The influence of pressure stimulus intensity on pain perception and neuromuscular performance in healthy males.

Submitted for the Degree of Doctor of Philosophy

At the University of Northampton

2013

Annika Elisabeth Wing

ABSTRACT

Participation in elite level sport is inherently linked with exposure to pain (Bartholemew et al., 1998; Heil, 1993) with those able to tolerate it whilst maintaining performance levels gaining a competitive edge over their opponents (Egan, 1987). Studies have shown that the presence of experimental pain reduces muscle performance (Farina et al., 2002; Farina et al., 2008) when pain is induced via hypertonic saline injection but this is an invasive method of pain induction whereby it is unclear if there is any interference of normal muscle functioning due to the injection of fluid into the muscle (Wing et al., 2011a). The series of studies presented within this thesis aimed to investigate whether this relationship existed when pain was induced via a non-invasive Gross Pressure Device (GPD) and subsequently the effect of severity of pain on muscle performance. The final stages of the research explored the possible location of inhibition; central or peripheral.

The GPD was established to be a reliable method of pain induction at pain perception threshold level both inter-session and intra-session. A protocol for use was established in pilot testing which was followed throughout the research. Pain perception threshold level was established for all participants prior to undertaking the experimental trials as well as their maximal ramped contraction within an isometric knee extension measured on an isokinetic dynamometer which was subsequently used for normalisation. The experimental trials consisted of three explosive isometric voluntary contractions of which the peak maximal voluntary contraction (MVC) was selected for analysis. Electromyographic activity (EMG) of the vastus lateralis and semitendinosus were also measured during the contraction to indicate neural activation. Immediately following each painful trial participants were asked to rate the painfulness of the condition on a visual analogue scale (VAS).

Inducing pain at perception threshold level produced a mean reduction in maximal force by 9%-12% when pain was induced ipsi-laterally, and 11% when induced contra-laterally. When pressure exerted by GPD was doubled and then trebled from previously established pain perception threshold, a greater reduction in maximal force was reported (18% & 21% respectively). When placebo and nocebo conditions were introduced and therefore there was a disparity between the expected pain severity and the stimulus applied, while there was a significant difference in the perception of pain severity reported via VAS ($F = 27.971$; $p < 0.05$), no significant difference in force output was found ($\chi 2(2) = .452$, $p>0.05$).

The studies presented within this thesis have demonstrated that pain induced at perception threshold level reduced muscle performance even when a non-invasive method of pain induction was used. Furthermore it was found that there is an incremental decrease in performance relative to severity of pain induced. Concomitant reduction in force and EMG suggest that inhibition is likely to be neuronal but the level of this could not be established. However, the fact that pain induced contra-laterally produced similar reductions in force as ipsi-lateral pain induction suggests inhibition is unlikely to be at peripheral level. Furthermore manipulation of the severity of pain perceived was found not to alter muscle performance, suggesting that inhibition is unlikely to be at cortical level; rather it is likely to be sub-conscious. Further work should be conducted to determine the mechanisms by which pain reduces muscle performance and the effect of pain on dynamic, multi-joint tasks in order that effective strategies can be developed for athletes to perform when experiencing pain.

KEY WORDS: Pain severity, Isometric contraction, Neural activation, Pain perception, GPD

CONFERENCE PROCEEDINGS

European Congress of Sport Sciences (2011). Oral Presentation: "The severity of experimentally induced pain influences muscular performance during maximal voluntary isometric knee extensor contractions"

British Association of Sport and Exercise Sciences (2011). Oral Presentation: "Expectation of pain intensity does not influence neuromuscular performance but does influence pain perception during a maximal isometric knee extension task"

ACKNOWLEDGMENTS

I would firstly like to thank the many people who volunteered as participants in the studies conducted within this thesis. For some this was involvement in one study, whilst others participated in all the studies, from the original pilot work right through to the final study which involved participation in nine studies over a period of 4 years. It is never easy to recruit participants, but in a pain study the appeal of taking part is even lower than most, so I am genuinely grateful to all of you for your time and willing. Needless to say, without you this research could not have been done.

I would also like to thank Prof. Joseph Maguire and Dr. Michael Atkinson of Loughborough University who in different ways inspired this PhD. Prof. Maguire first inspired the topic of study, and it was through Dr. Atkinson's enthusiasm for research and always putting the question before any preference for methods that this project evolved in the manner it did.

I would also like to acknowledge the contribution of my supervisory team; Prof. Jackie Campbell, Dr. Peter Jones, Ass. Prof. Tony Kay and Dr. Stuart Raleigh who have guided me through the research process and the subject-specific skills and knowledge required in each discipline underpinning my investigations. It has been really interesting to see the different perspectives each of you brings to the topic based on your individual expertise and this has been incredibly valuable in developing the interdisciplinary aspects of the research which was my initial aim. I hope that the process of working with a social scientist hasn't been too frustrating for you all, but instead has provided you with some amusement along the way and ultimately a greater sense of achievement in the end product which you helped to create.

