figure 2.4**) keeps the thick filaments centred by binding the myosin to the Z-disc and strengthening this disk as it interlocks within the disk and also adds passive force increase to the production of the force of the muscle (Bubbico and Kravitz, 2010). Its' absence when the muscle is stretched, results in failure of interdigitation that may also lead to membrane damage (Morgan, 1990, Proske and Morgan, 2001) and misalignment of myosin filaments (Morgan and Allen, 1999).



Figure 2.4 The microstructure of muscle with emphasis on the non-contractile cytoskeletal proteins. Adapted from website http://: www. Imgarcade.com.

Another important non-contractile protein which prevents disruption of the cytoskeleton is desmin. It is one of the proteins located at the level of the Z disk (Lieber *et al.*, 1996) that is responsible for horizontal structure of the sarcomeres (Waterman-Storer, 1991) and transmission of tension both longitudinally and laterally (Morgan and Allen, 1999). As shown in **Figure 2.5**, it maintains the sarcomere's appearance and connects adjacent Z lines from myofibrils and has been shown to transmit



force from myofibrillar force (active and passive generators to the muscle surface and to the muscle tendon junction. It is also involved in the positioning, distribution and function of the mitochondria (Paulin and Li, 2004).

Figure 2.5 The cytoskeletal protein, desmin. Adapted from website http//: www.

Laminin is an important component of the extracellular matrix that is primarily needed for building of skeletal muscle during embryonic developmental stage (Zou *et al.*, 2014). It has been related to

enhancement of satellite cell proliferation and its role in the post exercise regenerative muscle function has also been recorded in mouse models of muscular dystrophy exposed to downhill running.

Other non-contractile structural proteins include nebulin which is a large protein that primarily acts as a ruler to regulate thin filament length (Wang *et al.*, 1996) and talin which links the cytoplasmic domain of integrin beta subunits to actin filament (Critchley and Gingras, 2008). Talin is important for attachment of the filaments to the lipid bilayer (Drenckhahn and Franke, 1988) and is expressed in the distal tip cell. Reduction or lack of this protein results in severe defects in gonad formation because of aberrant distal tip cell migration and disruption of oocyte maturation (Cram *et al.*, 2003). Vinculin is a membrane cytoskeletal protein found in focal adhesion plaques that is involved in the linkage of integrin adhesion molecules to the actin cytoskeleton (Brown *et al.*, 1997) whereas dystrophin forms a link between actin and associated cytoskeleton and is found throughout the sarcolemma including the neuromuscular junction. Studies have indicated that lack of dystrophin may lead to muscular dystrophy and may result in permanent damage to fibers. Muscle fibers lacking dystrophin have also been shown to be more susceptible to eccentric damage and it has been suggested that EIMD may lead to some muscle diseases (Morgan and Allen, 1999).

In conclusion, the presence of these non-contractile proteins enhances the stability of the muscle structure minimising the amount of ultrastructural damage which occurs in the muscles following eccentric exercise.

#### 2.1.3 Systemic markers of post-exercise inflammatory response in venous blood

**2.1.3.1.** Creatine phosphokinase (CPK) and lactate dehydrogenase (LDH): With EIMD to the muscle membrane, the intracellular enzymes present in muscle, creatine phosphokinase (CPK), more commonly referred to as creatine kinase (CK) and lactate dehydrogenase (LDH) leak into the blood and systemic concentrations increase (Brancaccio *et al.*, 2007). The mechanism of the increase in CK following muscle damage has been described as a consequence of both metabolic and mechanical factors. In addition to the local tissue damage with sarcomeric degeneration from Z-disk fragmentation, in 1976 Fink and Luttgau already described how metabolically exhausted muscle fibres exhibit a decrease in the membrane resistance following an increase in the internal free calcium ions (Ca<sup>2+</sup>), which promotes the activation of the potassium channel.

These two enzymes are thus commonly used as indirect indicators of EIMD. Blood LDL concentrations have been found to peak by 6h post exercise and return to resting levels by 48h post exercise (Maughan *et al.*, 1989), while CK concentrations are markedly elevated for 12-24 h after the exercise bout (Semple *et al.*, 2007).

Both LDH and CK however often show a large intra- and inter-individual variation (Peters *et al.*, 2005) and it should be taken into account that they are affected by other kinases. In the case of CK, these include mitochondrial CPK, CPK-immunoglobulin complexes and CPK derived from cardiac muscle (Martinez-Amat *et al.*, 2005). In addition, total levels have also been found to depend on age, gender, ethnicity, muscle mass, climatic condition and exercise mode and intensity (Brancaccio *et al.*, 2007; Mckune *et al.*, 2012), all of which can affect enzyme tissue activity and subsequent serum levels (Baird *et al.*, 2012). Furthermore, both CK and LDH comprise different isoenzymes that are distinguished by slight differences in their structure. In the case of LDH, the isoenzymes are LDH-1 (in the heart and red blood cells), LDH-2 (in white blood cells), LDH-3 (in the lungs), LDH-4 (in the kidneys, placenta, and pancreas), and LDH-5 (present in liver and skeletal muscle) (Epstein and Butler, 2015). Isoenzymes of CK include CK-MM (found in the muscle), CK-BB (found in the brain) and CKMB (found in the heart) (Grossman, 1982). An elevation of total blood concentrations is therefore not necessarily always well correlated with the severity of skeletal muscle damage (Peters *et al.*, 2005).

#### 2.1.3.2 Inflammatory response

Following muscle damage, an inflammatory reaction sets in and is associated with an invasion of neutrophils and macrophages into the damaged fibres within 6h (Armstrong *et al.*, 1991; Peake *et al.*, 2005). These secrete reactive oxygen and nitrogen species, cytokine factors and proteolytic enzymes that lead to initial tissue degradation (Clarkson and Sayers, 1999). Neutrophils remain present in the damaged muscle up to 24h post exercise, while resident macrophages are active for several days after EIMD and produce pro-inflammatory cytokines including interleukin (IL) -1 $\beta$  and tumor necrosis factor (TNF- $\alpha$ ) that activate the breakdown of damaged muscle tissue (Peake *et al.*, 2005), as well as IL-6, which initiates an anti-inflammatory response (Peters, 2004).

A further systemic response to EIMD and subsequent inflammation results in an acute phase response, similar to that which occurs with infections, surgery and trauma (Fallon, 2001). While muscle damage results in elevated levels of CK, myoglobin and LDH (Overgaard *et al.*, 2002), release of IL-6 from activated macrophages in the tissues and subsequently cortisol from the adrenal cortex stimulates hepatic production of C-reactive protein (CRP; Peters *et al.*, 2005; Semple, 2006).

Venous blood CRP concentration is therefore a sensitive indicator of the inflammatory response which is commonly used in exercise studies on humans. Studies have shown that circulating CRP concentrations rise substantially 24-48h after acute eccentric exercise (Peters *et al.*, 2005; Semple, 2006).

With EIMD and inflammation, the activation of nuclear factor kappa B (NF $\kappa$ B), a protein complex that controls transcription of DNA, cytokine production and cell survival, also occurs. Peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1 $\alpha$ ), primarily involved in the regulation of carbohydrates and lipids in the human body (Liang and Ward, 2006), stimulates mitochondrial biogenesis and promotes the remodelling of muscle tissue to a fibre-type composition that is metabolically more oxidative and less glycolytic in nature (Liang and Ward, 2006).

NF $\kappa$ B and PGC-1 $\alpha$  are therefore both mediators in the inflammatory process and play a role in cellular repair. Lowering NF $\kappa$ B levels reduces inflammation and increasing PGC-1 $\alpha$  levels leads to the creation of more mitochondria that generate energy for cell growth (Ward, 2012), also improving the contractile function of dystrophic muscle and reducing the level of inflammation (Crane *et al.*, 2012).

A prominent feature of an inflammatory response to EIMD is swelling in the isolated area around the injured tissue. Accumulation of fluid has been shown to result from influx of Na<sup>+</sup> into the muscle cells following cell membrane damage and increased membrane permeability (Mckune *et al.*, 2012) and the slow removal of muscle breakdown products such as protein fragments from the extracellular matrix, both of which attract water (Clarkson *et al.*, 1992). This swelling may peak 5-8 days after EIMD, beginning inside the muscle and spreading to subcutaneous spaces after about 5 days (Ezeilo, 2002).

#### 2.2. Delayed onset muscle soreness (DOMS)

DOMS is a predictable painful condition that begins within 24h after exercise, peaks at 48 to 72h after unaccustomed eccentric exercise and may last several days (Ernst, 1998; Mancinelli *et al.*, 2006). Its intensity is closely associated with the degree of structural damage and intensity of the eccentric exercise (Pilladi *et al.*, 2013). The symptoms of DOMS include a decrease in ROM and joint stiffness, reduction in muscle strength, muscle pain and tenderness. In terms of the mechanism, exercise-induced mechanical disruption of muscle cell sarcomeres and sarcolemmas, and a reduced membrane excitability (Armstrong *et al.*, 1991; Fitts, 1994) leads, during repeated contractions, to more extensive damage and, ultimately, to necrosis of some muscle fibres. The injury triggers a local inflammatory response that is accompanied by some oedema and the breakdown products of injured tissues, sensitise nociceptors (Proske and Morgan, 2001). The physiological mechanisms associated with the reduction of muscle cell sarcomeres are thought to be the exercise-induced mechanical disruption of muscle exercise are thought to be the exercise-induced mechanical disruption of muscle strength post eccentric exercise are thought to be the exercise-induced mechanical disruption of muscle cell sarcomeres and sarcolemma, and a reduced membrane excitability (Armstrong *et al.*, 1991; Fitts, 1994). In patients with exercise-induced DOMS during the first three days following unaccustomed eccentric exercise, studies have also confirmed disorganization of myofibrillar proteins, especially in and around the Z-disk (Cluett, 2010; Bubicco and Kravitz, 2010). This includes the

absence of the main z-disc protein,  $\alpha$ -actinin, in z-disc alterations (Yu and Thornell, 2002; Yu *et al.*, 2004) that appeared to be related to the increase in desmin.

These findings support those of earlier rodent studies including those of Barash *et al.* (2002) in which desmin content and immunohistochemical appearance were increased following eccentric exercise in rats that were subjected to single bout of exercise consisting of 30 eccentric contractions induced by stimulation of the peroneal nerve for 650ms at 100Hz with a small nerve cuff at two min interval with the contralateral leg serving as the control. Loss of desmin staining occurred 12h post eccentric exercise, and full recovery was seen by 72h. It was suggested that the desmin content in each cell may thereafter have increased by 15fold between 72-168h post eccentric contractions. In this study, increased expression of desmin was associated with muscle protection from exercise-induced injury.

However, it currently remains unclear whether myofibrillar disruption or membrane breakdown is the result of eccentric contraction (Friden *et al.*, 1981; Petrof *et al.*, 1993; Lynch *et al.*, 2000).

A large study of 344 male Fischer rats subjected to downhill interval treadmill running for 90 min (Armstrong *et al.*, 1983), on excitation-contraction (EC) coupling showed four ultrastructural changes in the arrangement of the transverse (t)-tubules and the disposition of triads after the downhill running exercise. Greater changes were observed 2-3 days post eccentric exercise.

In terms of the possibility of membrane disruption occurring with EIMD, animal models have revealed that EIMD will also result in disruption of the sarcolemma of muscle during the first hour post exercise (Yu *et al.*, 2002). Lieber *et al.* (1994) reported that in the rabbit model, cytoskeletal disruption including a loss of immunostaining of desmin, was followed by increased membrane permeability. In 1996 this laboratory reported that the sarcolemmal proteins including dystrophin and laminin were also completely disrupted following EIMD (Lieber *et al.*, 1996).

Recent human studies, on the other hand, have not confirmed that eccentric exercise that results in DOMS causes sarcolemmal disruption and loss of desmin (Yu *et al.*, 2002; 2013). Lieber and Frieden 2002) and Paulsen *et al.* (2012) have suggested that contraction induces extracellular or intracellular membrane disruption that may induce hydrolysis of structural proteins such as the desmin intermediate filament network and myofibrillar disorganization in form of z-band streaming or complete disruption. It is further hypothesised that this results in fibre necrosis and inflammatory cell infiltration that activates the nerve endings and perception of pain. This possibility however requires further examination and Yu *et al* (2013) suggest that the search for greater clarity regarding the exact mechanisms explaining the functional and structural alterations in human skeletal muscles after eccentric exercise, should continue.

Vasodilatation following massage has been shown to be caused by the release of neuropeptide Y (NPY) and calcitonin gene-related peptide (CGRP), potent vasoregulatory neuropeptides (Jönhagen *et al.*, 2004). These peptides have also been shown to be involved in the modulation of pain and increased detectability of CGRP in human muscle using microdialysis followed by radioimmunoassay after hard eccentric exercise and have been related to increased DOMS (Jönhagen *et al.*, 2006). This supports an earlier finding of Homonko and Theriault (1997) who reported increased concentrations of CGRP in the motor neurones of medial gastrocnemius muscles in rats 72h after eccentric downhill running confirming that the peripheral nervous system initially reacts to EIMD by releasing CGRP.

In addition to nociceptive, vasoregulatory and proinflammatory actions, CGRP has also been suggested to exert trophic effects, activating proliferation of fibroblasts and endothelial cells in response to stress. Detection of CGRP after eccentric exercise, can therefore also be assumed to reflect tissue regeneration (Jönhagen *et al.*, 2006).

Eccentric exercise and EIMD also results in the production of prostaglandin E2, lipid autocoids derived from arachinonic acid and produced in response to inflammation. In addition to causing intracellular fibre swelling, elevated circulating prostaglandin E2 concentrations sensitise group IV afferent fibres of muscle connective tissue associated with a dull aching pain and stiffness (Friden *et al.*, 1988; Clarkson and Hubal, 2002; Proske and Allen, 2005; Lewis *et al.*, 2012). Damage to the sarcoplasmic reticulum (SR) and structure of the t-tubules has been shown to impair its release of  $Ca^{2+}$  and result in activation of proteolytic enzymes which degrade structures within the muscle fibre resulting in fibre swelling, muscle soreness and inflammation (Armstrong *et al.*, 1991).

Descriptive rating scales commonly used to measure pain intensity experienced following exerciseinduced DOMS originated in psychological and medical experimentation (Freyd, 1923; Kneele, 1948). They most commonly include the visual analog scale (VAS) shown in **Figure 2.6** in which individuals are asked to rate their pain arbitrarily on a continuous 10cm line with varying degrees of perceived pain intensity indicated below the line (Drake *et al.*, 2012). Today variations of these scales are commonly used in clinical and experimental settings and graduated to assess the effectiveness of treatment interventions. Weber *et al.* (1994), for example, examined the effect of three different therapeutic modalities in untrained female subjects following high-intensity eccentric contractions of the elbow flexors which induced DOMS and reductions in muscle strength, using a seven- point soreness rating scale (Talag, 1973) to collect soreness perception data. Subjects verbally reported the number on the scale that best corresponded with their symptoms when the extremity was at rest and returned after the pretest to repeat the soreness rating at 0, 24, or 48h.



**Figure 2.6** Comparison of Visual Analog and Graphic Rating Scales for Assessing Pain following Delayed Onset Muscle Soreness. Adapted from Mattacola *et al.* (1997)

Of further interest to the focus of this thesis is that in recent work examining DOMS, inactive transcutaneous electrical nerve stimulation (TENS) pads are frequently used as placebo in partially blinded studies. TENS is a non-pharmacologic treatment for pain relief electric current produced by a device is used to stimulate the nerves for therapeutic purposes (DeSantana *et al.*, 2008). TENS pads are usually connected to the skin using two or more electrodes and cover the complete range of transcutaneously applied currents used for nerve excitation although the term is often used with a more restrictive intent, namely to describe the kind of pulse produced by portable stimulators used to treat pain.

#### 2.3 Performance decrements following exercise-induced muscle damage

#### 2.3.1 Muscle strength

A prolonged increase in strength loss is an indicator of EIMD and considered as one of the most reliable indirect markers thereof (Eston *et al.*, 2007). The level or amount of muscle strength loss depends on the intensity of the eccentric exercise and its duration (Sayers and Hubal, 2008).

Numerous studies have reported the loss ability of the eccentrically induced damaged muscle to generate force (Brown *et al.*, 1996). Clarkson *et al.* (1992) reported a post exercise strength loss of 50% and that it that took more than 10 days for the muscle to recovery fully, whereas in the study of Howell *et al.* (1993) strength loss of 30% was evident on the day following the exercise with about 70% of muscle recovery occurring by 3 days post exercise.

In terms of the physical mechanism, it has been attributed to the length – tension relationship of muscle and overstretching of sarcomeres (Howell *et al.*, 1993). During eccentric contraction and exercising at longer compared to shorter muscle lengths, a greater number of sarcomeres are operating on the descending limb of this length- tension curve and are stretched to the point of "no myofilament overlap." Failure to re-interdigitate results in their disruption (Eston *et al.*, 2007).

McKune *et al.* (2012) emphasises that the small volume of tissue damage immediately after eccentric exercise does not account for the large reduction in strength and that this lack of association has led to the contention that much of the early loss of function following eccentric contractions results from impairment of excitation-contraction coupling (Sayers and Hubal, 2008). This has in turn, been related to disruption of the  $Ca^{2+}$  cycling mechanism and is a well described cause of the muscle strength deficit following EIMD. Koh (2008) suggests that impaired  $Ca^{2+}$  release from the SR occurring with EIMD is responsible for reduced excitation-contraction coupling and that this impairment could occur at any point in the chain of events between depolarization of the muscle cell membrane and the release of  $Ca^{2+}$  from the SR which has important implications for the post exercise force deficit as  $Ca^{2+}$  is required for force production in the myofibrils.

The initial physical damage of individual muscle fibres during eccentric contractions that may cause disruption of the normal permeability barrier provided by the cell membrane as well as disturbed functioning of the SR, allows  $Ca^{2+}$  which is present in higher concentrations in the extracellular spaces, to enter the fibre down its electrochemical gradient, causing intracellular  $Ca^{2+}$  accumulation (Armstrong *et al*, 1991). If the  $Ca^{2+}$  influx overwhelms the  $Ca^{2+}$  pumps and free intracellular  $Ca^{2+}$  concentrations rise, these activate a number of  $Ca^{2+}$ -dependent proteolytic and phospholipolytic pathways and degrade structural and contractile proteins and membrane phospholipids (**Figure 2.7**). For example elevation of intracellular  $Ca^{2+}$  levels results in loss of CK activity from the fibres through activation of phospholipase A2 and subsequent production of leukotrienes (Armstrong *et al.*, 1991). In addition, the lower  $Ca^{2+}$ -intracellular availability for the contractile process results in reduced maximal contractile force.