I would also like to acknowledge the help I received from others outside of the supervisory team who have helped me out when needed. In particular I would like to thank Lucy Hammond and Dr. Stuart Allen who at various points have been a real support both academically and on a personal level.

Finally I thank my family and friends for their support, especially Ella & Fran who have always been there to pick me up when I have most needed it. And of course, my parents (currently proof reading!) who never see that much of me these days but support me regardless. They bought me up to believe that following the path of least resistance rarely leads anywhere exciting, so you should never be afraid of a challenge – something I believe has served me well throughout life, but particularly during the course of this PhD.

DECLARATION OF ORIGINALITY

This thesis submitted for the degree of Doctor of Philosophy entitled "*The influence of pressure stimulus intensity on pain perception and neuromuscular performance in healthy males*" is based on work conducted by the author in the School of Health at The University of Northampton mainly during the period between February 2009 and July 2013.

All the work recorded in this thesis is original unless otherwise acknowledged in the text or by references. If necessary for the deposit of this thesis in the institutional repository, permission to disseminate third party material has been sought and granted by copyright holders.

None of the work has been submitted for another degree in this or any other University.

Name: Annika Elisabeth Wing

Signed ..... Date .....16/10/2013.....

TABLE OF CONTENTS

GLOSSARY OF ACRONYMS

DOMS	Delayed onset muscle soreness
EMG	Electromyographic
GCT	Gate control theory
GPD	Gross pressure device
IQR	Interquartile range
MVC	Maximum voluntary contraction
SD	Standard deviation
SG	Substantia gelatinosa
ST	Semitendinosus
VAS	Visual analogue scale
VL	Vastus lateralis

TABLE OF TABLES

TABLE OF FIGURES

1 INTRODUCTION

"Your training partner's name is pain. You start out trying to ignore him. Can't do it. You attempt to reason with him. No way. You try to strike a bargain. Hah. You plead. You say 'Please stop, please go away. I promise never ever to do this again if you just leave me alone.' But he won't. Pain only climbs off if you do. Then you're beaten."

(Martin, No date)

Pain is a likely consequence of regular participation in elite sport (Bartholemew et al., 1998), both during performance and as a consequence of long term exposure to the rigours of the elite athlete's lifestyle (Heil, 1993). There is an inherent association between pain and successful performance in elite sport (Egan, 1980) and therefore the ability to cope with pain and still maintain optimal physical performance becomes an important issue for a competitive athlete. The commercialisation of sport means that there is a great deal of money invested into the elite levels by sponsors, broadcasters and governments. In the UK for example Olympic sports are set to receive £125 million a year in the run up to the 2016 Olympic Games. With such a substantial investment there is an expectation of success in return. UK sport sets targets for each sport and failure to achieve these targets means the sport suffers a cut in funding; for example table tennis, wrestling, handball and basketball all had their funding removed entirely following an unsuccessful Olympics in London (uksport.gov.uk). This means that for the governing bodies there is a need to achieve success at world class levels on a yearly basis in order to maintain the levels of funding they have and therefore the support they can offer their athletes.

This pressure to perform results in a focus upon every facet of sporting performance in order that where improvements can be made, they are. The amount an athlete needs to improve their performance from year to year varies by sport and individual so it is hard to put a figure on what would be considered a worthwhile change in performance measures. However, a study reporting the smallest worthwhile change in performance for elite triathletes suggests a ~0.5% decrease in race time is the smallest important change an athlete requires in order to be competitive (Paton & Hopkins, 2005), which is comparable to track athletes who are thought to require a 0.3-0.5% decrease of race time depending on discipline, or field athletes requiring a

0.9-1.5% improvement in distance (Hopkins, 2005). These figures are based on past results and are therefore not direct predictors of success for future competition, but they demonstrate that the improvements required at the elite level are marginal due to the tight nature of competition at that level.

The number of variables thought to affect performance in sport is vast, ranging from physiological adaptations to training and psychological preparedness through to the social context of the sport and participant. It is however recognised that pain management is a central factor in performance enhancement (Addison et al., 1998) and therefore it is believed that an increased understanding around the interaction between pain and the neuromuscular system may allow for development of interventions to overcome the deficits in performance experienced and thereby help athletes to achieve gains in performance. Experimental muscle pain has been reported to significantly reduce maximal strength (Ervilha et al., 2004; Graven-Nielsen et al., 1997; Farina et al., 2004, 2005) but as yet no comprehensive explanation for the mechanism of this has been identified.

Research over the past 60 years has established that there is a difference in the way athletes and non-athletes respond to pain (Low et al., 2005; Ryan & Foster, 1967) with athletes reported to have a higher tolerance for pain (Janal et al., 1994; Ryan & Foster, 1967) which alters depending on their chosen sport (Pen & Fisher, 1994; Straub et al., 2003). The level to which the athlete competes is also a differentiator of their tolerance towards pain, with high level athletes able to endure heightened severity of pain in comparison to lesser able athletes (Scott & Gijsbers, 1981). Studies investigating the influence of pain on performance have reported that the presence of experimental muscle pain induced via hypertonic saline injection reduces maximal muscle strength (Ervilha et al., 2004; Farina et al., 2004, 2005; Graven-Nielsen et al., 1997) and the ability to carry out complex tasks (Brewer et al., 1990). Whether athletes develop the ability to tolerate pain because they engage in sport, or whether they take part in sport because they are better able to tolerate pain remains unclear and is an area requiring further investigation as data suggesting any form of causal relationship is not available (Tesarz et al., 2012).

A further important consideration is the method by which pain was induced in the studies described above (hypertonic saline injections) which was highly invasive by nature (Ervilha et al., 2004; Farina et al., 2004, 2005; Graven-Nielsen et al., 1997). As the saline solution was injected directly into the muscle in which performance was measured it is not possible to determine that the changes identified were purely as a result of the pain experienced rather than confounding variables associated with the introduction of a foreign agent within the

muscle. The use of injections is also ethically sensitive and difficult to control where the severity of pain induced is altered, thereby limiting the scope of investigations using these methods to account only for differences between "pain" versus "no pain" rather than being able to examine the complexities of the pain-performance relationship which is necessary in order to gain a clear understanding of the mechanisms underpinning any changes in performance identified.

The aim of the current research is to gain a better understanding of the mechanisms associated with reductions of muscle performance related to the presence of pain. Figure 1.1 illustrates the structure of the current research which takes the form of a series of progressive studies building upon the knowledge gained in each one.

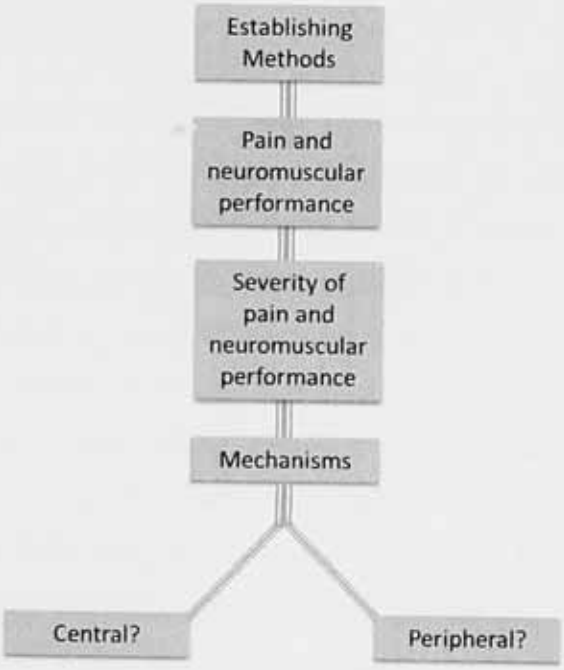


Figure 1.1. Overview plan of the current research

In order to investigate the potential mechanisms by which pain influences muscular performance it was firstly essential to establish a method by which to induce pain reliably and in a non-invasive manner so that any changes in performance reported could be attributed to the pain stimulus and not confounding variables. Once methods to induce pain were established, the relationship between pain severity and muscle performance was examined. Establishing whether there is a relationship between the two allowed for proceeding studies to manipulate expectation and thereby explore the possible location of pain related changes. The location of inhibition associated with performance losses was the final focus of the research in order to suggest whether level of inhibition is likely to be central or peripheral.

2 LITERATURE REVIEW

2.1 INTRODUCTION

This chapter reviews pain mechanisms and their influence upon athletic performance, the development of pain theories, and some possible explanations for the physiological mechanism of pain sensation and experience. The literature regarding pain in the athletic domain was also reviewed with regard to athletic pain tolerance, methods of experimentally inducing pain, and how anticipation of pain may limit performance. The International Association for the Study of Pain (IASP) defined pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage (Loeser & Treede, 2008). It may be interpreted as a warning system that places a limit on sports capabilities (Prokop, 2000) in an attempt to prevent potential damage to the individual.

Pain can be induced in a variety of ways and so the measurements used to indicate level or severity of pain can also vary significantly from study to study. There are however, two thresholds that can be standardised across all methods of pain induction; pain perception threshold and pain tolerance threshold. The maximum pain that an individual can tolerate is often used to compare across groups of individuals and their ability to deal with a particular pain stimulus (Ryan & Kovacic, 1966) and is referred to as 'pain tolerance'. A limitation to this method of measurement is that taking someone to their pain tolerance threshold can be ethically problematic and hence the use of this measurement needs to be clearly justified in the methodology. An alternative threshold measure is 'pain perception threshold', defined as the lowest perceived sensation that is labeled by the participant as painful. This has been found to be the more reliable measure of pain in the literature (Loeser & Treede, 2008) and as it is at the lowest end of the pain scale, makes it more appealing to researchers working in an ethically sensitive field.