**Figure 2.7** Reduced intracellular  $Ca^{2+}$  concentrations results in a force deficit accompanied by an inflammatory response following eccentric exercise (adapted from McKune *et al.*, 2012).

#### 2.3.2 Joint flexibility

A reduction in voluntary range of motion (ROM) in joints is another consistent finding following EIMD (Callaghan, 1993) which is also regarded as one of the most valid and reliable indicators of EIMD (Armstrong, 1990). ROM decreases immediately following exercise and can be reduced by 20-45 degrees. Although recovery begins within 24h, full recovery is not achieved until 10 days after exercise (Jones, 1987). Joint flexibility has been quantified by measuring actual change in range of ROM within joints using goniometers (Clarkson and Sayers, 1999), changes in muscle length (Eston *et al.*, 2007) or actual standard flexibility tests such as the sit and reach (S&R test; Barlow *et al.*, 2004).

In terms of the mechanism, Clarkson and Sayers (1999) attributed the "stiffness" of the muscle following EIMD to the ultrastructural damage with consequent excitation - contraction coupling and cross-bridge formation which results in the muscle fibres failing to return to their optimal resting length. This increase in the number of contracted fibre segments has been suggested to related to the previously described elevation in resting cytosol  $Ca^{2+}$  levels which occurs following EIMD (Sayers and Hubal, 2008). Furthermore, the reduction in ROM following EIMD may also be partly attributed to a concomitant increase in fluid accumulation and swelling (McKune *et al.*, 2012).

#### 2.3.3 Muscle fatigue

Eccentric exercise produces a long lasting low frequency muscle fatigue (Clarkson and Sayers, 1999). Skeletal muscle fatigue is a progressive decline of muscle force production or power output that occurs during or after repeated muscle contractions. The mechanism causing fatigue is not easy to identify due to multivariate nature of the fatigue process and the complexity of the pathways involved (Kano *et al.*, 2012). Therefore, to provide insights into the mechanisms of muscle fatigue, many studies have been performed on isolated whole muscles or single fibres as an expedient to permit greater control over the experimental environment (Kano *et al.*, 2012).

Possible low-frequency muscle fatigue following acute high intensity has also been attributed to the above-described depression in the maximal rate of  $Ca^{2+}$  release and  $Ca^{2+}$  uptake from the SR immediately after exercise by Hill *et al.* (2001). Confirming the suggestions of Jones (1996) that a reduction of  $Ca^{2+}$  release by the SR was one of the likely mechanisms of low-frequency fatigue, he was the first to report the effect of an intense exercise bout on the functional characteristics of sarcoplasmatic  $Ca^{2+}$  release,  $Ca^{2+}$  uptake and  $Ca^{2+}$ -ATPase activity and muscle contractile characteristics in human muscle. Upon stimulation,  $Ca^{2+}$  required for the contractile process, is released by the SR resulting in muscle contraction; the subsequent removal of  $Ca^{2+}$  from the contractile proteins back into the SR by the SR  $Ca^{2+}$ -ATPase results in relaxation of the muscle.

During high-intensity exercise, accumulation of blood lactate (Tesch *et al.*, 1978) and the associated fall in pH have also been attributed to the development of muscular fatigue (Brooks, 1991). The decrease in pH associated with elevated levels of lactate production is well known to impede performance by inhibiting key glycolytic enzymes (MacClaren *et al.*, 1989) and myosin-ATPase (Peters, 1984) causing performance deficit. The rate of lactate removal which has been shown to correlate with H+ uptake from the blood (Peters, 1984) is therefore indirectly indicative of the recovery process and crucial to the successful performance of repeated bouts of exercise (Tesch *et al.*, 1978; Peters-Futre *et al.*, 1987).

Removal of blood lactate occurs through various mechanisms in the body. Tracer studies have shown that during light exercise most of the lactate is taken up by skeletal muscle, reconverted to pyruvate, and then oxidised in the mitochondria via the Krebs cycle (Brooks and Gaesser, 1980). Other major organs of lactate uptake post exercise include the heart, the inactive muscle fibre and the liver (Peters, 1984).

Previous research has demonstrated that the elimination of post exercise blood lactate can be optimised by light activity (30-45% VO<sub>2</sub>max) since moderate activity increases cardiac output and blood flow to the lactate-consuming tissues, increasing the rate of post-exercise lactate uptake (Peters-Futre *et al.*, 1987). Some researchers have speculated that blood lactate removal might also be accelerated during massage recovery due to increased blood flow and therefore better distribution of lactate to the tissues responsible for lactate-uptake (Wiktorsson-Moller *et al.*, 1983).

It is, however, currently accepted that it is not the removal of the lactate *per se*, that results in muscle fatigue, but rather the associated release of  $H^+$  ions and drop in muscle pH which is well correlated with a rise in blood lactate concentrations (Peters, 1984). Interestingly, the uptake of  $H^+$  ions during the post-exercise recovery, is equally optimised by an increase in blood flow to the skeletal muscle (Peters-Futre *et al.*, 1987), but research examining the effect of massage on  $H^+$  ion uptake post EIMD does not appear to have been conducted.

#### 2.4 The repeated bout effect (RBE)

The ultrastructural disruption seen after eccentric contraction and the level of disruption is known to be reduced if the subjects have been previously exposed to this type of training. This phenomenon is known as the repeated bout effect (RBE; Cheung *et al.*, 2003). It refers to the adaption where a single bout of exercise strengthens the muscle for the following eccentric bouts (McHugh, 2003) However, the contraction intensity of the initial bout must be high enough to induce a protective effect against subsequent eccentric bouts. Nosaka *et al.* (2001) stated that even as few as two eccentric contractions that are performed at a maximum level have been shown to induce this protective adaptation, whereas

Maes and Kravitz (2003) reported that repeated bouts of lower intensity eccentric exercise performed 1-6 weeks before the initial higher intensity eccentric bouts have been shown to consistently reduce DOMS and EIMD. Evidence confirms that during the RBE there is a shift towards recruiting slow twitch motor units and cellular changes with addition of sarcomeres and inflammation. These include cytoskeletal adaptations as the first line of defence to protect against damage (McHugh, 2003). Barash *et al.*(2002) contend that the increase in desmin content that was demonstrated in a study of rats following damaging contractions could provide mechanical reinforcement against excessive sarcomere strain caused by a repeated bout of eccentric exercise. Paulsen *et al.* (2012) have also shown that passive and active movements alter intramuscular pressure and cause stimulation of mechanoreceptor nerve endings. However, this was not supported by recent studies where z-streaming was proposed to be the hallmark of muscle damage after eccentric exercise (Friden *et al.*,1981; Friden and Lieber, 1992) and to represent myofibril remodelling (Yu *et al.*, 2002, 2003, 2004; Carlsson *et al.*, 2007, 2008). According to McHugh (2003) one specific mechanism for RBE has not been identified.

#### 2.5. Manual massage (MM)

#### 2.5.1 Introduction

Massage has been promoted as a means to help prepare athletes for competitions and to improve the performance and recovery of injury after exercise (Cassar, 2004). It is one of the modalities that has been used since early civilisation and is thought to have benefits in sport (Callaghan, 1993) and its use has increased in the past years (Jönhagen et al., 2004). There are various forms of MM that are being used, but the basic techniques of "classic massage" remain unchanged and are also referred to as "Swedish massage" (Hofkosh, 1985; Holey, 1991). These include effleurage (light stroking), petrissage (kneading, also referred to as Tuina Therapy or deep tissue massage) and frictions. Although some also identify deep tissue massage, myofascial release, trigger point and craniosacral massage, the three main techniques used in sport are primarily a combination of effleurage, petrissage and frictions. The majority of research has used Classic Western massage or Swedish massage to investigate the effects of MM with very few studies having used other techniques such as myofascial trigger point massage or focused on one individual technique (Reynolds, 2010; Callaghan, 1993). Although post-event MM may have a substantial effect in reducing post-exercise fatigue level and helping recovery when applied to athletes (Wakim, 1985), massage is however also used by athletes before an event (Callaghan, 1993) and found to be of value (Balke et al., 1990). As the focus of this dissertation is on the therapeutic value of MM in the post-exercise recovery process, this review will be restricted to studies MM used after exercise involving a large eccentric component.

#### 2.5.2 Effleurage technique

Although frequently used in sport massage, effleurage is not usually applied as a stand-alone technique. It consists of light strokes that are delivered by hand following the contour of the body, with no attempt to manipulate the deeper tissue (Cafarelli and Flint, 1992), but is usually used in preparation for deeper massage to the tissues and performed in the direction of the lymph and venous flow (Moraska, 2005). If conducted at the end of the massage, it is used for the purpose of relaxation or to achieve a "flushing" effect (Tappan and Benjamin, 2005).

In one of the earliest studies, Hansen and Kristensen (1973) examined the comparative effect of effleurage, shortwave diathermy and ultrasound on the clearance rate of inert gas using radioactive xenon <sup>133</sup>Xe to measure of capillary blood flow (Aladj *et al.*, 1991), in the *triceps surae* of the right calf. After 5minutes (min) of application of these three techniques to the calves of 12 human subjects, effleurage was found to result in increased <sup>133</sup>Xe clearance from the muscle (p<0.01). This, however, decreased within 2min after cessation of the effleurage, becoming negative by 10 min after the massage. Shortwave diathermy and ultrasound induced no significant changes in the <sup>133</sup>Xe clearance from muscle.

In follow up work including a focus on the functional benefits of effleurage alone, Tiidus and Shoemaker (1995) studied the effect of this modality on post-exercise recovery in healthy volunteers with one leg assigned to massage and the contralateral leg acting as control. Ten minutes of effleurage was administered for 4 days post-eccentric exercise. During this 96h post-exercise recovery period there were no differences in the rate of recovery of isometric and dynamic *quadriceps* peak torque measures in the massaged and control leg. Blood flow, determined using pulsed Doppler ultrasound velocimetry, was also not improved significantly in the massaged limb, while muscle soreness was only attenuated at 48-96h post exercise in the massaged leg. It was concluded that massage was not an effective treatment modality for enhancing long term restoration of post-exercise muscle strength and its use in controlling DOMS in sport should be questioned.

A subsequent clinical controlled trial conducted by Stanley *et al.* (2001) examined the passive tension and stiffness properties of the plantar flexors of the ankle joint in 19 subjects who received effleurage alone post eccentric exercise. They did not find a positive effect on passive muscle stiffness and maximum tension compared to a control group that had no significant intervention. In a more recent clinically controlled trial (CCT), Reynolds (2010) also found that 10min of effleurage had no significant effects on passive gastrocnemius muscle stiffness properties when compared with 10min rest in 12 patients and attributed this finding to the pressure of effleurage not being sufficient to produce mechanical effects. Fehrs (2010) however later proposed that effluerage stimulates the nerve endings and that this reflex effect may result in the release of neurotransmitters, vasopressin and oxytocin leading to a relaxation response, consequently decreasing blood pressure and reducing stress. This hypothesis requires further examination.

In 2012 Drake *et al.* re-examined the effect of 10min of effleurage immediately following an eccentric biceps protocol and found that there was no significant attenuation of pain or enhanced recovery of isometric torque deficit assessed using electromyography (EMG), despite a significant mechanomyography (MMG) interaction, confirming the earlier findings of Tiidus and Shoemaker (1995).

Despite the indication for further research that is necessary to confirm whether effluerage activates a relaxation response as described by Fehrs (2010), according to the work done to date, it can be concluded that there is little current evidence in favour of a beneficial effect of effluerage alone as a massage modality. This justifies its exclusion in a systematic review of the optimal physiological effects of MM in recovery from EIMD.

#### 2.5.3 Petrissage and effleurage combined with petrissage

The kneading technique (petrissage) used on deeper tissues, is a technique in which the skin and underlying tissues are mobilized in circular and rotational motion. It is a powerful technique applied with the whole palm of the hand (Miernik *et al.*, 2011) which has been shown to be particularly effective in mobilizing fluids in very deep muscles and applying a stretch to the fibres involved (Mackenzie, 2000).

In sport, the kneading pressure used on athletes is generally higher compared to that applied on sedentary individuals (Jönhagen *et al.*, 2004). However, the scientific evidence regarding the physiological and functional benefits of the use of petrissage appears to lack consensus and the practice has remained controversial.

With regard to the effects of petrissage on neuromotor activity, there appears to be a dichotomy of contradictory responses. On the one hand Morelli *et al.* (1990) and Sullivan *et al.* (1991) focussed on the effect of petrissage alone in the proximal aspects of the *triceps surae* muscle of 16 adults without neuromuscular impairments on the Hoffman reflex (H- reflex). As a reflectory reaction of muscle response measured by electromyography (EMG) after electrical stimulation of afferent sensory fibres which bypasses actual activation of the muscle spindle, the H-reflex represents monosynaptic reflex activity in the spinal cord and is analogous to the stretch muscle reflex.



Figure 2.8 The monosynaptic stretch reflex (adapted from Dick, 2003).

The ipsilateral monosynaptic reflex is outlined in **Figure 2.8**. Stimulation of muscle spindles (receptors) that detect abrupt stretching in the muscle, activate the nerve impulse along the sensory neuron, stimulating ion channels in response to the stretch and depolarising the membrane and creating greater potential (Fritz, 2013). On reaching its threshold, the greater potential causes the ion channels to open and results in the action-potential spreading along the sensory neuron. This travels through the nerve that innervates that particular muscle back to the spinal nerve and to dorsal roots ganglion and into the spinal cord, synapsing immediately with the motor neuron. The motor neuron takes impulses into the ventral horn of the grey matter to the spinal nerve through motor neuron and back to the same muscle which is then stimulated to contract and shorten. This counteracts the initial stretch, protecting the muscle from overstretching (Ezeilo, 2002).

In comparison, the H-reflex involves activation of the spinal stretch reflex induced by electrical activation of a percutaneous mixed afferent nerve (Armstrong *et al.*, 2008). It is a viable laboratory–induced measure of motorneuron excitability and its suppression after fatiguing exercise as well as petrissage is well documented (Armstrong *et al.*, 2008; Sullivan *et al.*, 1991).

In well cited studies, Morelli *et al.* (1990) and Sullivan *et al.* (1991) reported reductions in H-reflex amplitude indicating an inhibition of spinal alpha ( $\alpha$ ) motorneuron excitability of short term duration while massage was administered. Morelli *et al.* (1990) reported that all massage-control pairings were statistically different (p < 0.01) with as much as a 71% decrease in H-reflex amplitudes observed during the massage, while the principal finding of Sullivan *et al.* (1991) was that MM reduced the amplitude of H-reflex of ipsilateral *triceps surae*. These researchers proposed that rapidly adapting cutaneous and/or muscle receptors may be responsible for mediating these responses, the intensity of which are dependent on the amount of pressure exerted. While the cutaneous receptors, activated by light fingertip pressure during effleurage, facilitate spinal reflex activity, stimulation of the deep tissue receptors by tendon pressure was proposed to inhibit activity along the reflex pathway as measured by the H-reflex. According to Morelli *et al.* (1990) this would imply transient relaxation and reduced power output of the muscles following petrissage.

On the other hand, an earlier study by Ask *et al.* (1987) in which 8 participants were assigned to either 10 min of effluerage and petrissage (massage group) or to a control group not receiving treatment in a

cross-over design study, reported that average power at 50% maximum voluntary contraction (MVC) after massage was 11% greater than in the untreated control group. These findings were supported by Brooks *et al.* (2005) who reported that 5min of manual effluerage and friction massage on the forearm and hand had a greater positive effect on power-grip performance immediately after maximal exercise in healthy adults than no treatment and/or stretching. While these researchers attributed this finding to a rise in circulating  $\beta$ -endorphin concentrations and metabolic recovery facilitated by massage-induced increases in circulation, the exact mechanism remains unclear.

As adequate flexibility and ROM are believed to be beneficial in terms of injury prevention and optimal muscular performance, considerable research has focussed on the effect of massage on this area. In addition to above-described inhibition of the H-reflex (Morelli *et al.*, 1990), massage is known to increase ROM and flexibility by affecting both the muscular and connective tissue (Moraska, 2005). One cause of injury may be due to myofascial trigger point (MTrP) activity. MTrPs are tightly contracted regions within muscle tissue characterised by a hyperirritable taut band with defined pain referral patterns and result in limitation of strength, loss of muscle function and muscle shortening (Moraska, 2005). Davidson *et al.* (1997) who reported that deep tissue massage and mobilization of surrounding soft tissue including MTrPs facilitated healing of Achilles Tendonitis in rats, attributed their findings to this mechanism.

The findings on the effects of petrissage (in most cases preceded by effleurage) on attenuating the contractile force deficit and reduced flexibility accompanying EIMD, are, however, not consistent. A thorough systematic review of well-designed CCTs is necessary to clarify the present state of the knowledge in this regard.

Most of these studies also investigated the effects of petrissage on markers of DOMS and muscle inflammation/fatigue associated with EIMD.

In 1994, Weber *et al.* examined the effect of 8 min of effleurage and petrissage when compared to (i) 8 min of microcurrent electrical stimulation, and (ii) 8 min of upper body ergometry in 40 untrained female subjects following high-intensity eccentric contractions of the elbow flexors which induced DOMS. There were no significant changes noted between the experimental and control groups in soreness rating, maximal voluntary isometric contraction, and peak torque recorded immediately and at 24 & 48h post exercise.

In 1997 Lightfoot *et al.* conducted a randomised controlled trial (RCT) showing that immediate post exercise massage does not attenuate DOMS or reduce the exercise-induced deficit in muscle strength in 12 males and 19 females. All subjects performed heel drop exercise 4x15 with 100% of their body weight and then 24h following this eccentric exercise, one group underwent a stretch routine, the

second, 10 min of petrissage and the third, no treatment. No significant difference was reported between the groups in muscle soreness at 0, 24 & 48h post exercise, muscle strength and limb girth.

In the same year, Tiidus showed that massage had an effect on inflammatory response and may be able to affect the development of muscle soreness. However, he proposed that for massage to be able to disrupt the initial stages of muscle damage and inflammatory response, it will have to be applied 1-2h following eccentric exercise. This supported the findings of Smith *et al.* (1994), who, when examining the effects of athletic massage on DOMS, serum CK concentration and neutrophil count on 14 subjects randomly assigned to either control or massage groups, found that a 30 min of effleurage and petrissage performed within 2h after exercise, resulted in prolonged elevation of blood neutrophil concentrations (suggesting that neutrophils had migrated from inflamed muscle), reduced serum CK concentrations, and an apparent reduction in DOMS.

Hemmings *et al.* (2000) were unable to add support to these findings in a CCT conducted on 8 amateur boxers who received a combination of petrissage and effleurage immediately following punching exercise with a large eccentric component vs. untreated controls performing the same exercise, but not receiving massage thereafter. Although massage reduced perceptions of recovery (p>0.01), the deficit in punching force and recovery of blood lactate concentrations, were not improved in the group receiving massage (p>0.05).