2.2 PAIN AND ATHLETIC PERFORMANCE

Elite level sport often not only results in, but also requires pain to be experienced and endured either during the performance (Egan, 1987; Major, 1996), or as a consequence of long term exposure to the rigours of the elite athlete's lifestyle (Heil, 1993), and is widely accepted by athletes as part of their daily life (McMaster & Troup, 1993). The physiological and psychological requirements of pain in terms of information processing are thought to be given a high supra-spinal priority as pain identification and avoidance is implicit in order to survive (Price, 2000), a suggestion supported by numerous studies examining brain imaging (Derbyshire et al., 1997, 1998), cognitive performance (Crombez et al., 1998a, 1998b, 1999;

Eccleston & Crombez, 1999) and changes in cortical and behavioural responses when exposed to a pain condition during performance (Lorenz et al., 1997; Lorenz & Bromm, 1997). Given that pain is so deeply associated with elite level sport and that it may be an important performance factor, it warrants further investigation.

2.2.1 ATHLETIC PAIN TOLERANCE

Differences in pain tolerance of athletic versus non-athletic participants have been reported with those involved in athletic pursuits better able to tolerate high levels of pain in comparison to their non-athletic counterparts (Giesbrecht et al., 2005; Hall & Davies, 1991; Ryan & Kovacic, 1966). Ryan & Kovacic (1966) used a novel Gross Pressure Device (GPD), where a plastic gridiron cleat was positioned over the tibia with a sphygmomanometer cuff then inflated against the tibia until the participant could no longer tolerate the pain. The researchers found that contact-sport athletes tolerated significantly higher levels of acute pressure pain than athletes from non-contact sports and that both athletic groups were better able to tolerate acute pressure pain than non-athletes. This finding was later supported by Janal et al. (1994) who found that athletes reported higher pain thresholds across three different methods of experimental pain induction (cold-pressor, cutaneous heat and tourniquet ischemic pain) in comparison to the non-athletic control group.

Pen & Fisher (1994) suggested a hierarchical tolerance to pain with those involved in contact sports being significantly more able to tolerate experimental pain than non-contact sport athletes, who in turn had higher pain tolerance levels than non-athletes. The authors suggested that this relationship was evident because individuals choose their sport, or involvement in sport, based upon their ability to tolerate pain, which would suggest that people with a low pain tolerance would not choose to take part in sporting activity. Conversely, those with high pain tolerance would find that they were able to tolerate the greater pain demands of contact sports, be more successful within the sport and hence would opt to take part in that type of activity (Nideffer, 1978; Pen & Fisher, 1994; Williams, 1978). This theory assumes that people will generate a number of schemata by which they can judge how suited they are to a sport by how it relates to their previous experiences (Taylor & Taylor, 1998).

No conclusive evidence exists to indicate cause or effect however, so an equally valid hypothesis could be that exposure to contact sports increases the ability to tolerate pain. Using anecdotal evidence, Egan (1987) suggested that the training undertaken by athletes may desensitise them to pain related to that particular sport and that it is not the innate ability of the athlete to cope with pain that is fundamentally important to performance, but instead it is their lack of sensitivity to pain that allows them to perform. This is in accordance with the

"adaptation-level theory", which implies that painful experiences can change the internal anchor points for subjective evaluation of pain and thereby determine ability to cope with pain (Anshel & Russell, 1994). Approximately 3-5 million injuries occur each year amongst recreational and competitive sportspeople (Kraus et al., 1984) and pain is the most frequently cited psychological condition associated with athletic injury according to sports medicine practitioners (Brewer et al., 1989). Whilst injury forms its own section in the literature distinct from pain, this suggests most sports-people will have had experience of injury at some point in their career which would likely also expose them to pain. It would be expected that the athlete therefore had dealt with painful situations more than a non-athletic counterpart. This could mean that, as hypothesised by Egan (1987), athletes become desensitised to pain and therefore better able to cope with a pain stimulus. Alternatively, it could mean that through exposure to pain they develop coping strategies allowing them to maintain muscular control and reduce the negative impact of pain as hypothesised by Scott & Gijsbers (1981). However, the precise mechanisms for this remain unclear.

In an effort to further understand the way in which athletes react to pain, Scott & Gijsbers (1981) conducted an investigation examining differences between athletic levels. The findings within the study are in agreement with the studies reported above, suggesting that athletes have a better tolerance for pain than non-athletes (Janal et al., 1994; Pen & Fisher, 1998; Straub et al., 2003). Importantly, Scott & Gijsbers (1981) also made a distinction between athletes based on performance level. Using ischaemia as the method of pain induction, they reported that national squad swimmers had significantly higher pain tolerance levels in comparison to club level swimmers, who in turn had significantly higher pain tolerance to those from a non-athletic group. The authors suggested that the athletes training to a high level have more control over their muscular system due to the amount of time spent training and are therefore able to limit the debilitating effects of pain. Therefore, it was the swimmer's ability to cope with the effects of pain, and not ability to tolerate pain itself that gave high level athletes an advantage over athletes of a lower ability or non-athletes.