Farr *et al.* in 2002 also found that 30min massage sessions consisting of effleurage and petrissage performed by a qualified masseur attenuated muscle soreness and tenderness that resulted in the development of DOMS in eight subjects after downhill walking. These subjects received massage on one limb 2h post –exercise. Although soreness and tenderness associated with DOMS was improved in the massaged legs, measures of strength and one-legged vertical jump displacement however did not differ significantly between the treatment and control legs. This led to the conclusion that massage may attenuate DOMS, but had no effect on strength and muscle function.

Jönhagen *et al.* (2004) examined the effect of a combination of 4min effleurage & 8min petrissage 10min, 24h and 48h following 300 maximal bilateral eccentric contractions of the *quadriceps femoris*. No improvement in recovery in maximum strength measured on a leg dynamometer, functional tests on single-leg long jumps, pain (evaluated using VAS) post eccentric exercise was reported.

Frey Law *et al.* (2008) more recently studied the effect of deep tissue massage on post EIMD torque, mechanical hyperalgesia or pressure pain threshold (PPT) and perceived pain (DOMS). Forty participants were randomly assigned to either a 6min deep-tissue (effleurage and petrissage), cutaneous, light stroking of the skin (superficial touch) or control (quiet rest only) groups following an unaccustomed eccentric wrist extension. The treatment was performed 24-48h after eccentric exercise

and strength, pain, and sensory testing took place before and after treatment. Testers were blinded to group assignment for rest of the study. Although it was found that no significant group interactions were reported in terms of torque recovery in the massaged group, massage did reduce symptoms of myalgia by approximately 25-50%, and the reversal of DOMS symptoms was significantly greater in the treatment group and pain increased minimally with rest (p<0.0001). Deep tissue massage was able to decrease mechanical hyperalgesia and decreased stretch pain relative to the control group.

Dolgener and Morien (1993) determined the effect of 20min sport massage (effleurage and petrissage) on lactate disappearance following exhaustive exercise. Twenty-two male subjects were randomly selected into passive recovery (rest in supine position), bicycle recovery or massage groups. Subjects performed an exhaustive treadmill run before intervention. The subject were either massaged, rested or rode a bicycle for the total of 20min recovery time. Blood samples were taken and lactate concentration determined at rest, 3, 5,9,15 and 20min post exercise and led to the conclusion that massage did not remove lactate more than resting or as well as cycling at 40% of VO<sub>2</sub>max following exercise.

Due to the inconsistent, often conflicting, evidence that currently exists regarding the possible benefits of these two recovery modalities used in combination, the need for a robust systematic review in RCTs is confirmed. From a preliminary review of the literature focussing on studies that have examined the effects of the two massage modalities combined, it would also appear that petrissage is more effective when it is preceded by effleurage which would justify the exclusion of studies using only one of these techniques as therapeutic modality in a thorough systematic review of the beneficial effects of MM.

#### 2.5.4 Friction and tapotement techniques

Friction is defined as "an accurately delivered penetrating pressure applied through fingertips" (Galloway and Watt, 2004) that is administered either transversely to (cross-fibre) or parallel to (linear) the direction of tissues (Moraska, 2005). It is used to initiate a small, controlled inflammatory response to the affected area in order for breaking down scar tissue and separate adhered tissues. It has been found to influence cell behaviour in all soft tissues (Cyriax, 1984) and it was suggested by Van der Windt *et al.* (1999) that friction massage may stimulate the proliferation of fibroblasts and collagen fibre alignment. This had been confirmed by Davidson *et al.* (1997) who reported an improvement in gait in rodents with Achilles Tendinitis recording an improvement in both step length and frequency following friction massage. It was also confirmed by Fehrs in 2010 who reported break down of adhesive tissue that had developed and optimization of the quality of scar tissue.

Tapotement, another frequently used massage technique, is also known as percussion or hacking and involves repetitive light stroking movements that are applied with the ulnar portion of the hands or

cupped hands. It is usually performed prior to performance in sports to energize the muscle tissue and stimulate muscle spindles or Golgi tendon organs according the location of treatment (Kukulka *et al.*, 1986; Moraska, 2005). In addition, using the previously described local xenon <sup>133</sup>Xe wash out method to determine blood flow, Hovind and Nielsen (1974) reported an increase in skeletal blood flow comparable to exercise hyperemia observed in healthy uninjured adults who had not exercised and ascribed this increase to repetitive contractions resulting from the tapotement.

The majority of studies do, however, not use these techniques in isolation and incorporate them into Swedish or classic massage therapy sessions.

#### 2.5.5 Swedish/Classic massage trials

Classic western massage, also referred to as "Swedish" massage, is the most common form of massage that is used globally. It is a combination of a variety of techniques, frequently including effleurage, petrissage, friction, vibration, tapotement, percussion and stroking with the choice of combination differing between therapists depending on the therapist's experience and /or the intended clinical advantage desired (Weerapong *et al.*, 2005). In most of the studies a combination of western techniques has been used to investigate the effects of massage.

#### 2.5.5.1 Swedish Massage without preceding exercise

Although this thesis is primarily concerned with the use of different forms of therapeutic massage following EIMD, it is of interest to begin by examining the outcome of a few studies which have focussed on the effects of Swedish massage in the uninjured individual in whom EIMD was not induced.

In 1999 Leivadi *et al.* conducted a study comparing massage and relaxation therapy on 30 University dance students who were asked to continue their normal daily routine and school regimen, and were randomly assigned to two groups. The treatment group received 30min of whole body effleurage, petrissage and friction and the control group, 30 min of progressive muscle relaxation exercise twice a week for five consecutive weeks. The massage group presented with a significant increase in ROM of the shoulder joint and neck and although both groups reported lower levels of depression and anxiety, the stress hormone, cortisol, was evidently decreased in the saliva of the massage group.

Barlow *et al.* (2004) followed this up by investigating the immediate effect of MM on hamstring flexibility in a single blind study conducted on 11 physically active young men age (21+/- 3years). Hamstring muscles randomly received either 15min of effluerage, friction and petrissage performed by

a massage therapist bilaterally or 15min of supine rest. It was found that a single bout of hamstring massage did not have a significant effect upon "sit and reach" score, but a higher percentage of change in reach in those subjects who had pre-test reach score of less than 15cm. This study highlighted the need for further studies with a larger sample and to investigate the greater effect on flexibility in the individuals with lesser ROM.

In explaining the possible effect of MM on ROM, it is necessary to understand that fascial tissue is richly innervated with free nerve endings and mechanoreceptors and that Pacini corpulscles and Ruffini endings are nerve endings in the skin responsible for transporting impulses to central nervous system (CNS), particularly regarding pressure, pain and location (proprioception). While activation of motor nerves by motor neuron impulses relayed to muscle tissue cause contraction via the stretch reflex (Figure 2.7), Ruffini endings are activated by lateral stretch and Pacini corpuscles by slight pressure changes. This subsequently lowers the activity of the sympathetic nervous system and increases muscle relaxation (Van den Berg and Capri, 1999; Schleip, 2003), supporting the findings that deep tissue techniques have a relaxing effect on local tissues and whole organism (Schleip, 2003).

#### 2.5.5.2 Swedish massage after eccentric exercise

The majority of studies focussing on the effects of Swedish classical massage techniques used to attenuate the consequences of EIMD, investigated a variety of associated markers related to the functional deficits and levels of muscle soreness and pain experienced. In this initial review of the related literature, these will be presented chronologically with a focus on more recent work undertaken in the last 12 years.

In a RCT conducted by Hilbert *et al.* (2003), 18 healthy volunteers assigned to either a massage or control group performed six sets of eight MVC of the right hamstring, which were followed 2h later by 7min of effleurage, 1min of tapotement and 12min of petrissage versus 20min of applied placebo lotion and resting in the control group. Peak torque and mood, ROM, intensity and unpleasantness of soreness, and neutrophil counts were assessed at regular intervals during the 24h after exercise. There was no effect found on eccentric torque with massage but level of soreness was reduced 48h post exercise. The results showed that massage administered 2h after exercise did not improve muscle function, but there was improvement in intensity of soreness.

Zainuddin *et al.* (2005) examined the effects of Swedish massage on DOMS, swelling, and recovery of muscle function, in five healthy men and five healthy women with no history of upper limb injury and resistance training, using contralateral arms for treatment and control phases. Subjects received 10 min of Swedish massage on exercised arm 3h following maximal eccentric exercise of the elbow flexors.
MM had a significant effect (p<0.05) in reducing DOMS and also improving swelling in the upper arm muscles, but showed no effect on muscle function.

A randomized self-controlled comparative study on the effect of classic massage (effleurage that was performed distal to proximal, kneading that was performed in forward circular movements, picking up that was done from proximal to distal and shaking) and dynamic soft tissue mobilization (DSTM) massage techniques on hamstring muscle length (estimated from the height of straight leg raise) in 35 competitive female hockey players was completed by Hopper *et al.* in 2005. The passive straight leg raise and passive knee extension were used to measure indirect hamstring length, before, following and 24h after treatment. It was concluded that massage had a significant short term effect on hamstring length (p < 0.01), but was not maintained over a 24h period. Both DSTM and classic massage had a significant effect on hamstring length.

Mancinelli *et al.* (2006) used a randomized pre-test/post-test control group design, to determine whether post-exercise classic massage has an effect on DOMS and physical performance in women collegiate athletes. Twenty-two female basketball and volleyball players received either thigh massage (17 min effleurage, petrissage & manual vibration treatment group, n=11) or no treatment n=11) on a day of predicted peak soreness following 4-day training routine. There were improvements in vertical jump displacement, perceived soreness and algometer readings for the massage group, but not muscle length. This study promotes the use of Swedish MM in collegiate athletes for the control of muscle soreness, pain pressure and improved muscle power.

In a contralateral RCT, Willems *et al.* (2009) examined the effect of MM (25min of effleurage, petrissage, tapotement and effleurage), on DOMS in the *quadriceps* muscles and single leg vertical jump performance after a 20min downhill walk carrying a load equal to 10% of their body mass in seven active females. DOMS was reduced by MM at 48hr post-exercise in the *rectus femoris* and *vastus lateralis* (p<0.05), but not in the *vastus medialis*. The decline in jumping performance with each leg after the downhill walking was decreased (p<0.001) by 19% and 21% in control and massaged legs, respectively (p<0.05). Reductions in DOMS in the massaged leg after downhill walking were muscle-specific.

Abad (2010) studied the effects of classical massage on DOMS perception, ROM, limb girth and maximum strength performance after muscle damage following strenuous eccentric exercise. Eighteen males were divided into the following three groups: massage-only, exercise-only (control group), and exercise + massage (experimental group) according to their 1-RM value. Muscle damage was induced by 30 supramaximal eccentric contractions (6 sets of 5 repetitions at 110% 1RM). Six minutes of classical massage was applied on the exercise and massage group immediately post exercise and DOMS, ROM and limb girth were assessed 24, 48, 72 and 96h after exercise and maximum strength

after 48 and 96h. No difference was detected in experimental and control groups in all parameters assessed. The authors concluded that classic massage did not minimise symptoms associated with DOMS.

Pilladi *et al.* (2013) evaluated the effect of massage compared to active exercise on DOMS. Thirty subjects were divided into groups of either 20 min of effleurage, petrissage, friction, tapotement, stroking on quadriceps muscles (massage group) or 5-6 sets of active exercise separated by 2-3min of rest (control group). Measurements of pain using VAS and knee joint functional capacity using a functional knee rating scale showed that massage had a positive effect in ameliorating DOMS.

Han *et al.* (2014) examined the effect of massage on the muscle pain of the gastrocnemius muscle and gait. In this randomized trial the control subjects received 20 min placebo (TENS pads put on the leg) and treatment group 20 min of light stroking, skin rolling, friction and milking. The induced muscle damage response was induced in the subjects by walking up and down the stairs in a five storey building. The massage reduced the pain significantly compared to the control group and change in gait was seen in terms of distance, step length, stride length and temporal parameters (ambulation, heel on off time, stride velocity). The pain relief correlated with gait, suggesting a greater effect of massage after DOMS.

Shin and Sung (2015) focussed on the effects of massage on muscular strength and proprioception after EIMD. Subjects were randomly divided into a placebo group (n=10) which was fitted with TENS pads for 15 min following EIMD exercise (20 x up & down stairs) or an experimental group (n=11) in which the stair climbing EIMD exercise was followed by 15 min of effleurage, milking, friction and skin rolling in the gastrocnemius muscle. Blood lactate concentrations were assessed using a Lactate Pro analyser before and after exercise and proprioception by dual inclinometer. The findings revealed that massage to the *gastrocnemius* muscle improved muscle strength and proprioception, stimulating the superficial layer of this muscle. A tension equal to one gram and a stretch of a 1 micrometer (in length) was found to be enough to activate the muscle spindle. Ward (2012) attributes their findings to the psychological aspects of touch therapy. He argues that the idea of placing hands on the individual could trigger psychological relaxation or the perception that the treatment is doing something favourable. Moreover, he highlights the duration of the treatment as playing a role in achieving positive results when there is excessive pain or excessive EIMD.

From this descriptive review on the effects of Swedish massage, it would appear that the evidence is quite conclusive that when a combination of 3 or more Western classical techniques are used, the effect of MM is positive.

As the majority of the research in this field has however examined a variety of different outcome measures and the number of studies are small and in some cases not randomised, a systematic review in which outcome measures are subdivided and classified into homogenous groups, is needed to provide greater clarity regarding the efficacy of MM in attenuating the functional and physiologic deficits associated with EIMD.

#### 2.5.6 Manual (classic) massage combined with other therapies

The combination of warm-up, stretching and massage decreased the negative effects of eccentric exercise in the study by Rodenburg *et al.* (1994). 50 people, were randomly assigned to a treatment and a control group, performed eccentric exercise with the forearm flexors for 30 min and treatment group additionally performed a warm-up and underwent a stretching protocol before the eccentric exercise and massage afterwards. Functional and biochemical measures were obtained before, and 1, 24, 48, 72 and 96h after exercise. Outcome measures were CK, maximal force and the flexion angle of the elbow. The results were however, inconclusive, the objective measures did not yield more unequivocal results than the subjective DOMS scores.

Andersen *et al.* (2013) examined the acute effects of massage or active exercise in relieving muscle soreness in 20 healthy female volunteers in a RCT. The subjects performed eccentric contractions for the upper trapezius muscle on a Biodex dynamometer to induce DOMS. DOMS presented 48h later, at which stage subjects either (a) received 10 min of massage of the trapezius muscle or (b) performed 10min of active exercise (shoulder shrugs  $10 \times 10$  reps) with increasing elastic resistance (Thera-Band). Initially, one treatment was randomly applied to one shoulder while the contralateral shoulder served as a passive control. Two hours later, the contralateral resting shoulder received the other treatment. The subjects rated the intensity of soreness on a 0–10scale, and a blinded examiner took measures of pressure pain threshold (PPT) of the upper trapezius immediately before treatment, 0, 10, 20 and 60min after the treatment.

Massage compared with control, significantly reduced the intensity of soreness and increased pressure pain threshold (PPT) (i.e. reduced pain sensitivity) when compared to the alternative treament. For both types of treatment, the greatest effect on perceived soreness occurred immediately after treatment, whereas the effect on PPT peaked 20min after treatment. However, active exercise using elastic resistance provided similar acute relief of muscle soreness as did massage (Andersen *et al.*, 2013). These researchers concluded that although massage can be used to reduce DOMS acutely, for example, to prepare for competition or strenuous work, the effect was found to be should be temporary with the greatest effects occurring during the first 20min after treatment and diminishing within an hour.

#### 2.5.7 Combination trials focussing on changes in blood flow

In the study by Cambron *et al.* (2006) six different massage techniques were used including Swedish (effleurage and petrissage), deep tissue, myofascial release, trigger point, sports and craniosacral massage. Massage ranged between 30-90min in duration. The subjects that received Swedish massage had greater reduction in blood pressure (BP) than subjects who had other forms of massage. The length of the massage and techniques performed were however, not controlled and the length of the technique was based on the student's perception of the subject's need.

Numerous studies have also evaluated the effect of massage on blood flow using venous occlusion plethysmographs and showed significant increase in average blood flow (Callaghan, 1993). Examining the hypothesis that an increase in blood flow may lead to elevation of the amount of the oxygen delivered to the injured tissue, hence improving healing or return to homeostasis process (Hunt, 1990), Callaghan (1993) used venous occlusion plethysmography and Doppler ultrasound to determine arterial and venous blood flow during effleurage massage on the *quadriceps* muscles (Tiidus and Shoemaker, 1995). They found no effect of effleurage quadriceps arterial or venous flow in both the light and deep effleurage techniques that were performed for 10min (Tiidus, 1997). Moreover, Shoemaker *et al.* (1997) also found no positive results on the effect of effleurage, and petrissage massage on blood flow of the quadriceps muscles, but light muscle contractions showed increase in blood flow post eccentric exercise compare to MM.

Although the validity of the evidence must be questioned as venous occlusion plethysmography was used for measurement of blood flow during massage (Tiidus and Shoemaker, 1995), and Doppler ultrasound was used to determine arterial and venous blood flow during effluerage massage on the quadriceps muscles. Shoemaker *et al.* (1996) found no positive effect of effluerage, petrissage or tapotement massage on blood flow of the quadriceps muscles, but light muscle contractions showed increase in blood flow and then concluded that the light contraction may actually have more effect in enhancing blood flow post eccentric exercise compare to MM.

Since blood flow and the rate of lactate redistribution are likely to be near maximal following highintensity exercise, it seems unlikely that massage could improve these factors during recovery. In fact, recent research has suggested that massage recovery is no more effective than rest recovery in promoting blood lactate removal (Micklewright *et al.*, 2006), while recently it has been reported that a combined active and massage recovery intervention is more effective than rest, and as effective as continuous active recovery for the elimination of blood lactate following a simulated 5km cycling time trial (Monedero and Donne, 1993). However, interpretation of the contribution of massage to the recovery process in this study is difficult because the combined recovery interventions included an initial active phase. In the combined active-massage-active group, the effect of the massage phase on blood lactate reduction is consequently obscured by the initial active phase. The effectiveness of massage and active recovery in terms of the elimination of lactate from the blood following an exhaustive bout of high-intensity exercise and the hypothesis that blood lactate removal would be accelerated by active recovery and combined massage-active recovery, compared to rest, massage, or combined rest-massage recovery, was therefore subsequently re-examined by numerous researchers.

Gupta *et al.* (1996) also investigated the comparison of blood lactate removal during the period of recovery in which the subjects were required to sit down as a passive rest period, followed by active recovery at 30% VO<sub>2</sub> max and short term body massage (10 min of kneading and stroking) as the three modes of recovery used. After exhaustive intermittent exercise was performed on a bicycle ergometer blood lactate concentration was recorded at 0, 3, 5, 10, 20, 30 & 40min post-exercise and no remarkable difference between massage and a passive type of sitting recovery period was reported. It is concluded from the study that the short term body massage is ineffective in enhancing the lactate removal and that an active type of recovery is the best modality for enhancing lactate removal after exercise.

These findings were confirmed by Monedero and Donne (2000) who conducted a study on 18 healthy trained male cyclists' subjects examining the effect of recovery interventions on lactate removal and subsequent performance. Subjects were allocated to either control passive recovery where the subject seated at rest on the chair, active cycling 50% VO<sub>2</sub>max, effleurage, combined stroking and tapotement massage and cycling all done 15min post eccentric exercise. There was no significant difference between passive rest and massage group, but there was significant improvement with other interventions compared to massage and passive rest. Combined recovery was found to be better than passive (p<0.01) and either active or massage (p<0.05) in maintenance of performance time. Removal of blood lactate during combined recovery was significantly better than passive at 3min and significantly better than passive, active, and massage at 15 min. Therefore, the combination of these modalities showed more positive effect for maintaining maximal performance with active recovery.

Robertson *et al* (2003) examined the effects of leg massage compared with passive recovery on lactate clearance, muscular power output, and fatigue characteristics after repeated high intensity cycling exercise. Nine male games players' subjects attended the laboratory on two occasions one week apart and at the same time of day. After five min of active recovery, the intervention was either 20 min of passive supine rest or 20 min of leg massage (effleurage and non-specified techniques), assigned in a randomised cross over fashion an either 20 min of leg massage or supine passive rest following high intensity exercise subjects performed a second standardised warm up and a 30sec Wingate test. Capillary blood samples were drawn at intervals, and heart rate, peak power, mean power, and fatigue index were recorded. Massage was applied five min to the back of the left leg, five min to the back of

the right leg with the subject in a prone position on a standard treatment couch. The subject then assumed a supine position, and massage was applied to the front of the right then left leg (each for five min). The results of the study showed no measurable physiological effects of leg massage compared with passive recovery were observed on recovery from high intensity exercise, but the subsequent effect on fatigue requires further investigation because a significantly lower fatigue index was observed in the massage trial (p = 0.04) post exercise.

#### 2.5.8 Unspecified massage therapy

In the recent publication of Crane *et al.* (2012), the type of MM used was unfortunately not specified. But interestingly, compared to unmassaged muscle cells, the tissue from massaged legs had different levels of the two key proteins, NF $\kappa$ B and PGC-1 $\alpha$ . The authors regarded this as a positive finding that promotes the healing of muscle cells as lowering NF $\kappa$ B levels has been shown to reduce inflammation and increasing PGC-1 $\alpha$  levels to lead to the creation of more mitochondria that generate energy for cell growth and muscle repair. Massage was also found to have attenuated the production of TNF $\alpha$  and IL-6 inflammatory cytokines.

Limitations observed in this study however include the changes in blood circulation to both legs and the difficulty in isolating each leg in one subject. The additional muscle injury to the area resulting from the muscle biopsies could also have influenced the outcome of the study.

Bakowski *et al.* (2008) found that massage decreased muscle soreness by 10-20% with no significant change noted in ROM of the massaged arm when the arms of the 14 healthy subjects were randomly assigned to either massage or control. The subjects performed eight sets of concentric and eccentric actions of the elbow flexors with each arm. One arm was massaged for 10 min 30 min after exercise and contralateral arm rested. The measurements were taken pre-exercise, post-exercise, 10min, 6, 12, 24, 36 48, 72 and 96 h. Although there was a 10%-20% decrease in soreness in the massaged arm, it was not significant. No difference in ROM and upper arm circumference noted within the groups.

#### 2.5.9 Massage therapy following exercise-induced muscle damage in rabbits

In an extensive study performed on rabbits, Hu *et al.* (2007) looked at the effect of Tuina (kneading) therapy on muscle growth following muscle damage induced by heavy beats with a 2kg hammer on the middle belly of *gastrocnemius* muscle 9cm away from calcaneus muscle in an attempt to simulate the damaging effects of eccentric exercise on muscle, 114 six-month old male rabbits were divided into four groups (I) observational group (n=36) which were exposed to eccentric exercise, but received no

treatment, (ii) control group (n=36) which received massage treatment 24h after the injury, (iii) Tuina group (n=36) which received massage treatment two hours after the injury and normal group (n=6) which did not undergo any intervention and no activity. The force and the frequency of the technique were measured with dynamic testing instruments. Sixteenth day after the modelling, six rabbits in each subgroup were sacrificed and in a normal group, six rabbits were sacrificed on the second day after the other groups.

The Tuina group had greater number of satellite cells (analysed using light microscope, where a semiquantitative count of PCNA staining positive of satellite cells were counted). The cells that had PCNA positive satellite were changing to brown colour) compared to the remaining groups. The control group had more satellite cells than observation group but less than the Tuina group. There was no change with normal group. The authors of this study concluded that Tuina therapy improves muscle recovery by increasing the number of satellite cells promoting an increase in regenerative skeletal muscle cells and more rapid recovery of the function of the muscle, delay DOMS and reducing muscle soreness (Hu *et al.*, 2007; Marong and Jin, 2009) and attributed this to a reduction in muscle spasm, more oxygen and nutrient provision that increased metabolic activity which were also observed. However, the effect of Tuina therapy on contractile proteins following eccentric muscle damage was not observed.

In a rodent study, Marong and Jin (2009) found that Tuina applied pre-exercise was able to reduce the level of muscle damage cause by eccentric exercise in DOMS, moreover, Tuina was more effective in delaying DOMS and controlling the damage of lipid peroxidation.

Muscle regeneration initiated shortly after injury can be limited by fibrosis which affects the degree of recovery and predisposes the muscle to reinjury (Best *et al.*, 2008). Massage has been identified as one of the useful strategies to enhance skeletal muscle repair through increased vascularisation also including gene therapy, exercise and neuromuscular electrical stimulation.

In another study by Butterfield *et al.* (2008) on New Zealand white rabbits performed a bout of eccentric contraction resulting in muscle damage. One leg was then subjected to cyclic compressive loads and the contralateral was put as a control. The rabbits had a peroneal nerve cuff inserted surgically and subcutaneous interfaces for controlling *tibialis anterior /exterior hallucis longus* muscles in both legs showed facilitation of recovery, fibre necrosis attenuation and reduction of leukocytes infiltration. MM was found to have improved recovery of function and the wet weight of the tissue was also reduced by the compressive loading. It also attenuated the damaging effects of inflammation in the rabbit model. However, Butterfield suggests further investigation on the translational efficacy of these findings.

According to the author's knowledge no work has to date been done on effect of massage on satellite cells in humans. These preliminary findings on animal models are positive, but replication in humans is a valuable directions for future research.

#### 2.5.10 Conclusion

Due to the heterogeneity of the study designs and the diversity in the outcomes measured, it is difficult to reach an objective conclusion regarding the efficacy of MM in positively affecting the physiological recovery post EIMD. While greater homogeneity regarding intervention strategies and design should be considered for the future and recent work performed in animal studies should be extended to humans, a formal systematic review and where possible, meta-analyses are required to reach a conclusion regarding the state of the knowledge in the field.

However, the increase in the use of VT both in sport poses the question as to whether this modality results in the same or greater benefits than MM or whether is it because MM is time consuming and mostly requires a qualified therapist to perform, that the popularity of VT is increasing.

#### 2.6. Vibration Therapy (VT)

#### 2.6.1 Introduction

In 1895, Dr John Harvey Kellog invented a machine that creates mechanical vibration. It was later used to improve circulation when applied to parts of the human body and referred to as VT. Today this type of modality is mostly used in the athletic settings (Cafarelli *et al.*, 1990).

Vegar and Imtiyaz (2012) describes VT as a periodic alteration of force, acceleration and displacement over time in form of mechanical oscillation. The energy is transferred from an actuator (vibration device) to a resonator (the human body or body part). First use of vibration in sport was in 1987 by Nazarov *et al.* using the principle that if the vibration stimulation is placed on the distal muscle, the effect will be transmitted to the proximal muscle and improve athlete's performance.

There are two types of VT, whole body vibratory therapy (WBVT) and localized VT (LVT). Both work on the principles of amplitude, frequency, and magnitude of the oscillations which determine intensity. The extent of the oscillatory motion determines the amplitude (peak to peak displacement, in mm) of the vibration, the repetition rate of the cycles of oscillation denotes the frequency of the vibration (measured in Hz), and the acceleration indicates the magnitude of the vibration (Cardinale and Bosco, 2003). Low-amplitude (range: 3-10 mm), low-frequency (range: 15 - 44 Hz) mechanical stimulation of human body (has been hypothesised to be a safe and effective way to improve muscle strength (Cardinale and Bosco, 2003), while the best pain reducing frequencies range between 50 and 200 Hz (Lundenberg *et al.*, 1984).

#### 2.6.2 Whole Body Vibration Therapy (WBVT)

WBVT is performed with the subject standing on a vibration machine/plate and results in transference of vibratory stimuli to the whole body. Chronic treatments of WBVT have been shown to produce improvement in neuromuscular properties of human skeletal muscle and frequent contractions from the muscles which then improves muscle soreness (Aminian-Far *et al.*, 2011).

Early reported benefits of VT include an improvement in circulation, blood flow to the skin, an increase in skin temperature and decreased accumulation of lactate in the blood (Friden *et al.*, 1988). More recent reports include mention of improvements in bone density, muscle mass, reduced joint pain and stress and activation of metabolism (Summer and Pletcher, 2014). Furthermore, a training program that includes vibration has been shown to enhance the strength, power and length of muscle (Issurin and Tenenbaum, 1999; Delecluse *et al.*, 2003). It has been found to cause stimulation of muscle spindles, increasing their afferent activities (Ayles *et al.*, 2011) and reducing background tension if applied before exercise (Bosco *et al.*, 1999; Veqar and Imtiyaz, 2012). These studies have resulted in the frequent current use of VT as an exercise intervention. This application of VT is however not the focus of this study.

As is summarised in Table 2.1, WBVT has been found to enhance power output, PPT, and active ROM during recovery from DOMS. Bosco *et al.* (1999a) first reported the effect of regular WBVT (26 Hz) applied over a period of 10 days in 14 subjects assigned to experimental and control groups resulting in enhanced mechanical power and jumping height in the subjects receiving regular daily WBVT sessions. The acute effects of WBVT (of non-specified frequency) were examined on the lower limbs of six female volleyball players randomly assigned to control (n=3) and experimental (n=3) groups. Those receiving WBVT following a maximal dynamic leg press, significantly improved performance to a "level that could have otherwise taken several weeks to obtain". The adaptive responses of human skeletal muscle to vibration exposure were examined in six female volley ball players and significant enhancement with the treatment was reported in both average velocity and power (p<0.05; Bosco *et al.*, 1999b). Studying this reaction on the upper limbs of 12 national level boxers randomly assigned to a control or experimental group, Bosco *et al.* (1999c) subsequently reported that vibration @ 30Hz (1 min x 10repetitions) was applied to the experimental group post eccentric exercise, increased the average mechanical power force.

After Edge *et al.* (2008) published work reporting no benefit of WBVT at a low frequency (12 Hz) on running performance recovery following a high-intensity interval training (HIIT) session in terms of serum CK,  $VO_2$  and blood lactate. In 2010, Sui *et al.* examined the immediate effects of 2 vibration frequencies that yielded the same maximum acceleration on peak torque and stiffness of knee extensor

	Outcomes Measured	Results	Comments
WBVT			
Bosco <i>et al.</i> (1999a)	Power output- vertical jump	Positive	26Hz
Bosco <i>et al</i> . (1999b)	Power output- maximal dynamic leg press	Positive	Frequency not specified
Edge et al. (2008)	Serum CK, CRP concentration	No difference	Following HIIT
	VO <sub>2</sub>	No difference	
	Blood lactate concentration	No difference	
Sui et al. (2010)	Tissue stiffness	No difference	26Hz or 40Hz post
	Knee extensor and flexor peak torque @ 26Hz vs 40Hz vs control	Positive in both 26 and 40Hz	eccentric exercise
Aminian-Far et al.	DOMS	Positive	
(2011)	СК	Positive	
	MVC	Positive	
Marshall and Wyon (2012)	Vertical jump	Positive	No immediate effect post exercise. Long-term
	Active ROM	Positive	positive effect noted
	Leg anthropometry (thigh and calf circumference)	Positive	
Wheeler and Jacobson	Flexibility	No difference	Compared to light
(2013)	DOMS (VAS)	No difference	exercise
	Explosive power	No difference	

**2.1** Outline of recent studies conducted on the efficacy of Whole Body Vibration Therapy (WBVT) on measures of physical performance and DOMS on inflammatory markers

WBVT: whole body vibration therapy; ROM: range of motion; CK: creatine kinase; CRP: C-reactive protein; DOMS: delayed onset muscle soreness; HIIT: high intensity interval training; MVC: maximum voluntary contractions; VAS: visual analog scale; vs: versus

and flexor muscles. Ten recreationally active subjects performed 10 x 60sec static half squats intermitted with a 60sec rest period between bouts on a platform with no vibration (control) and two vibration frequencies (experimental groups). CON peak knee extensor and flexor torques examined within 5min before and after vibration frequencies of 26 and 40Hz were found to be significantly greater than that in a control group (p<0.05). Greater positive change of ECC peak torque of knee flexor after vibration was only seen in 26Hz frequency group, compared to control group. There were no significant differences obtained in tissue stiffness in the quadriceps and hamstring muscles using Young's modulus as index of tissue stiffness (Sui *et al.*, 2010).

In a study by Aminian-Far *et al.* (2011) on WBVT and prevention and treatment of DOMS, in which 22 females and 10 males untrained university students were randomly placed into either a WBV training (n=15) or non-WBV-training (n=17) group, both groups performed six sets of 10 maximal voluntary eccentric contractions to induce DOMS. The WBVT-training group received vibration loading for 1min at 35Hz in a squatting position on the vibratory platform (100degrees of knee flexion) prior to the

eccentric exercise. The greatest decrease in muscular strength occurred in the in the control group and PPT was found to be more decreased in WBV-training group than in the control group. Plasma CK levels were greater in the control with muscle soreness being greater in the control group.

Barnes *et al.* (2011) examined the acute effect of WBVT on performance using the commercial machine (Galileo Sport, Novotec, and Pforzheim, Germany) which has motorized teetering platform that produces side to side alternating sinusoidal vertical vibration (SAV) to the body. Equal numbers of subjects (n=8) were randomly assigned to either WBVT or control treatment groups. Subjects completed a bout of eccentric exercise after they underwent either VT (26Hz) or control (no treatment) immediately post exercise, 12h and 24h post exercise. Significant decreases in isometric (ISO) and concentric (CON) strength measures occurred in both groups. VT did not attenuate muscle force loss and improve the recovery of muscular performance after strenuous bout of eccentric exercise.

Marshall and Wyon (2012) reported that a beneficial supplemental training intervention increased jump and active flexibility in highly trained dancers. These researchers investigated the effect of 4 weeks WBVT on jump height, active ROM and calf and thigh circumferences in 17 dance students randomly assigned to an intervention group (WBVT) for 30sec at 35Hz frequency, 8mm displacement for 2 weeks and then 40sec at 40Hz for the last two weeks) or control group (which performed the same type of exercises without vibration stimuli). Significant (p<0.05) improvements in the experimental group were noted over time for vertical jump, active ROM for both legs (p<0.01) and anthropometric data, when compared to the control group.

Wheeler and Jacobson (2013) determined the effects of WBVT on DOMS (using VAS), measures of perceived pain/soreness, flexibility and explosive power after EIMD in 20 healthy subjects randomly assigned to experimental (WBV) or the control group (Light exercise), immediately following eccentric exercise and again immediately after receiving treatment. No significance (p > 0.05) was found within or between groups when comparing pre-assessments and post-assessments of DOMS, flexibility, or explosive power. It was concluded that WBV is as effective as light exercise in reducing the severity of DOMS and may be used as a recovery option in addition to current treatments. Their findings were however not supported by those of Xanthos *et al.* (2013) who studied the effect of WBV and traditional intervention as a recovery technique on running kinematics and jumping performance following EIMD.

Although the WBVT has been studied by many researchers, this descriptive overview confirms the conclusion reached by Animian –Far (2011) that the efficacy of WBVT is not yet conclusively proven due to contradictory results. As summarized in Table 2.1 of the 7 studies conducted since 1999, only 4 did not show a positive on attenuation of muscle strength losses, one showed a failure to improve markers of inflammation.

This could be due to different mechanisms of action. One mechanism that has been described is elicited neuromuscular activation (Bosco *et al.*, 1999, Cardinale and Bosco, 2003), whereby the local tendon and muscle vibrations stimulate muscle spindle and I $\alpha$  fibres (Figure 2.1), which mediate the monosynaptic and polysynaptic pathways (Hagbarth and Eklung, 1985; Siedel, 1988).

Although the evidence is therefore primarily positive, the sample sizes are relatively small and a larger scale randomized controlled study needs to be conducted to confirm these results before the evidence is overwhelmingly positive. Furthermore the timing of the WBVT sessions is often prior to exercise and a clear differentiation between the efficacy prior to, during the course of and following exercise needs to be examined.

## 2.6.3 Local Vibration Therapy (LVT)

During LVT used in physiotherapy settings, the therapist holds or uses a hand-held vibratory device to the specific part of the body treated such as calf, thigh or shoulder. The effects of VT include motor pool activation. Comprising a group of motor spinal neurons that innervate same muscle, motor pools with multiple neurons produce finer movements. The frequency of vibratory stimulation as well as the initial length of the stimulated muscle has been shown to be highly correlated with motor neuron recruitment (Ayles *et al.*, 2011).

One of the most frequently used innovative LVT tools that is currently on the market, are the percussive vibratory massagers. These frequently have nodes that release vibration at different intensities and their heads ensure that the users experience a deep massage when placed on a particular body segment experiencing pain. Manufacturers claim that they result in reduction of muscle soreness and enhance healing to the muscle as the powerful vibration released from hand held devices, relax the muscle (Percussive Massager, 2015).

Balke *et al.* (1989) compared the effects of manual and mechanical massage on recovery from muscular and physiological fatigue on 16 subjects assigned to either MM or LVT (using a thumper machine/sport percussive massager) groups. They concluded that there were physiological and local recuperative benefits from both modalities when compared to rest. As the sample size of this study was small and it is not clear whether the manual techniques used were as vigorous as the VT using a mechanical machine, the comparative findings of this early study with regard to the relative benefits of MM and VT were questioned (Callaghan, 1993).