The conclusions drawn by Scott & Gijsbers (1981) were based on the assumption that pain has a debilitating effect on an organism and prevents it from functioning normally. Therefore, through repeated exposure to painful conditions, the athlete learns to cope with the limitations and adapt to pain. However this explanation was speculative and not what the study specifically examined and therefore further investigation into coping strategies and learning to deal with pain associated to sports performance is needed. While it is unclear whether greater pain tolerance is inherent or learnt, it is apparent that regardless of the type of pain induction

method used, the type (contact or non-contact) and level (national-, club-level or non-athlete) of athlete influences the individual's ability to tolerate pain.

2.2.2 PAIN COPING STRATEGIES IN ATHLETIC PERFORMANCE

Individuals may have a physiological tolerance to pain, or it may be that certain individuals have a better ability to cope with pain during performance (Bartholemew, 1998), which would suggest that the differences reported between athletes and non-athletes in their response to pain stem from pain coping techniques and cognitive strategies that are either learned as a result of sports performance (Jaremko et al., 1981; Ryan & Foster, 1967) or lead the individual to choose a certain sport (Pen & Fisher, 1994). The cognitive strategies of former Olympic cyclists for dealing with the pain associated to their performance was analysed by Kress (1999) in a qualitative investigation in which several higher order themes common amongst those interviewed were outlined showing that pain, preparation, mental skills, mind and body, optimism, control and "house in order" were all areas thought to be important in tolerating pain during performance. Those that were physically and mentally prepared for the competition and the pain associated with it were found to experience less pain than those lacking these attributes. Accordingly Kress (1999) concluded that pain within the performance was simply a perception rather than a physiological fact.

In an investigation looking at the use of cognitive strategies in sport, Gauron & Bowers (1986) reported a marked reduction in pain experienced by injured athletes suffering chronic pain, again suggesting an element of psychological control that can be trained within individuals. In relation to sports performance Spink (1988) analysed the use of different cognitive strategies and linked a dissociative cognitive strategy with pain reduction and improved performance in a swim test. Attentional focus (Ahles et al., 1983; Nouwen et al., 2006) and anxiety (Villemure & Bushnell, 2002) have also been indicated to alter pain perception in experimental studies and an argument can therefore be made that it is the interpretation of a pain stimulus rather than the intensity of the stimulus itself that determines a person's ability to cope with pain. This would suggest limitations may be imposed through psychological mechanisms rather than physical (Pen & Fisher, 1994) and therefore the athlete could be taught to develop coping mechanisms or techniques to deal with pain. Individuals within sport may tolerate pain in different ways and with varying levels of success (Ryan & Kovacic, 1966) but in many sports there is an inherent association of pain with performance (Iso-Ahola & Hatfield, 1986), and therefore the way in which individuals react to pain can contribute to performance. Pain and the likelihood of pain occurring can induce a fear or feeling of perceived threat, which may decrease an individual's level of pain tolerance and hence

adversely affect performance (Friedman et al., 1985) regardless of what their baseline tolerance for pain is. It is therefore important to all levels of sport and to all performers.

The ability of pain to take control of an organism can result in performance decrements (Scott & Gijsbers, 1981) as the athletes are no longer able to utilise their full muscular potential, hence offering one possible explanation for performance losses associated with pain. Whilst such behaviour was not found to occur with elite swimmers in the Scott & Gijsbers' study, Brewer et al. (1990) found that pain did inhibit motor performance during complex tasks where pressure pain was used to induce a pain response. The authors suggested that the pain stimulus induced a state of over arousal, which in turn had a negative effect on the performance of complex tasks. Linked to the inverted-U arousal and performance relationship (Yerkes & Dodson, 1988) this suggests that there may be an optimal level of pain, above and below which performance decreases. In a further investigation Birch et al. (2000), concluded that when exposed to pain during low precision tasks that muscle activity would decrease, whereas in high precision tasks there was no reported effect of pain which may introduce other variables such as selective attention that need to be considered when looking at pain responses in relation to performance.

The competitive sports environment should also be viewed as a variable influencing pain tolerance levels according to Sternberg et al. (1998) who analysed experimental pain sensitivity in male and female collegiate athletes two days before a competition, immediately following the competition, and two days after the competition. Comparing these results to those of matched non-athlete controls, they found that competition dramatically reduced the perception of noxious stimuli and concluded that competition induces both hyperanalgesic and analgesic states that are dependent on the body region tested and the pain assessment methodology, therefore the pain that is felt in competition cannot always be generalised between individuals. It does however provide a further factor in need of consideration when searching for the optimal level to be achieved on the inverted-U scale in a competitive environment and this could be very different from a training environment. This could have significant implications on coaching practices and preparing the performer for competition. It suggests that not only is exposure to pain important in learning to deal with its effects (Egan, 1987) but also competition in order for the athlete to recreate similar emotional states to those experienced when performance is measured (Sternberg et al., 1998).