A summary of the recent studies investigating the physiological effects of LVT on recovery from EIMD is given in Table 2.2

	Outcomes Measured	Results	Comments
Cafarelli et al. (1990)	Static contractions of	No effect	Percussive vibration
	quadriceps-rate of fatigue		massage
Bosco <i>et al.</i> (1999c)	Power output- boxing	Positive	30Hz
Issurin and	Explosive strength	Positive	44Hz
Tenenbaum (1999)			
Sinahara et al. (2005)	Inhibition of the H-reflex	Positive	75Hz, 30 min
Bakthiary et al. (2007)	IMVC	Positive	50Hz
	DOMS	Positive	
	Serum CK concentrations	Positive	
Kinser <i>et al.</i> (2008)	Flexibility	positive	30Hz combined with stretching
	Flexibility	Negative	30Hz
	Explosive strength	Negative	All groups
Sands et al. (2008)	Split ROM	Positive	Using stretching & 30Hz,
	PPT in BF	No difference	54 x 77x 32 cm vibrator
	PPT in VL	No difference	placed below heel &
			rearward thigh
Herda et al. (2009)	Passive ROM	Negative	No difference between
	Torque	Positive	passive stretch LVT
	Surface EMG	No difference	control
	Mechanographic amplitude	No difference	_
	of gastrocnemius soleus		
Broadbent et al.(2010)	DOMS	No effect of LVT	Randomised controlled
	IL-6		trial post
	Lymphocytes		
	Neutrophils		
	Histamine		
Ayles et al. (2011)	Mechanosensitivity	Positive	No difference
	PPT	Positive	
	DOMS	Positive after 24 h	
Barnes (2011)	Isometric concentric force	No change	26Hz
Lapole and Perot	Passive stiffness	Positive	
(2011)	Musculo-tendinous	Positive	
	stiffness		
	Tendinous reflex	Positive	
	H-reflex	Positive	7
Lau and Nosaka	ROM	Positive	65Hz post eccentric
(2011)	DOMS	Positive	exercise
	Swelling	No effect	

**Table 2.2** Outline of recent studies conducted on the efficacy of Local Vibration Therapy (LVT) on

 measures of physical performance and DOMS on inflammatory markers

	Muscle strength recovery	No effect	
	Serum CK	No effect	
Mohammadi and	ROM	Positive	50Hz prior to eccentric
Sahebazamani (2012)	Muscle soreness 24-96h	Positive	exercise
	post exercise		
Xanthos et al., 2013	DOMS	No difference	26Hz

ROM: range of motion; h: hours; CK: creatine kinase; DOMS: delayed onset muscle soreness; LVT: localised vibration therapy; BF: biceps femoris; VL: vastus lateralis; IMVC: isometric maximum voluntary contractions; H-reflex: Hoffman reflex; IL: interleukin; EMG: electromyography; PPT: pain pressure threshold

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Cafarelli *et al.* (1990) studied the effect of percussive vibratory massage on recovery from repeated submaximal contractions. Twelve male subjects were assigned to either 5 min of rest (control) or 5 min of percussive vibratory massage after performing repeated, static contractions of the *quadriceps* at 70% MVC. The rate of fatigue was calculated from a regression line fit to the decline of the periodic MVC. No effect on the rate of fatigue in control and vibrated conditions found, it was concluded that short-term recovery from intense muscular activity is not augmented by percussive vibratory massage.

Shinohara *et al.* (2005) examined the influence of prolonged vibration of a hand muscle with the purpose of studying amplitude of the stretch reflex, motor unit discharge area and force fluctuations during steady, submaximal contractions. The experiment was conducted the non-dominant hand of 32 adults (15 men and 17 women) without neuromuscular disorders. The experimental group (n=20) received vibration (75Hz for 30 min) on the first dorsal interosseus muscle and control group (n=12) received no VT. All subjects performed the tasks with the index finger of the non-dominant hand before the experiment. LVT increased the amplitude of the latency component of the stretch reflex, the discharge rate of motor units and force fluctuation. The authors concluded that the differential changes in the amplitudes of the H- and stretch reflexes are indicative of a change in the sensitivity of the muscle spindle before and after an intervention and the suppression of the short term latency reflex (or response time) in the first dorsal interosseus muscle is probably the result of inhibitory input from higher centers.

Moreover, Bakhtiary *et al.* (2007) examined the effect of LVT (50Hz) on left and right *quadriceps*, hamstrings and calf muscles for 1min applied before downhill walking on 10° declined treadmill at 4km/h) on 50 healthy non-athletic subjects, randomly assigned to VT or non-VT groups (no intervention). The isometric maximum voluntary contraction force (IMVC) of left and right quadriceps muscles and PPT was measured 5, 10 and 15cm above the patella and mid-line of the calf muscle of both lower limbs before and after the treadmill walking. There was an attenuation of the post exercise deficit in IMVC and PPT and decrease in DOMs and CK concentrations in the VT group.

Kinser *et al.*, in the same year, examined the effects of simultaneous vibration-stretching on flexibility and explosive strength in 22 subjects allocated to vibration-stretching (VS; n=7), vibration alone (VF, n=8) and stretch only (n=7) groups. Vibration (30Hz, 2mm for 10s) was localised to four body sites.

Right and left forward-split flexibility was measured using the distance between the ground and anterior suprailiac spine, while explosive strength variables included flight time, jump height, peak force, instantaneous forces, and rates of force development. A combination of vibration and stretching treatment was found to have a large positive impact on flexibility, while not altering explosive strength.

Sands *et al.* (2008) assessing the use of LVT (30Hz, 22mm displacement) and stretching to enhance acute ROM and its influence on PPT in 10 gymnasts with one side split randomly assigned to the experimental condition, and the other side split was assigned as the control used a 54 x 77x 32cm come vibration device placed below the rearward thigh and heels was used to perform both side splits, Gymnasts were also assessed for PPT using an algometer on the *biceps femoris* (stretched) and *vastus lateralis* (not-stretched) bilaterally. Vibration improved split ROM more than stretching alone, but did not show a difference in PPT in either the stretched or non-stretched muscles.

Herda *et al.* (2009) examined the acute effects of passive stretching vs. prolonged vibration on voluntary peak torque, percent voluntary activation, peak twitch torque, passive ROM, musculo-tendinous stiffness, and surface electromyographic (EMG) and mechanomyographic amplitude of the *medial gastrocnemius* and *soleus* muscles during IMVC of the plantar flexors. 15 subjects were assigned to passive stretch, prolonged vibration or control groups and performed the IMVC. Passive ROM assessments before and after 20 min of the interventions. Passive ROM increased by 19% and musculotendinous stiffness decreased by 38% after the passive stretch, but neither changed after the vibration or control conditions. Both passive stretch and vibration elicited similar neural deficits (i.e., gamma loop impairment) that may have been responsible for the strength loss.

In 2010 Broadbent *et al.* assessed the effect of LVT on DOMS and associated inflammatory markers after downhill running in 29 male recreational runners who performed a downhill run for 40 min. They were randomly allocated to a LVT group or control group and underwent once-daily sessions of LVT on the upper and lower legs. Pre-run and for five days after the run, DOMS (using VAS) and selected plasma inflammatory markers were assessed in both groups. LVT (50Hz) significantly reduced calf pain 96h post-run, gluteal pain 96h and 120h post-run. Blood IL-6 concentration 24h and 120h post-run was also observed. It was concluded that an application of 50Hz vibration significantly reduces IL-6 and lymphocytes. A substantial decrease in histamine 24h and 120h post-run was also noted, but there were no clear substantial effects of LVT on neutrophils and lymphocyte subsets and histamine.

Ayles *et al.* (2011) examined the effect of LVT on 16 healthy males performed eccentric exercise inducing DOMS in the *tibialis anterior* muscle on one randomly selected leg and the contralateral leg served as a control. DOMS peaked at 24 hrs post EIMD On day 1 following exercise, segmentally related site PPTs reduced significantly when LVT was applied concurrently to the DOMS affected

*tibialis anterior* muscle (p<0 .04)compared to baseline mechanosensitivity or extra segmental control vibration.

In 2011, Lapole and Pérot investigated the effects of tendon vibration on the *triceps surae* stiffness of healthy subjects. The vibration program consisted in 14 days of 1h daily Achilles tendon vibration applied at rest. After the program, musculo-tendinous stiffness was significantly decreased (p=.01) and maximal passive stiffness was reduced (p <0.001). The tendon reflex also significantly decreased. The results showed that VT had positive effect in reducing the stiffness and could be of benefit to the athletes.

Lau and Nosaka (2011) conducted a randomized crossover design study on 15 male subjects who performed 10 sets of 6 maximal contractions of the elbow flexors 4 weeks apart. After exercise the one arm received 30 min of VT at 30 min after exercise and 1, 2, 3 and 4 days post-exercise. The other arm did not receive any intervention (control). The dominance and non-dominance of the arms were counter balanced as well as the order of treatment and control conditions. Results indicated that LVT was effective in reducing DOMS at 2 to 5 days after exercise and recovery of ROM, but did not have any effect on swelling, recovery of muscle strength and serum CK activity.

In recent studies by Mohammadi and Sahebazamani (2012) on the influence of LVT (50 Hz) on selected of the functional markers of DOMS. The study was done on 30 college males who were randomly selected to vibration group or control group. LVT was applied prior to the exercise at 50Hz vibration for 1 min on upper limb. They then performed 5 x 10 repetitions of eccentric contraction, at 85% of one rep maximum. Results showed that LVT had positive effects on ROM reducing muscle soreness 24, 48, 72 and 96h post exercise.

Imityaz *et al.* (2014) 45 healthy, but non-athletic females were distributed into three groups (n=15) including a VT group (VTG), a massage therapy group (MTG) and the control group (CG no intervention). MM was performed 15min prior to eccentric exercise in MTG and 50Hz mechanical vibration in supine position on the belly and the tendons of *biceps brachii* for five min prior to exercise in VTG. The researchers reported changes in blood LDH and CK concentrations and related these to the repetition maximum of the dumbbell exercise finding that VT and MM therapy have an equal effect on the prevention of DOMS. MM was effective in restoration of concentric strength, whereas VT resulted in earlier reduction of pain.

A broad descriptive overview of 15 recent studies outlining the effect of LVT on physical performance and summary thereof provided in Table 2.2 indicates that the majority of recent studies used a frequency range of 26-50Hz, although Shinahara *et al.*, (2005) also reported positive effect on H-reflex suppression using 75Hz. The majority of these recent studies also used LVT as an intervention to optimise training effect, rather than as a therapeutic modality post EIMD. Interestingly, of the six studies that focused on measures of flexibility (with and without concurrent stretching), five (83%) showed positive effect, while of the seven focusing on contractile force (and related aspects), five (71%) showed positive effect. Other positive outcomes were those with the attenuation in DOMS (two of three; 67%), while the effect on post EIMD and markers of inflammation, was not consistent.

A robust systematic review in which studies are selected according to carefully chosen inclusion criteria is therefore warranted.

#### 2.6.4 Conclusion

As summarised in Tables 2.1 and 2.2 and concluded by Osawa and Oguma (2013), LVT does appear to have positive effects in promoting relaxation of the muscle and increased joint flexibility. In a metaanalysis of 23 LVT and WBVT studies performed to date, Osawa and Oguma (2013), showed that VT had significant effects on flexibility alone as well as a significant additive effect on flexibility training, compared to identical conditions without VT. They attributed this to the suppression of the central nervous system owing to motor pool excitability (Vegar and Imtiyaz, 2012), a decrease in pain sensation (Lundeberg *et al.*, 1987), increased blood flow , relaxation of stretched muscles (Lythgo *et al.*, 2009) and inhibition of the muscular antagonist mediated by the GTO-IIB afferent neuron pathway (Stone *et al.*, 2003).

With reference to the tendon reflex referred to by Osawa and Oguma (2013), it has also been implicated in explaining the effects of application of LVT in order to reduce muscle stiffness. This is an ipsilateral polysynaptic inhibitory reflex activated by pressure on the GTOs or severe contraction of muscle which places tension on the tendons and their receptors. The effector response is relaxation of the agonist muscle (Ezeilo, 2002).

As the majority of these studies have focused on VT as a supplementary exercise training modality, it will be of interest to determine whether the analysis on the use of VT in reducing post EIMD muscle stiffness will confirm these findings.

## 2.7. Conclusion

This descriptive review of the related literature therefore reveals that in terms of the efficacy of LVT in enhancing recovery from EIMD, the studies display less heterogeneity, and those completed following EIMD are focusing on a particular muscle localized group and hence a comparison between the efficacy of LVT and MM was regarded as a viable comparison.

Of the 10 studies discussed, 5 appear to report positive results in terms of recovery post-EIMD. However the outcomes measured vary considerably. A similar conclusion was reached following the descriptive survey of MM.

A thorough systemic review of recent studies directed at specific outcome measures and meeting preset inclusion criteria in terms of study design and outcome measures, is therefore warranted before more clarity regarding the current status quo of the scientific evidence can be obtained.

## CHAPTER THREE

## Methodology

According to Petticrew and Roberts (2006), a systematic review comprehensively identifies, appraises, and synthesizes all the relevant studies on a given topic and is particularly pertinent to research in which there is uncertainty about the outcome of the effectiveness of an intervention. Ulman (2011) emphasises the importance of reducing selection bias by establishing a detailed and comprehensive plan and search strategy *a priori* and including a meta-analysis component to synthesise the data into a single quantitative summary.

After a clear identification of the research questions in Chapter One, this systematic review has been approached as follows:

#### 3.1 Completion of a comprehensive literature search and literature review

The online PubMed/MEDLINE EBSCOhost, Science Direct, Google Scholar and Research Gate databases were searched using the following key words: Massage, Manual massage (MM), Vibratory massage/therapy (VT), Eccentric exercise, Muscle damage, DOMS. Reference lists of potentially useful recent research publications were also searched to identify additional articles.

Thereafter a comprehensive purely descriptive literature review was written describing the state of the knowledge in each of the related areas of interest and including evidence obtained from both animal and human studies. This review is presented in Chapter Two.

#### 3.2 Determination of the criteria required for inclusion of trials in the systematic review

A trial was included in the systematic review if it

- (i) examined the effects of Swedish massage or a combination of a minimum of two classical MM techniques following induction of EIMD on humans in either gender or made use of local vibration therapy (LVT) before or following induction of EIMD
- (ii) could be classified as a prospective RCT. This included both self-controlled trials examining responses between left and right limbs and trials of cross over design in which there was evidence of randomisation.
- (iii) did not incorporate any additional treatment which would alter the independent massage variable specified in (i). In all cases, the control group was not to receive any alternative therapy including pharmaceutical aids, stretching and dynamic soft tissue mobilization (DSTM)
- (iv) was conducted on humans

- (v) was available as a full text publication in English, Portuguese, Germany or Dutch
- (vi) included outcome measures to quantify
  - measures of joint flexibility
  - measures of muscle strength and/or power output
  - muscle soreness/DOMS
  - systemic markers of inflammation
  - blood lactate concentrations and/or markers of muscle fatigue following EIMD.

# 3.3 Screening of the studies located and determination of whether they be included/excluded in the systematic review.

An exclusion criterion was the use of WBVT. As most WBVT is currently being used as exercise intervention (Cardinale and Bosco, 2003), rather than post-EIMD therapeutic modality, this was not regarded as fundamental to investigation of the research question examined in this thesis.

## 3.4 Critical appraisal of the studies to be included in the systematic review

Each study was appraised in terms of design, intervention, number of subjects and outcome measures quantified and assessed for homogeneity by two independent reviewers in order to ensure inter-rater reliability (Ullman, 2011).

As the trials investigating the effects of massage following induction of EIMD on humans examined too diverse a range of physical measures of muscle function and/or biochemical/immunological outcomes, it was decided not to pool the studies in the presentation of results, but present them in separate tables for the purpose of later analysis of results in terms of MM and LVT. The studies were therefore subdivided the studies investigating the effects of MM and LVT according to the following five categories/groups of outcome measures:

- measures of joint flexibility
- measures of muscle strength and/or power
- muscle soreness/DOMS
- systemic markers of inflammation
- blood lactate concentrations and/or markers of fatigue

Five tables were constructed and each study meeting the above-mentioned inclusion criteria and falling into the specified outcome category, was completed in a standardised pre-defined fashion in terms of author/s (date), trial design, intervention, number of subjects, outcome measures, and primary findings

for both MM and LVT. In the cases of excellent studies not quite meeting all inclusion criteria, they were included in the tables to provide additional insight to the reader, but highlighted and not included in the formal systematic review and quantification of final results.

#### **3.5** Synthesis of the studies

The outcomes of the studies contained in each table were thereafter analysed for general trends and summarised in the text above each table. In addition, the number of positive outcomes for each of the MM and VT trials was summarised in a separate concluding table (**Table 4.4.3**) with a graphic representation (**Figure 4.4.4**.) A statistician was consulted to verify the best appropriate statistical techniques to be used in classifying and interpreting the results and offer assistance in terms of how to present the findings.

#### 3.6 Statistical analyses

Due to the diversity of the outcome measures reported in the existing published research trials on the topic of the efficacy of MM and LVT following EIMD and unavailability of raw data providing sufficient detail regarding the outcomes specifying means and variances of the change post-EIMD in many of the qualifying studies, a formal meta-analysis involving statistical "pooling" of means and SDs, could not be done.

Following tabulation of the findings of the review, broadly categorised according to the five outcome measures listed above, a binary outcome summary for the trials in each category in which the similar methodology was used and that were therefore comparable, was created. As some of the studies examined more than one of the specified outcome measures, they appeared in more than one table.

After conducting a final numerical count of the number of studies with positive and negative findings in terms of the efficacy of MM and LVT in relation to each of the five outcomes examined, 2 x2 contingency tables were drawn up for each outcome measure examined and a single quantitative summary of the findings was obtained using Fishers Exact Test to establish whether the difference between MM and LVT for each specific outcome measure was statistical significant or not, using a pre-set probability level of 0.05.

Finally a qualitative comparison of the findings of the systematic review and those of Imityaz *et al.* (2014), the only comparative trial which have been appropriately presented to date on LVT vs. MM, was also conducted.

**3.7 Dissemination of the outcome of the systematic review.** This is described in Chapter Five and will result in a publication in an appropriate peer reviewed journal, and is also to be presented at a conference.

## CHAPTER FOUR

## Results

# 4.1 Introduction

Only randomized controlled trials (RCT's) that met the inclusion criteria in terms of manual massage (MM) and vibratory therapy (VT) detailed in Chapter 3 are summarized in the tabular analyses of the results which have been categorised according to

- measures of joint flexibility
- measures of muscle strength/power output
- muscle soreness/DOMS
- systemic markers of inflammation
- blood lactate concentrations and/or markers of fatigue

For the purpose of the actual systematic review, only RCT's that met all inclusion criteria, were included. Of the 63 studies screened, 28 articles satisfied the inclusion criteria and were included in the systematic review.