2.2.1 EXPERIMENTAL PAIN INDUCTION

Numerous methods of inducing pain have been reported within the literature such as electric shock (Neddermeyer et al., 2008; Tursky, 1972), hypertonic saline injections (Arendt-Nielsen et al., 1996; Ervilha et al., 2005), delayed onset muscle pain (DOMS) (Dannecker et al., 2008) and thermal pain stimuli (Dannecker et al., 2008; Hatayama & Shimizu, 1993; Neddermeyer et al., 2008). All of these methods of pain induction are conducted in a laboratory setting which means that a number of experimental variables must be considered besides the method of pain induction, including pain measures used, environmental cues, experimenter appearance and possible personal biases and their effect on the participant (Neddermeyer et al., 2008). Inducing pain experimentally allows the experimenter to control the experience of the participant to a certain extent and to investigate effects of pain on 'normal' subjects. However, a major limitation of inducing pain experimentally in a laboratory is that they are inherently safe (Pen & Fisher, 1994) which means that the individual knows that they are going to come to no harm and hence the emotional, or fear, component of pain is reduced.

Much of the current literature relating to pain and muscle performance used invasive methods of injecting saline to induce pain (Arendt-Nielsen & Graven-Nielsen, 2008; Ervilha et al., 2004, 2005; Farina et al., 2004). Whilst this has been found to be a valid and reliable method of pain induction (Ervilha et al., 2004), there are associated limitations to studying pain in this way. Firstly, there is little control over duration of pain stimulus, meaning that it becomes ethically sensitive and limits the opportunity for within session repeat testing. It is also very difficult to control for intensity of pain induced which limits investigations to pain versus no pain comparisons rather than the more complex issue of examining the relationship between levels of pain and performance. The current research used a GPD to induce pain which allowed for careful control of pain intensity, was transient in nature and non-invasive, and therefore did not interfere with muscle physiology.

2.3 THEORIES OF PAIN

To fully understand the effects that pain has on the body and how this may impact upon sports performance, an appreciation of the different theories of how pain is experienced by individuals may be valuable. In 1664 Descartes proposed a direct wire theory of pain in which he contended that any external stimuli could only be acknowledged by an individual via messages being sent from the periphery to the brain. Until the mid-eighteenth century all sensations were deemed to be characterised in this way, not accounting for differences in sight, touch and sound which were later identified (Melzack & Wall, 1988).

The major development in differentiation of sensations came via the first scientific proposition of specific nerve energies, presented by Müller (1842), in which it was suggested that external objects were only identified and interpreted by the brain due to sensory nerves which contained symbolic or coded data describing the sensation (Pearce 2005). Müller (1842) proposed that the type of sensation was different for every stimulus and was a function of the nerve, not the stimulus; pain was therefore the function of a unitary sensory system. This was considered to encompass all somatosensory information (including pain) which was thought to be transmitted by a straightforward channel from the pain organ to a pain centre in the brain. This later became known as the specificity theory (Pearce 2005).

2.3.1 SPECIFICITY THEORY

The specificity theory assumes that there is a single sense of touch which encompasses within it the sensation of pain. However this is an issue of contention as research conducted by Von Frey (1894) categorised this into four distinct modalities (touch, warmth, cold and pain) each with its own projection system to the brain. Von Frey also suggested that the skin contained different 'spots' responsible for each of the four modalities and through development of pin prick and touch sensitivity measures (Von Frey's Hairs) and advances in anatomical investigations, he was able to demonstrate the existence of free nerve endings in the skin which he argued to be pain receptors. However his research was based upon many assumptions, and subsequent investigations into the anatomical correlations described by Von Frey have not supported his theories (Melzack & Wall, 1962). It did however provide a platform on which to develop more specific research regarding the existence of pain pathways.

Torebjork and colleagues (1970) discovered that nociceptor units do not form single sensory spots on the skin as suggested, but instead have extended receptive fields with free nerve endings found to serve multiple somatosensory modalities (Pearce 2005). Experiments based upon the specificity theory were also carried out to demonstrate that there is a direct relationship between receptor type, fibre size and quality of experience (Rose & Mountcastle, 1959; Sinclair, 1955) with fibre size being thought to be specific to each modality and therefore supporting the idea that specific nerve energy is in existence for the different touch modalities. Research surrounding this particular theory demonstrated that there are more complex processes going on with regard to how we experience pain and suggested that through identification of a pain pathway there may be the possibility of mediating the pain experience (Rose & Mountcastle, 1959) however the precise mechanisms, especially in relation to neuromuscular performance required further investigation.