## 4.2 Manual massage (MM)

A summary of controlled trials performed on the effects of MM on measures of joint flexibility in human subjects following eccentric exercise is provided in Table 4.2.1. As is apparent from the trials summarized in this table, of the five controlled trials conducted on measures of joint flexibility following eccentric exercise on a total number of 82 participants, only four trials (n=72) were randomized and therefore fully qualified for inclusion in the systematic review. None revealed positive outcomes.

Authors (date)	Trial design	Intervention	N	Outcome Measures	Primary findings
(date) Abad <i>et</i> <i>al</i> .(2010)	RCT (treatment vs. 2control groups	<b>Group 1</b> : 6min of classical massage alone (no preceding exercise) <b>Group 2</b> : 30 supramaximal ECC contractions of elbow flexors + passive rest <b>Group 3</b> : classical massage immediately after 30 supramaximal ECC contractions of elbow flexors	18	ROM of elbow joint before, at 0, 24, 48, 72 & 96h	findings No significant difference between groups (p>0.05)
Bakowski <i>et al.</i> (2008)	RCT (arm to arm comparison)	10min massage, 30min after 8 sets of CON & ECC actions of elbow flexors with each arm Control: no treatment	14	ROM	No difference in ROM noted control and treatment arms (p>0.05)
Mancinelli <i>et</i> <i>al</i> .(2006)	RCT (treatment vs.control group)	17min effleurage, petrissage & manual vibration on a day of predicted peak soreness following 4-day training routine Control: no treatment	22	Length of <i>quadriceps</i> <i>femoris</i> muscle	No significant differences between groups (p>0.05)
Zainuddin <i>et al.</i> (2005)*	CCT (Arm to arm comparison)	10min Swedish massage 3h post ECC contraction of elbow flexors One arm received massage; other = control	10	ROM (elbow joint) before, at 30min, 1-4, 7, 10 & 14 days post- exercise	No significant difference in ROM between treatment & contralateral $\operatorname{arm}(p > 0.05)$
Hilbert <i>et al.</i> (2003)	RCT (treatment vs control group)	20min effleurage, percussion & petrissage 2h post 6 x 10 maximal ECC hamstring contractions Control= placebo lotion	18	ROM (knee joint) at 6, 24 & 48h)	No significant change between groups (p>0.05)

Table 4.2.1 Summary of controlled trials performed on the effects of manual massage (MM) on measures of flexibility in human subjects

DSTM: dynamic soft tissue manipulation; min: minutes; h: hour; ROM: range of motion; RCT: randomized controlled trial; CCT: clinically controlled trial; vs: versus; \*: not randomized; CON: concentric; ECC: eccentric

A summary of 14 controlled trials involving a total of 293 participants which investigated the effect of MM on the muscle strength and /or power in human subjects following eccentric exercise is provided in Table 4.2.2. Of the 14 trials, 11 were RCTs (n=255), only four trials (36%; Shin and Sung, 2015; Mancinelli *et al.*, 2006; Brooks *et al.*, 2005; Williams *et al.*, 2009) showed significantly positive improvement in the strength/power output of the massaged groups/limbs.

Authors	Trial design	Intervention	Ν	Outcome	Primary findings
(date)	_			measures	
Shin and	RCT	<b>Group1</b> (n=10):	21	EMG,	Increased
Sung (2015)	(treatment vs.	EIMD exercise (20 x		sonography,	activation of
	2 control	up & down stairs)		Proprioception (	gastrocnemius
	groups	placebo: nerve		dual	fibres and muscle
		stimulation pad.		inclinometer)	strength
		Group 2 (n=11):			
		EIMD exercise			
		+15min of effleurage,			
		milking, friction, skin			
		rolling- gastrocnemius			
	D.CTT	muscle	10		
Abad <i>et</i>	RCT	Group 1 : 6min	18	Muscle strength	No significant
<i>al.</i> (2010)	(treatment vs.	classical massage		(elbow flexors)	difference between
	2 control	alone (without			control & treatment
	groups	Crown 2: 20			groups
		Group 2. 50 supramaximal ECC			
		contractions of elbow			
		flexors + passive rest			
		Group 3: classical			
		massage immediately			
		after 30 supramaximal			
		ECC contractions of			
		elbow flexors			
Willems et	RCT	5min effleurage, 5min	7	One leg vertical	Significantly
al. (2009)	(leg to leg	petrissage, 5min		jump	greater increase in
	comparison)	tapotement & 10min		displacement	the massaged leg
		effleurage immediately		(before, at 24,	@ 48h (p<0.05)
		following 20min		72h)	
		downhill walk			
	DOT	Control: rest			
Frey Law <i>et</i>	RCT	Group I: superficial	44	MIT wrist	No significant
al.(2008)	(treatment vs.	touch		extension using	differences in post-
	control groups	Group 2:		dynamometer	exercise recovery
		1 min effleurage,		before treatment	of torque between $(n \ge 0, 0.5)$
		1 min effleurage		treatment	groups ( $p > 0.03$ )
		Group 3: Control : rest		ucalificiti	
		48 h following FCC			
		contraction of wrist			
		extensors			
Mancinelli et	RCT	17min effleurage.	22	Vertical jump	Significantly
al. (2006)	(Treatment vs.	petrissage & manual		displacement	greater increase in
()	control group)	vibration on a day of			the massage group
		predicted peak			(p<0.05)
		soreness following 4-			- /
		day training routine			
		Control: Post exercise			
		rest			

**Table 4.2.2** Summary of controlled trials on the effect of manual massage (MM) on muscle strength and power in human subjects following eccentric exercise

Zainuddin et al. (2005)*	CCT (Arm to arm comparison)	10min Swedish massage 3h post eccentric contraction of elbow flexors One arm received massage Control: no treatment (contralateral arm)	10	Elbow flexor strength using dynanometer	No significant changes in the reduction in MVI torque between arms ( $p=0.82$ )
Brooks <i>et al.</i> (2005)	RCT (blinded) 4 groups (n=13):	5min effleurage & friction to forearm & hand immediately after maximal isometric hand exercise Groups 1& 2: massage (dominant & non- dominant hand) Group 3: control Group 4: shoulder & elbow stretching	52	Power grip measurements on hand dynamometer at 5min	Greater effect on post exercise grip strength in massage groups (n=26) than both control groups ( $p$ <0.05)
Robertson <i>et</i> <i>al</i> . (2004)	RCO (treatment vs. control)	Treatment: 4x5min effleurage & kneading to back & front of both legs following 6 x 30 sec high intensity bouts of cycle ergometry. Control: passive rest	9	Peak & mean power output during Wingate cycling test	No significant effect of massage compared to control (p>0.05)
Jönhagen <i>et</i> al. (2004)	RCT (Leg to leg comparison)	4min effleurage & 8min petrissage 10min, 24h & 48h.following 300 maximal bilateral ECC contractions of <i>quadriceps femoris</i> . Control: no treatment/rest	16	Strength measured on leg dynamometer	No differences in loss of functional strength between the treatment & control legs (p>0.05)
Hilbert <i>et al.</i> (2003)	RCT(treatment vs control group)	20min effleurage, percussion & petrissage 2h post 6 x 10 max ECC hamstring contractions Control= placebo lotion	18	Peak torque of knee flexors using isokinetic device at 6,24 & 48h post exercise	No significant difference between the control and treatment group (p>0.05)
Farr <i>et al.</i> (2002)	RCT (leg to leg comparison)	30min effleurage & petrissage tone leg 2hr post 40min weighted downhill walk on treadmill Control : no treatment	8	Isometric & isokinetic strength; single leg vertical jump displacement at 1,24,72 &120h post walk	No significant differences between the treatment & control legs ( $p>0.05$ )

Hemmings	СО	Treatment: 50-60	8	Punch force (N)	No significant
et al.	(treatment vs.	strokes/min petrissage		registered on	difference between
(2000)*	control groups)	30 strokes /min		ergometer	the groups
, ,		effleurage		C	(p > 0.05)
		immediately after			
		punching the boxing			
		ergometer			
		Control: passive rest			
Rinder and	СО	6min leg effleurage &	20	Quadriceps	Increase in 50%
Sutherland	(treatment vs.	petrissage after		performance:	MVC in massage
(1995)*	control)	maximum number of		Total number of	group and decrease
	,	leg extensions @ 50%		50% MVC leg	observed with
		MVC		extensions	control group
		Control: passive rest			
Weber <i>et al</i> .	RCT	8min massage at 0 &	40	MIC & peak	No significant
(1994)	(treatment vs.	24h post exercise		torque assessed	between the groups
	control groups)	Group1: effleurage		on a Cybex 11+	( <i>p</i> >0.05)
		&petrissage		isokinetic	• /
		Group 2 : electrical		dynamometer	
		stimulation		before, at 0, 24h	
		Group 3: Upper body		post exercise	
		ergometry		•	
		Group 4: 10 reps of			
		post eccentric			
		contraction of elbow			
		flexors until fatigue			
		Control: passive rest			

UAC: upper arm circumference; MVI: maximal voluntary isokinetic; min: minute; h: hours; vs: versus; n: newton; CCT: clinically controlled trials; RCT: randomized controlled trials; RCO: randomized crossover design; CO: cross over design \*: not reported as randomized; MIC: maximal isometric contraction; MIT: maximum isometric torque; reps: repetitions; ECC: eccentric; EIMD: eccentric induced muscle damage; MVC: maximum voluntary contractions

A summary of 15 controlled trials performed on the effects of MM on muscle soreness, DOMS and related neuropeptides in human subjects following eccentric exercise is provided in Table 4.2.3. As is apparent from the trials summarized in this table, 12 RCTs involving (n=242) participants have been conducted on the effects of MM on muscle soreness and/or DOMS. Nine (75%) thereof showed a positive effect in reducing these measures. No evidence of an attenuation of the exercise-induced functional strength loss or biochemical markers of muscle soreness (CGRP and NPY) were, however, reported by Jönhagen *et al.* (2004).

Authors	Trial design	Intervention	Ν	Outcome	Primary findings
(date)	The woode		1,	Measures	go
Han <i>et al.</i> (2014)	RCT (treatment vs. control groups)	20 min massage( light stroking, skin rolling, friction, milking) vs. Placebo (inactive TENS pads) following up and down stairs in five- storey building	21	Muscle pain in gastronemius using algometer	Significantly reduced amount of pain in treatment group
Anderson <i>et</i> <i>al.</i> (2013)	arm comparison)	of <i>upper trapezius</i> muscle on dynamometer 48h post-exercise: 10min petrissage, friction, effleurage. 2h later: contralateral repeat Control limb: passive rest	20	10); Before, 0, 10, 20, 60min after treatment	reduced PMS; PPT@ most time points (p<0.05)
Abad <i>et al.</i> (2010)	RCT (treatment vs. 2 control groups (massage only; exercise only)	6 min of classical massage (effleurage, petrissage, tapotement) immediately after 30 supramaximal ECC contractions of elbow flexors	18	DOMS	No significant difference in the treatment group
Willems <i>et al.</i> (2009)	RCT (leg to leg comparison	5min effleurage, 10min petrissage & tapotement 10min effleurage in <i>quadriceps femoris</i> immediately following 20min downhill walk	7	DOMS on scale of 1-10	Significantly lower at 48h post- exercise in <i>rectus</i> <i>femoris</i> and <i>vastus lateralis</i> (p<0.05)
Frey Law et al.(2008)	RCT (Treatment vs. Control group)	Group1: superficial touch Group 2: 1min effleurage, 4min petrissage; 1min effleurage, Group 3: Control : rest 48 h following ECC contraction of wrist extensors	44	PPT DOMS using VAS 0h, 48h, post treatment	Significantly greater reduction in PPT & DOMS in Group 2 compared to Group 3 (p<0.05)
Bakowski <i>et</i> <i>al.</i> (2008)	RCT (arm to arm comparison)	10min massage, 30min after 8 sets of CON& ECC actions of elbow flexors with each arm Control: no treatment	14	Perceived Soreness before exercise, after ex,10min, 6,12,24,36,48, 72 and 96h	Non-significant 10%-20% decrease in soreness was noted with control compare to control

**Table 4.2.3** Summary of controlled trials on the effect of manual massage (MM) on muscle soreness and/or DOMS in human subjects following eccentric exercise.

Mancinelli <i>et</i>	RCT	17min effleurage	22	PPT using	Decrease in
al (2006)	(Treatment vs	netrissage & manual	22	algometer	pressure pain
<i>ui.</i> (2000)	(Treatment vs.	vibration to thighs on a		argonneter	threshold in
	Control group)	day of predicted peak			massaged group
		day of predicted peak			massaged group
		soreness following 4-			( <i>p</i> <0.05)
		day training routine			
		Control: rest			
Zainuddin et	CCT	10min Swedish massage	10	Muscle	20%-40%
al. (2005)*	(arm to arm	3h post ECC		soreness	decrease in
	comparison)	contraction of elbow			soreness in
		flexors			massaged arm
		One arm received			( <i>p</i> <0.05)
		massage			• <i>′</i>
Jönhagen <i>et</i>	RCT	4min effleurage	16	VAS (1-10)	Significant
al. (2004)	(leg to leg	8min petrissage	_	Muscle CGRP	difference
	comparison)	10 min 24 48 h		& NPY	between legs were
	comparison)	following 300 maximal		concentrations	observed
		bilateral ECC		in each leg	00501700
		contractions of		in cach leg	
		contractions of			
		quadriceps jemoris			
TT'11 4 4 7	DOT	Control: no treatment	10	T 1 C	D 11 1
Hilbert <i>et al.</i>	RCI	20 min effleurage,	18	Level of	Decreased level
(2003)	(treatment vs	percussion & petrissage		soreness	of soreness in
	control/placeb	2h post 6 x 10 maximal			treatment group at
	o group	ECC hamstring			48h post-exercise
		contractions			( <i>p</i> <0.05)
		Control= placebo lotion			
Farr <i>et al</i> .	RCT: counter	30min effleurage &	8	DOMS at	Very significant
(2002)	balance trial	petrissage to one leg 2h		1,24,72,120h	reduction in
	(leg to leg	post 40min weighted		post walk	muscle soreness
	comparison)	downhill walk on			& tenderness in
		treadmill			massaged leg at
		Control: no treatment			24h (p<0.001)
Lightfoot <i>et</i>	RCT	Group1: stretching	31	DOMS	No significant
al. (19972)**	(treatment vs	Group2:10min			difference
( )	control)	petrissage			between groups
		Group 3: control			Secure Browps
		following 60 reps of			
		heel-drop exercise			
Smith <i>et al</i>	RCT	30min Swedish massage	14	DOMS at 8	Reduced DOMS
(1001)	(treatment us	(affleurage &	17	21 18 72 06	from 24 06h post
	(incatinent vs	patrissage) 2h past ECC		27,70,72,70	$\frac{10112}{24-9011} \text{ post}$
	control group)	allow extension /flower		& 12011 post	exercise $(p < 0.05)$
		elbow extensor/mexor		exercise	in massaged
		CACICISC.4-5 Sets at			group
XX7 1 · · ·	DOT	Control: rest	40	9	
weber <i>et al.</i>	KUI	8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8	40	Soreness	INO SIGNIFICANT
(1994)	(treatment vs	24h post exercise		rating scale	aifference
	control)	Group1: effleurage		scale (1-10)	between the
		& petrissage(n=10)			groups
		Group 2: electrical			( <i>p</i> >0.05)
		stimulation(n=10)			
		Group 3: Upper body			
	1		1	1	

	Group 4: Control (n=10)		
	Control: rest		

RCT: Randomized controlled trial; CCT: clinical controlled trial; min: minutes; PMS: Perceived muscle soreness; DOMS: delayed onset muscle soreness; PPT: pain-pressure threshold; VAS: visual analog scale; h: hour; vs.: versus \* not randomized \*\*:petrissage only; MVC: maximum voluntary contraction VAS: visual analog scale CGRP: Calcitonin gene-related peptide; NPY: neuropeptide Y; TENS: transcutaneous electrical nerve stimulation; ECC: eccentric; CON: concentric

A summary of controlled trials on the effect of MM on systemic markers of inflammation in human subjects following eccentric exercise is provided in **Table 4.2.4**. Six trials were located of which four RCTs were included involving 51 participants. For CK, one of the two trials (50%) showed a decrement for 6 days (Smith *et al.*, 1994). For neutrophil count, one (Smith et al., 1994) showed decrease in the first 1h and the other (Hilbert *et al.*, 2003) showed no difference. Cortisol decreased in the first 5h in one trial (Smith *et al.*, 1994). Only one trial (Crane *et al.*, 2012) examined TNF $\alpha$ , NF $\kappa$ B, IL-6 and PGC-1 $\alpha$  and found that massage had a beneficial effect on these markers of an inflammatory response.

Authors	Trial Design	Intervention	Ν	Outcome	Primary findings
(dates)	_			Measures	
Crane et al.	RCT	10min of MM:	11	Serum TNFa	ΤΝFα & ΝFκΒ
(2012)	(Leg to leg	• 2min effleurage		Serum NF <sub>K</sub> B	reduced ( <i>p</i> <0.05) at 0h
	comparison)	• 3min petrissage		Serum IL-6	only
		• 3min slow		Muscle PGC-1a	IL-6 ( <i>p</i> >0.05) reduced
		stripping		concentrations	after 2.5h;
		• 2min effleurage		immediately &	PGC-1α higher in the
		Control: no treatment		2.5h post	massaged leg 2.5 h
		after EIMD		intervention	after treatment.
					( <i>p</i> >0.05)
Zainuddin <i>et</i>	CCT	10min Swedish	10	Plasma CK	CK peak values in
al. (2005)*	(arm to arm	massage 3h following		Concentration	massaged arm lower
	comparison)	ECC contraction of			than that in control
		elbow flexors			arm
		One arm received			( <i>p</i> <0.05)
TT'11		massage	10	NT . 11	
Hilbert <i>et</i>	RCT	20min effleurage,	18	Neutrophil	No significant
al.(2003)	(treatment	percussion &		count	difference between
	vs. control	petrissage 2h post 6 x			groups ( $p < 0.05$ )
	group)	10 maximal ECC			
		namstring contractions			
		lotion			
Forr at al	PCT:	30min effleurage &	8	Plasma CK at 0	No significant
(2002)	counter	petrissage tone leg 2h	0	24 48 72 96	difference between
(2002)	balance trial	following 40min		and 120h post	limbs at any of time
	(leg to leg	weighted downhill		exercise	intervals $(p < 0.05)$
	comparison)	walk on treadmill		CACICIDE	
	companyon)	Control: no treatment			
Lightfoot <i>et</i>	RCT	Group1: stretching	31	Plasma CK	No significance
al. (1997)*	(treatment	Group2: 10min of	-	levels	changes between the
	vs. control)	petrissage			groups
	,	Group 3: passive			5 1
		control			
		post 60 repetitions of			
		heel-drop exercise			
Smith <i>et al</i> .	RCT	30min Swedish	14	Serum Cortisol	Decreased
(1994)	(treatment	massage (effleurage		Serum CK	concentration of
	vs. control	and petrissage) 2h		Neutrophil	cortisol during first 5h
	group)	following eccentric		concentration	post-exercise
		elbow extensor/flexor			Reduced post-exercise
		exercise: 4-5sets at			CK throughout 6 days,
		MVC			reduced post-exercise
		Control: rest			neutrophil for 1h post
					exercise ( $p < 0.05$ )

**Table 4.2.4** Summary of controlled trials on the effect of manual massage (MM) on systemic markers of inflammation in human subjects following eccentric exercise

RCT: randomized controlled trial; TNF $\alpha$ : tumor necrosis factor –alpha; NF $\kappa$ B: nuclear factor kappa B; IL-6: interleukin-6; PGC-1 $\alpha$ : peroxisome proliferator-activated receptor coactivator 1 $\alpha$ ; CK: Creatine Kinase; vs: versus; h: hour; min: minutes; \*: petrissage only; MVC: maximum voluntary contractions; EIMD: exercise induced muscle damage; ECC: eccentric; CCT: controlled clinical trials

A summary of controlled trials examining the effect of MM on blood lactate concentrations and/or systemic markers of fatigue in human subjects following eccentric exercise is provided in Table 4.2.5. Three trials (n=52) out of seven were included in the analysis and showed no significant change in blood lactate concentration and/or on muscle fatigue.