2.3.2 PATTERN THEORY

The pattern theory, developed by Weddell et al. (1948) and Sinclair (1955) suggests that pain is a product of intense stimulation of non-specific receptors (Pearce 2005). In contrast to the specificity theory which postulates that pain is interpreted as "painful" as a result of the sensory nerves that describe the sensation (Muller, 1842), the pattern theory suggests that pain perception is a result of the spatial and temporal patterns of the stimulus itself (Noordenbos, 1959). This would mean that the way in which we experience pain could not be mediated through psychological intervention or increased exposure (and therefore desensitisation) to pain, but instead the pain stimulus itself would have to be altered in order to maintain normal neuromuscular functioning. This would require either a change of the stimulus itself (e.g. remove pain from the condition) which is not possible in the case of sports performance, or would require a sophisticated training mechanism in which the way the body interprets the pain stimulus could be altered. This has however received little empirical support and has been usurped by subsequent theories meaning that minimal investigations have been carried out. It is however worth noting that the physical stimulus itself does remain a key factor in any performance reductions and physiological variables should be considered throughout any work in this field (Noordenbos, 1959).

2.3.3 AFFECT THEORY

The Affect theory was the first to centre upon the psychological element associated with pain and the main advocate of this was Marshall (1894) who believed that pain was not simply a sensory quality but that it also had a negative affective quality which could compel an individual to perform an action, or alter behaviour, subconsciously. Physiological theories focus upon pain as the primary sensation and relegate motivational or emotional elements as secondary processes (Wall, 1978). However, the argument could be made that sensory, motivational and cognitive processes occur in parallel and are therefore able to interact with one another to determine the performance output (Melzack & Wall, 1988). This was the first group of theories to consider an interdisciplinary approach to the problem and was subsequently developed by Melzack and Wall (1965) who tried to bring together all the components of what they believe to be a complex mechanism leading to the pain experience.

2.3.4 GATE CONTROL THEORY

The premise of the Gate Control Theory (GCT) is that there is no clinical evidence to support a direct wire approach to pain transmission (Melzack & Wall, 1965). A series of interconnecting nerves transmit a message from the site of the stimulus to the brain, and the junction between

each serves as an opportunity for modulation of the message to occur (Wall, 1978). The first point of possible modulation is the dorsal horn, located in the spinal cord which is where stimuli from the periphery are first sent before being redirected to the brain. The dorsal horn is the central point for understanding the GCT as described by Melzack & Wall (1956) but the authors themselves acknowledge that the pain pathway is complex and indirect and cannot be simplified without consideration of situation and individual.

GCT uses the metaphor of a gate which controls the level of pain an individual feels (Melzack, 1993) within which pain is thought to be modulated on at least three levels (peripheral, spinal and supra-spinal sites) and is conceptualised as a dynamic, interlocking series of biological reactive mechanisms (Wall, 1995). In order for a nociception to be interpreted as pain it must first reach the brain which requires it to pass from the peripheral nerves, into the spinal cord (via the dorsal horn) and then up into the brain. The dorsal horn (or 'gate') modulates the number of impulses passing to the brain, thereby limiting and controlling pain perception (Melzack & Wall, 1965). When the amount of information that passes through the gate exceeds a critical level, the neural mechanisms for the pain experience and control are activated. The gate is assumed to be closed by activity in the large fibres which then decreases the effectiveness of the excitatory synapses and the experience of pain is reduced (Kolt & Mackler, 2003). Whenever a painful stimulus is detected it is transmitted via both large and small afferent fibres, but upon reaching the dorsal horn the neural signals travelling via the rapid conducting large fibres interact with those transmitted via the slower Aδ and C-fibres and the result can be a suppressing of the activity conveying noxious information.

Volleys of impulses from large fibres reach and excite the T-cells first; later the effect is reduced by negative feedback through the substantia gelatinosa (SG). It is the gelatinosa cells of Laminae II and III and the transmission cells of laminae IV and V that are excited. In the small fibres, volleys of impulses activate positive feedback in the SG which exaggerates their effect on the T-cells. In turn the gelatinosa cells when excited by the small fibres can tonically inhibit the input from both large and small fibres by presynaptic inhibition (Melzack & Wall, 1965). Small fibres tend to be tonically active which holds the gate open. When C fibre activity is high there is a reduced tendency for gelatinosa cells to exert their presynaptic inhibitory influences on the T-cells which means that the excitatory effect of the Aδ and C fibres results in opening the gate so that the T-cells can fire (Melzack & Wall, 1965; Noodenbos, 1959). When a noxious stimulus is detected it activates all fibres which alters the balance between large and small fibre input (Wall, 1967). There is ongoing activation of the small fibres which in effect holds the gate open as explained above. When a noxious stimulus is present, there is a disproportionate activation of the large fibres as compared with small fibres (as these were previously largely inactive)

which leads to an increase in presynaptic inhibitory influences exerted by the gelatinosa cells to suppress and modulate the activity of T-cells; this then closes the gate.