Authors Trial design Intervention Outcome Primary Ν (date) Measures findings RCT Group1 (n=10): EIMD 21 Shin and BLa conc No Sung (treatment vs. 2 exercise (20 x up & significant difference down stairs).placebo (2015)control groups nerve stimulation pad. between Group 2( n=11): EIMD groups exercise +15 min of (p>0.05) effleurage, milking, friction, skin rollinggastrocnemius muscle Hemmings Counter 50-60 strokes/min 8 BLa conc No et al. 2000)\* significant balance trial petrissage (treatment vs. 30 strokes /min difference control groups) effleurage between immediately after groups punching the boxing (p>0.05) ergometer RCT Dolgener 20min of 22 BLa conc No Group 1: passive 3, 5, 9, 15 & significant and Morien (treatment vs. (1993) recovery (rest) difference control groups) 20min post Group2 : bicycle treatment between recovery groups in Group3: effleurage and BLa pertissage concentration after an exhaustive (p>0.05)treadmill run\*\*\* RCO 9 Robertson et BLa conc Treatment : massage No al. (2004) (treatment vs. Control: passive rest difference in 20min of effleurage and control) BLa conc kneading 5min post passive vs. 6x30 sec eccentric massage exercise on cycle group ergometer Monedero CO Group1: Passive rest 18 BLa conc Combined and Donne (treatment vs. Group 2: Active cycling massage & (2000)\* control) @ 50% V<sub>02</sub>max cycling was Group 3: Massage most Group4: combined efficient massage and cycling intervention Non-specified massage (p < 0.05)15min post 5km trial CO Group1: passive rest 10 BLa conc Lower BLa Gupta *et al* (1996)\*(treatment vs. Group 2: active rest @ for active 30% VO<sub>2</sub>max control) group than Counter Group 3: massage other groups. balance trial Kneading & stroking of No upper & lower limbs significant difference in

 Table 4.2.5 Summary of controlled trials on effect of manual massage (MM) on blood lactate

 concentrations and/or systemic markers of fatigue in human subjects following eccentric exercise

					massage vs. control group (p>0.05)
Bale and James (1991)*	CO (treatment vs. control)	Group1: passive rest Group 2: active rest @ 60% VO <sub>2</sub> max Group3: massage (17min each)	9	BLa conc	Significant decrease noted with massage compared to control group (p < 0.05)

RCT: randomized controlled trial; min: minutes; vs: versus; conc: concentration; EIMD; exercise induced muscle damage; MVC: Maximum voluntary contraction; \*: Not randomized; counter balance trial; vs: versus; BLa: blood lactate; conc: concentrations; CO: cross-over design; RCO: randomised crossover design

## 4.3 Vibratory Therapy (VT)

The results of studies that have compared the efficacy of local vibration therapy (LVT) with that nonmassaged controls or control limbs, are subdivided into those that examine the following outcomes: measures of flexibility, measures of strength and power output, DOMS and muscle soreness, fatigue and /or blood lactate concentrations and systemic markers of inflammation in human subjects and presented.

A summary of RCTs located on the effect of VT on measures of joint flexibility in human subjects in Table 4.3.1. As is apparent in the table, three trials involving (n=60) participants examined the effects of LVT following eccentric exercise and (67%) confirmed a positive effect of LVT on ROM and the other showed no effect.

yAuthors (date)	Trial design	Intervention	N	Outcome Measures	Primary findings
Mohammedi and Sahebazamani (2012)*	RCT (treatment vs. control)	LVT @ 50Hz with a vibrator pre-exercise for 1 min prior to 5 sets x10 reps of eccentric contraction @ 85% of 1-RM Control: no treatment following ECC exercise of <i>biceps</i> <i>brachii</i>	30	ROM of elbow joint	Increase in ROM in treatment group for 48h post exercise (p>0.05)
Lau and Nosaka (2011)	RCO (treatment vs. control, crossover design)	30 min LVT (non- specified) at 30 min, 1, 2, 3 and 4 days following ECC exercise Control: no treatment	15	ROM	Faster increase in ROM in treatment group compared to control ( <i>p</i> <0.05)
Herda <i>et</i> <i>al</i> .(2009)	RCO (treatment vs control)	Group 1: 20min of Passive stretch Group 2: prolonged vibration only Group 3: control (no treatment) after isometric maximal voluntary of the	15	Passive ROM	No change noted in VT vs control groups.

**Table 4.3.1** Summary of controlled trials on the effect of local vibratory therapy (LVT) on measures of joint flexibility in human subjects.

LVT: local vibration therapy; RCT: randomized controlled trial; RCO: randomized cross-over design; EIMD; exercise induced muscle damage \* intervention prior EIMD; ROM: range of motion; min: minutes; vs: versus; 1-RM: 1 repetition maximum; reps: repetitions; ECC: eccentric

plantar flexors

A summary of controlled trials on the effect of LVT on muscle strength and power output in human subjects is presented in Table 4.3.2. Four RCTs involving 70 participants met all inclusion criteria. Although 50% (Herda *et al.*, 2009; Shinohara *et al.*, 2005) reported that prolonged vibration increased the short latency component of the stretch reflex, the discharge rate of motor units and the fluctuations in force during contractions by a hand muscle, 50% (Barnes *et al.*,2012; Lau and Nosaka, 2011) did not confirm significant change following the LVT intervention.

Authors	Trial design	Intervention	Ν	Outcome	Primary findings
(dates)				Measures	
Barnes <i>et al.</i> (2012)	RCT (treatment vs. control) Cross over trial	5 set of 1min LVT with 26Hz immediately, 12h & 24h post 300 maximal eccentric contraction of q <i>uadriceps</i> of one leg on an isokinetic dynamometer Control: no treatment	8	Peak and average peak ISO tension & isokinetic CON & ECC torque prior to exercise, 24h & 48h post exercise	Significantly greater decrease in peak & average peak ECC torque 24h post exercise compared to control ( $p$ <0.05)
Lau and Nosaka (2011) Herda <i>et</i>	RCO trial (treatment vs. control RCO	30 min LVT (non- specified) at 30 min, 1, 2, 3 and 4 days following eccentric exercise Control : no treatment Group 1: 20min of	15 15	Maximal isometric torque using isokinetic dynamometer Voluntary	No significant difference in muscle strength post treatment VT group (p>0.05) No significant
al.(2009)*	(treatment vs control)	passive stretch Group 2: prolonged vibration only Group 3: control (no treatment) after isometric maximal voluntary of the plantar flexors		Peak torque (PT)	difference between groups(p>0.005)
Shinohara et al. (2005)	RCT (treatment vs. control) Control (n=12) Treatment (n=20)	30min LVT with electromagnetic vibrator on relaxed muscle post 2 sets of 10 constant –force contraction on the 1 <sup>st</sup> dorsal interosseous muscle of left hand & subsequently followed by another session of eccentric exercise Control: no treatment	32	Mean EMG amplitude of the S-L component of stretch reflex, discharge rate of motor units, muscle force on the left hand measured with a force transducer	Mean EMG amplitude of the S-L component of stretch reflex, discharge rate of motor units and fluctuations in force during contractions of the hand significantly enhanced (p < 0.05)
Bosco <i>et</i> <i>al</i> .(1999c)	RCT	30Hz LVT of 1 min of 10 reps on the upper limb during maximal arm curl with extra load of 5%BM	12	Mechanical power	Greater improvement in treated group vs control ( $p < 0.05$ )

**Table 4.3.2** Summary of controlled trials on the effect of local vibratory therapy (LVT) on muscle strength and/or power output in human subjects

LVT: local vibration therapy; RCT: randomized controlled trial; RCO: randomized cross over; min: minutes; reps: repetitions; vs: versus; &: ISO: isometric; CONC: concentric; ECC: eccentric; MVC: maximum voluntary contraction; \*no eccentric exercise; \*\*: vibration as exercise intervention; EMG: electromyography; S-L: short-latency; PT: voluntary peak torque
A summary of controlled trials on the effect LVT on DOMS and muscle soreness in human subjects provided in Table 4.3.3. Six trials were located with five trials (n=140) meeting the inclusion criteria. Four trials (80%) showed positive effect and one (Ayles *et al.*, 2011) showed no significant effect.

Authors	Trial design	Intervention	Ν	Outcome	Primary findings
(dates)				Measures	
Xanthos <i>et al.</i> (2013)**	RCT (treatment (vibration) vs. control (traditional)	10 reps of 1 min LVT (26Hz, 4.5mm) post ECC exercise & prior to the repeat of ECC exercise	13	DOMS Muscle soreness	No decrease in DOMS No difference in muscle soreness in treatment vs control (p < 0.05)
Mohammedi and Sahebazamani (2012)*	RCT (treatment vs. control)	LVT @ 50Hz with a vibrator pre-exercise for 1min prior to 5x10 reps of ECC contraction @ 85% of 1-RM Control: no treatment	30	Muscle soreness before, after, 24h, 48h, 72h and 96h	Less DOMS in the experimental group compared to control group ( $p < 0.05$ )
Ayles <i>et al.</i> (2011)	RCT (leg to leg comparison)	LVT@ 50Hz with vibrating device of 1cm <sup>2</sup> size of the probe was performed after ECC exercise. Control: no treatment	16	PPT (algometer) DOMS	No significant change in muscle soreness
Lau and Nosaka (2011)	RCT (treatment vs. control crossover design)	30min LVT (non- specified) at 30min, 1, 2, 3 and 4 days post ECC exercise Control: no treatment	15	DOMS	Less DOMS in the experimental group compared to control group $(p < 0.05)$
Broadbent <i>et</i> <i>al.</i> (2010)	RCT (treatment vs. control)	LVT 5mm with 40Hz 3 bouts of 1min vibration for 30min 5 days post exercise Control: no treatment	29	DOMS	Less DOMS in the experimental group compared to control group ( $p < 0.05$ )
Bakhtiary et al. (2007)*	RCT (treatment vs. control)	LVT 50Hz for 1 min on quadriceps, hamstring & calf muscles of left & right prior to 30min walk down a 10 <sup>0</sup> declined treadmill @4km/h Control: no treatment	50	DOMS measured with VAS after 24h	Less DOMS in the experimental group compared to control group ( $p < 0.05$ )

 Table 4.3.3 Summary of controlled trials on the effect of local vibratory therapy (LVT) on DOMS and muscle soreness in human subjects

LVT: local vibration therapy; RCT: randomized controlled trial; WBVT: Whole Body Vibration Therapy; \*: prior to exercise intervention; DOMS: delayed onset muscle soreness; deg: degrees; min: minutes; vs: versus; trad: traditional modality; \*\* control received an intervention; TT: time trial; HIIT high intensity interval training; VAS: visual analog scale; PPT: pain pressure threshold; ECC: eccentric

A summary of controlled trials on the effect of LVT on systemic markers of an inflammatory response is provided in Table 4.3.4. Only two trials examined the change in serum CK concentration, including 65 participants. One thereof (50%; Lau and Nosaka, 2011;) showed no significant change while that of Bakhtiary *et al.* (2007) showed significantly lower levels of CK in the treatment group.

Authors	Trial design	Intervention	n	Outcome	Primary findings
(dates)				Measures	
Lau and Nosaka (2011)	RCT (treatment vs. control : crossover	30min LVT (non- specified) at 30min, 1, 2, 3 & 4 days post ECC exercise	15	Serum CK concentration 4 days after exercise	No significant effect noted in serum CK in both groups (p>0.05)
Bakhtiary <i>et</i> <i>al.</i> (2007)***	RCT (treatment vs. control)	LVT 50Hz vibration with a vibrator for 1 min on quadriceps, hamstring and calf muscles of left & right prior to 30min walk down a 10 <sup>0</sup> declined treadmill @4km/h Control: no treatment	50	Serum CK concentration Measured after 24h	Significantly lower mean CK levels treatment group (p < 0.05)

**Table 4.3.4** Summary of controlled trials on the effect of the local vibratory therapy (LVT) on systemic markers of inflammation

LVT: local vibration therapy; RCT: randomized controlled trial; min: minutes; CK: creatine kinase; vs: versus; h: hours; \*\*\* intervention prior to eccentric exercise; ECC: eccentric

A summary of 2 trials which qualified for inclusion in the review and examined the effect of LVT on fatigue and /or lactate in human subjects following strenuous exercise is presented in Table 4.3.4. Only one trial involving a total of 12 participants, was conducted on blood lactate concentration and muscle fatigue showed no effect of LVT.

Table 4.3.5 Summary of trials on the effect of local vibra	tory therapy (LVT) on fatigue and /or blood
lactate concentrations in human subjects	

Authors	Trial design	Intervention	Ν	Outcome	Primary
(dates)				Measures	findings
Cafarelli et	RCT	4min percussive	12	Muscular	No effect on the
al.	(Treatment vs.	vibratory massage & 1		fatigue	rate of fatigue in
(1990)	control)	min of rest following		(calculated	control and
		repeated sets of static		from a	vibrated
		contractions of		regression line	conditions
		quadriceps muscle @		fit to the	
		@70% MVC and		decline of the	
		following 30min		periodic	
		cycling at 75% VO <sub>2</sub>		MVCs)	
		max.			
		Control: no treatment			

LVT: local vibration therapy; min: minutes; RCT: randomized controlled trial and control had intervention; \*: counter balance trial; vs: versus; CK: creatine kinase; CRP: C-reactive protein; MVC: maximum voluntary contractions

### 4.4 Collective findings:

Table 4.4.1 summarises the findings of a recent study (Imitiyas *et al.*, 2014) examining the comparative effect of LVT and MM in the prevention of delayed onset muscle soreness(DOMS) in terms of ROM, muscle strength and power, muscle soreness/ or DOMS, inflammatory markers and markers of muscle fatigue. Imityaz *et al.* (2014) reported greater efficacy of LVT in attenuating ROM and strength deficits following EIMD.

**Table 4.4.1** Summary of the randomized controlled trial of Imtiyaz *et al.* (2014) examining the comparative effect of local vibration therapy (LVT) and manual massage (MM) in the prevention of delayed onset muscle soreness (DOMS) in terms of range of motion, muscle strength and power, muscle soreness/ or DOMS, inflammatory markers and markers of muscle fatigue

Group1: 5 min LVT	45	ROM of elbow	ROM @48, 72 h significant recovery by LVT &
50Hz		joint (using	MM groups, no difference reported between groups
Group 2: 15 min non-		goniometer) &	No difference in MIF vs control group in LVT &
specified MM		MIF, RM	MM groups
Group 3: no treatment		measured	RM No difference between LVT groups and control
prior to eccentric exercise		before,	@48h
of elbow flexor muscles		immediately	LVT significant difference @pre & 48h post
using dumbbell with		post	exercise vs massage group( p<0.01)
elbow flexed from $(50^{\circ} -$		intervention,	MM significant recovery vs control group (p=0.00)
$170^{\circ}$ ) extension in $4-5$		0,24, 48, 72h	
sec.		post exercise	
		Muscle soreness	Reduction in muscle soreness in both LVT (24, 48
		measured	& 72h) & MM groups (48 & 72h post exercise)
		before,	vs control group; (p>0.05)
		immediately	
		post	
		intervention,	
		0.24, 48, 72h	
		post exercise	
		using VAV	
		Serum CK.	Significant difference in CK between the massaged
		LDH.	groups (LVT, MM) & control at 48h $(p=0.000)$ :
		concentration	VT: significantly less LDH vs control group
		measured	(n<0.05) @48h: MM group no significant
		hefore	difference( $n > 0.05$ )
		immediately	
		nost	
		intervention	
		18h post	
		-on post-	
		CACICISC	

LVT: local vibration therapy; MM: manual massage, VT: vibration therapy; MIF: Maximum isometric Force; RM: repetition maximum, LDH: lactate dehydrogenase; CK: creatine kinase

## 4.4.2 Comparison of MM and VT

As is shown in Table 4.4.1 and Figure 4.4.1, no studies supported an attenuation of the muscle stiffness resulting from MM applied following strenuous eccentric exercise. Although 50% of studies examining

the effect of LVT following strenuous eccentric exercise, confirmed a positive effect on post exercise ROM/and or flexibility, only 36% of 11 RCT studies (n=255) confirmed an improvement in the strength deficit following MM, two of four studies (50%) confirmed a positive outcome in strength following LVT. The difference between the efficacy of MM and LVT in improving strength deficit was not statistically significant (p>0.05).

Both MM and LVT displayed positive outcomes in terms of post-exercise muscle soreness and DOMS with a larger percentage of the total number of LVT trials being positive. This difference (75% vs 100%) was not statistically significant (p>0.05). Only one study showed a positive effect of MM on CK (p>0.05); while there was no evidence of blood lactate level being affected by either form of massage. The Fisher's Exact Test revealed that the difference between the efficacy of MM and LVT was not statistically significant (p=0,142). Due to the absence of a positive outcome, the Fisher's Exact Test could not be applied for the blood lactate MM vs. LVT.

**Table 4.4.3** The percentage of studies showing a positive effect of manual massage (MM) and local vibratory therapy (LVT) on measures of flexibility, strength, muscle soreness, inflammation and blood lactate concentration and comparison to the findings of Imtiyaz *et al.* (2014)

Outcome Meesure	Manual N	lassage		Local Vib	ratory T	herapy	Imtiyaz <i>et al.</i> (2014)
	No of trials (no of particip ants)	Posit ive outc ome	% +ve	No of trials (no of participa nts)	Positi ve outco me	% +ve	Result
Measures of joint flexibility	4 (72)	0	0 %	3 (30)	2	67%	positive @ 48, 72h post
Strength/Power	11 (255)	4	36%	4 (70)	2	50%	Greater difference after 48h in VT group
Soreness/ DOMS	12 (242)	9	75%	4 (140)	4	100%	Reduction in muscle soreness in both VT (24, 48 & 72h) & MM groups (48 & 72h post exercise) vs. control group
Inflammatory Marker (CK)	2 (22)	1	50%	2 (15)	1	50%	VT & MM positive @ 48h
Blood Lactate / Fatigue	3 (52)	0	0%	1 (12)	0	0%	Not assessed

MM: manual massage; VT: vibratory therapy; h: hour; CK: creatine kinase; h: hours; vs: versus; CK: creatine kinase; DOMS: delayed onset muscle soreness



**Figure 4.4.1** The percentage of studies showing a positive effect of manual massage (MM) and local vibratory therapy (LVT) on measures of flexibility, strength, muscle soreness, inflammation and blood lactate concentration. P >0.05; Fisher's Exact Test; DOMS: delayed onset muscle soreness; CK: creatine kinase

# CHAPTER FIVE Discussion

#### **5.1 Introduction**

Despite the popular appeal and widespread use of massage among sportsmen among sportsmen over centuries, a consensus in terms of its beneficial effects is difficult to obtain because of wide variations in techniques, time, area of body, and outcome measures.

Although every attempt was, however, made to ensure that the systematic review was as robust as possible, numerous differences in experimental design and execution made straightforward comparison of the trials difficult and excluded the possibility of pooling the results and completing a quantitative meta-analyses.

### These included the

- heterogeneity of trials and lack of standardization in massage procedures
- lack of sufficient data and the method used not being the same or comparable
- use of different methods and time points to measure similar outcomes. For example, Bakhtiary *et al.* (2007) measured serum CK concentrations 24h after exercise whereas Lau and Nosaka (2011) measured it 4 days post exercise.
- different timing and duration of the massage sessions
- variations in the exercise model used to induce muscle damage and the protocols used the lack of sufficient studies using randomized control groups
- fact that blinding as an inclusion criterion in massage based intervention studies, would not be realistic.
- rare use of placebo treatments

Nevertheless, every attempt was made to conduct a thorough systematic review of the state of the knowledge in this field by setting a number of inclusion criteria which were strictly implemented.

Firstly, classic western massage or Swedish massage is the most common type of MM that is currently used (Weerapong *et al.*, 2005). As previously mentioned, this consists a variety of techniques rather than only one technique such as effluerage alone. For this reason, use of at least two different techniques accepted in the classic massage or sports massage repertoire, was set as a fundamental inclusion criterion for this review.

Secondly only controlled trials in which there was a control group or limb that did not receive any alternative therapy, were included in this systematic review. All studies in which the control phases did not consist of passive rest, were excluded from this systematic review.

A third fundamental inclusion criterion that was rigidly applied in this review, was that of randomization. As is evident in the tabulation of the results, if it was not clearly stated in the description of the study that the division of participants or limbs was randomized, the study was not included in the assessment. In the case of otherwise valuable studies providing important results, they were described in tables and lightly highlighted, but not included in the final systematic review and concluding analysis.

Due to the clinical relevance being specifically focussed at physiotherapy settings, the systematic review focussed only on LVT as oppose to WBV which is usually applied in the full body and often as modality to enhance performance during exercise.

#### 5.2 Decrements in performance following EIMD

The first important finding of this systematic review was a lack of evidence in favour of a superior attenuation of the functional declines occurring following strenuous eccentric exercise namely, loss of flexibility and loss of strength of either modality (MM vs. LVT).

#### 5.2.1 Joint Flexibility

The loss of flexibility following eccentric exercise and EIMD was confirmed in each of the four RCTs on a total number of 72 participants (Table 4.2.1) prior to examining efficacy of massage in reducing this. As described on page 13, attaining a point of "no myofilament overlap and failure to re-interdigitate" results in ultrastructural damage. Clarkson and Sayers (1999) explain that this damage can result in excitation - contraction coupling and cross-bridge formation.

Despite the evidence MM has been shown to increase blood flow to the muscle (Kerschan-Schindl *et al.*, 2001), reduce sympathetic stimulation favouring muscle relaxation (Turnbull *et al.*, 1982), suppress the H- reflex (Sullivan *et al.*, 1991), the finding of this systematic review showed that none of the four RCTs included in the review, revealed a positive outcome. While the sample size was small (n=4), this does indicate that an attenuation of the loss of flexibility induced by EIMD, is not supported.

As is evident in Table 4.3.1, LVT however, had a positive impact on joint flexibility in two of the three RCTs (67%) conducted on 30 participants. In terms of a possible mechanism, Morelli *et al.* (1990) and Sullivan *et al.*, 1991 propose that the kneading action and pressure exerted on the GTO during LVT, reduces the H-reflex via suppression of the central nervous system owing to a decrease in motor neuron pool excitability (Anderson *et al.*, 2008).

Another possible reason is that mobilising and elongating shortened or adhered connective tissue while applying LVT may increase muscle-tendon compliance by activating myofascial trigger point (MTrP) activity, resulting in less stiff muscle-tendon units (Moraska, 2005).

Previous studies also suggest that the improvement in ROM by vibration is associated with pain alleviation (Lundeberg *et al.*, 1984; Pantaleo *et al.*, 1986), increase in blood flow (Kerschan-Schindl *et al.*, 2001), relaxation of stretched muscles (Turnbull *et al.*, 1982) and inhibition of muscular antagonist mediated by the Golgi tendon organ -Iβ afferent neuron pathway (Bove *et al.*, 2003).

However, the findings of this review and lack of significance obtained when a Fishers Exact test was applied to 2x2 contingency tables, does reject the null hypothesis set at the commencement of the study and not provide evidence that LVT is significantly more effective in attenuating loss of flexibility induced by EIMD. Due to the small sample size (n=4; n=3), a high p value may also have resulted from a Type II error and a need for further studies exists.

#### 5.2.2 Muscle Strength Deficits

Of the 11 RCTs including 255 participants which investigated the effect of MM on measures of muscle strength and /or power in human subjects following eccentric exercise summarised in Table 4.2.2, four trials (36%) showed a significantly positive improvement in the strength/power output of the massaged groups/limbs ( $p \le 0.05$ ). In the case of LVT, only four RCTs investigating parameters related to the strength deficit following EIMD and 2 of these studies (50%) showed a significant improvement. The difference between the efficacy of the two trials was not significant (p>0.05), but this may once again be a Type II error due to the small number of studies which met the inclusion criteria.

Of importance is however the fact that the attenuation of strength gains did take place in a number of studies using each modality. Although this is not a consistent finding, it has been reported following both MM and VT after EIMD.

Previous studies suggest that the mechanism responsible for this strength loss could be excitationcontraction coupling failure which plays an important role in force reduction observed after eccentric exercise (Hubal and Sayers, 2008), overstretching of sarcomeres during the lengthening of the muscles which occur with eccentric contraction (Howell *et al.*, 1993) as well as disruption in calcium homeostasis (Weber *et al.*, 1994) and loss of CK activity (Armstrong et al., 1991) that occurs with EIMD.

According to Vegar and Imityaz (2012), VT helps with the synchronization of motor unit activity by preventing sarcomere disruption and also improves muscular strength, power development and

kinaesthetic awareness. Bosco *et al.* (1999) also mention that vibration results in neuromuscular activation whereby the local tendon and muscle vibrations stimulate muscle spindle and I $\alpha$  fibres, which mediate the monosynaptic and polysynaptic pathways (Hagbarth and Eklung, 1985; Siedel, 1988).

#### 5.3 DOMS and systemic markers of inflammation

As detailed in Table 4.4.3, the results of this systematic review of randomized controlled studies firstly revealed that MM and LVT both had positive effects on attenuating the symptoms of DOMS. Trials performed on the effects of MM on muscle soreness, DOMS and related neuropeptides on 12 randomized controlled trials involving 242 participants showed a positive effect in reducing DOMS in 75% (n=9) of the studies, while each of the trials investigating the effects of LVT, on DOMS also showed a positive outcome. This confirms the findings of the meta-analysis completed by Torres *et al.* (2012) investigating the effects of MM on DOMS only.

In terms of DOMS, most of these studies compared healthy subjects receiving massage with the control group receiving no treatment (rest) only. As the outcomes were all measured using slight variations of VAS and were relatively homogenous and this allowed for a robust comparison. The only shortcoming in terms of rating of the study design, was the absence of blinding in most of the studies. While Hilbert *et al.* (2003) did however ensure that their studies were single blinded on the side of the researchers/blinded examiner/testers taking post intervention measures, and Hane *et al.* (2014) reported the use of a placebo intervention such as TENS pads, the failure to include this characteristic of good study design, as inclusion criterion, is a somewhat unavoidable limitation of this systematic review. Ullman (2011) emphasises, absence of a placebo treatment makes it impossible to exclude the psychological influence or effects.

Timing of the massage sessions is also an important extraneous variable. Torres *et al.* (2012) reported that massage applied after exercise is effective on muscle soreness only after 24h post exercise. This confirms the recommendation of Tiidus (1997) who showed that massage also had an effect on inflammatory response and may be able to affect the later development of muscle soreness. However, he proposed that for massage to be able to disrupt the initial stages of muscle damage or progression and inflammatory response, it will have to be applied 1-2h post eccentric exercise. This confirmed the speculation of Tidis in 1997 that if 30 min of massage (effleurage and petrissage) is performed within 2h after exercise will positively affect the migration of neutrophils from the site of the damage in muscle (Smith *et al.*, 1994). Unfortunately the timing of the MM sessions in this review was not consistent. This may account for some of the negative outcomes reported in terms of attenuation of DOMS.

In the case of VT, numerous researchers have found LVT applied before induction of EIMD effective in attenuating DOMS and inflammatory markers of EIMD and accelerating recovery from EIMD. The importance of this variable requires further investigation.

As is evident in Table 4.3.3, although LVT did appear to have greater consistency in attenuating the symptoms of DOMS, the Fisher's Exact test did not show superiority (p>0.05) of LVT over MM in reducing DOMS following EIMD. These findings are supported by the study of Imityaz (2014), who also found that LVT had the same positive effect as MM on symptoms of DOMS at 24, 48 and 72 hr post EIMD.

Interestingly, the reduction in DOMS was also associated with a reduction in PPT in measured using an algometer in two of the studies and with the intramuscular concentration of the related neuropeptide, muscle CGRP and NPY in the study of (Jönhagen *et al.*, 2006) *et al.* (2004). As this is, however, the only work having investigated this association, further studies are required in order to test the validity of this finding.

In terms of inflammatory markers found in the circulation associated with DOMS, the early finding of Tiidus (1997) who showed that massage also had an effect on inflammatory response and may be able to affect the later development of muscle soreness, was of interest.

As indirect measures of muscle damage, CK concentrations only showed a correlation with DOMS in 50% (n=2) of the trials in which it was measured (n=4). The large individuality in the link with DOMS and influence of genetics, age, and gender and dependence on the time-points at which measurements were taken as well as isoenzymes measured (as described on page 8), may have resulted in these discrepancies in the association between DOMS and these muscle enzyme levels in blood . However the work of Crane *et al.* (2012) reported attenuated levels of PCG1 $\alpha$ , confirming its role as potent vasoregulatory neuropeptides (Jönhagen *et al.*, 2004) and involvement in the modulation of pain. Furthermore the reduction in TNF- $\alpha$  and IL-6 in addition to NF $\kappa$ B confirm reduction of the level of inflammation (Crane *et al.*, 2012).

Insufficient studies have, however, investigated this relationship between DOMS and systemic markers of inflammation in order to verify the proposal of Kresge (1988) that massage reduces DOMS by increasing muscle lymph flow, circulation and muscle relaxation. Further in-depth studies investigating massage induced decrements in DOMS following EIMD that investigate at the correlation between these markers of inflammation and levels of DOMS after EIMD need to be undertaken.

The time-points at which blood sampling is taken after application of MM or VT also needs to be kept consistent between studies before a meta-analysis can be undertaken to reveal the association. The other factors that influence the ability of massage to give an effect are types and durations of massage

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as well as pressure applied varies based on the therapist's experience and preferences (Callaghan, 1993). The tissue healing and psychological effect of massage may also require attention for future research.

Ayles *et al.* (2011) have recently suggested that the positive effect of VT in attenuating DOMS could be due to the stimulation of muscle spindles and increase in its afferent activity that is caused by vibration; that vibration reduces the perception of pain through same mechanism of pain gait theory, leading to increase in background tension and motor unit activity (Weerapong *et al.*, 2005). As mentioned on page 72, the isolated muscle or tendon vibration could lead to activation of primary and secondary muscle spindle endings so as the I $\beta$  afferents from GTOs (Burke *et al.*, 1976). It also reduces I $\alpha$  afferent transmission because of an increased level of presynaptic inhibition. (Ritzmann *et al.*, 2013).

As described in the literature review, Tiidus (1997) states that there is very little evidence that MM has any significant impact on muscle recovery following an exercise and in support of the finding that light exercise on the affected muscles may be effective in improving blood flow and short-term reduction of muscle soreness than MM.

However, in the cross over design studies the effects of the RPE could have contributed to the positive results that was obtained for example, Lau and Nosaka (2011) found that vibration reduced the symptoms of DOMS 2 - 5 days after exercise. The ultrastructural disruption seen after eccentric contraction and the level of disruption appears to be reduced if the subjects have been previously exposed to this type of training. As Cheung *et al* (2003) mentions, the RBE in which a single bout of exercise strengthens the muscle for the following eccentric bouts (McHugh, 2003), may well have been an extraneous variable influencing the outcomes in all of the cross-over studies of DOMS in both MM and VT.

#### 5.4 Blood Lactate Concentration and/or Markers of fatigue

As discussed in the literature review, blood lactate concentration is not a marker of EIMD per se. It is only of interest in terms of indirectly reflecting the increased blood flow accompanying MM and LVT. While earlier numerous studies included this variable in their evaluations of the efficacy of MM, this appears no longer to be included in more recent studies focusing on the benefits of LVT. In the case of the MM studies, these were usually conducted in comparison to other intervention strategies such as active post-exercise recovery and stretching and often did not contain untreated control groups, hence did not qualify for inclusion in this systematic review as would anticipated according to the state of the knowledge regarding lactate clearance from the blood described on pages 15, 28 & 29 blood lactate uptake was not accelerated during post exercise recovery in the 4 studies reported (Table 4.2.5).

Only one study investigated another parameter which may provide an indirect indication of recovery from fatigue post EIMD, namely a regression line fit to the decline of repeated MCVs. This interestingly did also not provide a positive result in terms indirectly reflecting recovery from damage to the muscle cell.

## CHAPTER SIX

# Conclusion

The findings of this systematic review confirm that both MM and LVT potentially offer benefits in terms of more rapid recovery from the functional decrements of EIMD. However the potential superiority of one modality over another cannot be proven until such time as more randomised controlled clinical trials involving larger sample sizes and minimal bias, are conducted which will provide sufficient statistical power and quantitative data to undertake a thorough meta-analysis and /or statistical comparison of the two interventions.

# CHAPTER SEVEN

# **Directions for future research**

Future studies should involve larger sample sizes. The size of the sample was found to be one of the flaws in many studies done on massage and resulted in possible type II errors (Ernst, 1998).

The studies need to be randomised, controlled, blinded and homogenous in terms of the intervention used to induce EIMD, the age and gender of the trial participants and the outcome measures assessed.

The timing and duration of the massage sessions need to be standardized in future studies.

The time-points of blood/muscle sampling after application of MM or VT need to be kept consistent between studies before meta-analyses can be undertaken for the purpose of comparison of the two modalities.

Further in-depth studies investigating massage induced decrements in DOMS following EIMD that investigate at the correlation between TNF- $\alpha$ , IL-6, CGRP and NF $\kappa$ B markers of inflammation and levels of DOMS after EIMD need to be undertaken.

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# Appendix A

Léonard Féasson <Leonard.Feasson@univ-st-etienne.fr> I agree for using this figure in your thesis, with her reference. I thank you for the interest for our work. Good luck for your drafting Regards Prof. L. Féasson Le 18 janv. 2016 à 16:22, Silungile To Silungile Ntshangase

Jan 18 at 6:20 PM I agree for using this figure in your thesis, with her reference. I thank you for the interest for our work. Good luck for your drafting Regards Prof. L. Féasson

Le 18 janv. 2016 à 16:22, Silungile Ntshangase <<u>ntshangasesilungile@yahoo.com</u>> a écrit :

> Dear Dr Feasson,

>

> I am a Masters student in Sports Medicine at the University of Kwa-Zulu-Natal, South Africa and am undertaking a systematic review examining the efficacy of different forms of massage in alleviating exercise-induced muscle damage.

>

> As part of my literature review, I would like to include the following figure which was originally published by you in the article below in 2002:

>

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> Feasson L, Stockholm D, Freyssenet D, Richard I, Duguez S, Beckham JS and Denis C. Molecular adaptations of neuromuscular disease of associated proteins in response to eccentric exercise in human skeletal muscle. J Physiol. 543(1): 297-306, 2002.

>

> Please could I ask you for permission to replicate this figure in my thesis? I would so appreciate it!

>

> Looking forward to hearing from you,

>

> Silungile Ntshangase

> Masters Student in Sports Medicine

Pr Léonard FÉASSON Unité de Myologie Centre Référent Maladies Neuromusculaires Rares Rhône-Alpes CHU de St Etienne - 42055 Cedex 2

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# Appendix B

# RESULT SUMMARY: SYSTEMATIC REVIEW

# Contingency Tables

# (i) Flexibility

	Positive	Negative	TOTAL
MM	0	4	4
VT	2	1	3
TOTAL	2	5	7

p>0.05

## (ii) Strength and Power

	Positive	Negative	TOTAL
MM	4	7	11
VT	2	2	4
TOTAL	6	9	15

p >0.05

### (iii) Soreness/DOMS

	Positive	Negative	TOTAL
MM	9	3	12
VT	4	0	4
TOTAL	13	3	16
			p>0.05

### (iv) Creatine Kinase concentration

	Positive	Negative	TOTAL
MM	1	1	2
VT	1	1	2
TOTAL	2	2	3
			p>0.05

### (v) Blood Lactate concentration

	Positive	Negative	TOTAL	
MM	0	3	3	
VT	0	1	1	
TOTAL	0	4	4	
			p>0.05	