The GCT suggests that transmission of pain from the peripheral nerve to the spinal cord is subject to modulation by both intrinsic neurones and controls emanating from the brain (Dickensen, 2002). As every synapse allows opportunity for modulation this means that at any given point between the peripheral site at which the stimulus was detected, right up until it is translated in the brain, there can be any number of factors that can influence it. Cognitive processes are thought to function alongside affective, motivational and evaluative factors to determine somatic inputs (Melzack & Wall, 1965). Through its focus on the central nervous system and the dorsal horn in particular, this theory allowed for innovative developments in pain medicine due to the physiological identification of possible pain pathways (DeLeo, 2006) and the functioning of the gate is now widely accepted as being a factor determining pain perception (Addison et al., 1998).

2.3.5 PARALLEL PROCESSING MODEL

The parallel processing model builds upon both the sensory model for pain and the affect theory to try to explain how the two components work together to produce the final interpretation of pain (Addison et al., 1998). It contends that pain contains both an informational and emotional/motivational component; both of which are processed concurrently. Similar to the GCT information is thought to travel to the brain via the dorsal horn, however in the parallel processing model it is suggested that information about pain location, duration and intensity is sent to the brain parallel to sensory perceptual information about the pain (Addison et al., 1998). At a pre-conscious level therefore, there is a complex network of information being processed regarding both the physical nature of the pain and emotional information alongside it.

The two components of pain proposed within the parallel processing model become increasingly intertwined as the information begins to move from the present (perceptual motor information) to being stored as pain schemata and then finally to where it is stored as informational schemata of past experiences (Addison et al., 1998). The parallel processing theory contends that based upon historical experiences of pain an individual develops schemata that contain both informational and emotional aspects of the pain experience and therefore whenever pain occurs the individual cross references the experience to their existing schemata and the experience of pain that they have will be determined by which aspects of schemata they activate (Taylor & Taylor, 1998). Leventhal & Everhart (1979) found that it was the aspect of pain attended to that determined the pain experience for an individual; for example when an

individual focuses on informational elements of pain they experience significantly less pain than when their attention is directed towards emotional aspects of pain. This has direct consequences for practitioners working within sport, especially sport psychologists, who can help to train the performer to attend to helpful rather than detrimental cues (Meichenbaum, 1977; Nideffer, 1992; Zinsser et al., 2001).

2.3.6 NEUROMATRIX

The GCT highlighted the role of the central nervous system and the dorsal horn in modulating pain, however it is unable to explain phenomena such as phantom limb syndrome where the individual suffers pain from a limb that has been amputated or never existed. In this case there is no peripheral sensation that can be attributed as the cause for the perception of pain as there is no limb (or any peripheral nerves) able to transmit such signals. This suggests that the mechanism for pain perception is not simply due to inputs received, but instead involves further involvement of the brain and sensory system. The Neuromatrix (Melzack, 2001) suggests that pain is an integrative experience which is influenced by many different factors. Within the brain itself there are millions of neural connections which result in a multitude of electrophysiological activities, ultimately leading to a pain perception. Melzack (2001) proposed the existence of a neuromatrix comprising of a series of neural networks, which themselves consist of loops between the thalamus and cortex as well as the limbic system. This network is thought to be initially determined genetically and is then later shaped by sensory inputs to take its complete form.

In contrast to previous theories (Von Frey, 1894; Torebjork et al., 1970) that it is purely sensory inputs that determine the pain sensation, the neuromatrix contends that the loops allow parallel processing within which the cyclical processing and synthesis of nerve impulses develop a characteristic pattern; labelled the neurosignature (Melzack, 2001). The neurosignature is determined by multiple influences and past experiences, not just somatic sensory input. The theory holds that all inputs into the sensory system undergo cyclical processing and the neurosignature is imparted upon them in the neuromatrix to determine the eventual output pattern. This then informs future responses to the same stimulus, so is a data bank of stored information allowing an individual to react in an appropriate way based upon the environment, their own genetic makeup and previous experiences.

The group of theories discussed to this point aim to explain how a pain sensation is recognised by an individual, but they do not account for the way in which muscle performance is altered when pain is experienced which could account for any performance deficits observed during pain conditions (Lund et al., 1991). There have been three major theories proposed to

explain the effect of pain on muscle performance which form the basis for most empirical investigations surrounding pain and performance to date (Hodges & Tucker, 2011; Lund et al., 1991; Roland, 1986). These theories move beyond attempting to identify a pain pathway and seek to understand the interactions between pain and normal neurophysiological functioning to provide an insight into the mechanisms that may cause performance to change.

2.3.7 VICIOUS CYCLE THEORY

The vicious cycle theory contends that muscle activity increases in a uniform manner when pain is present independently of task (Roland, 1986), which has been shown through increased muscle activity and spindle discharge rate (Cram & Steger, 1983). However in the case of sustained activity, ischaemia is a likely outcome as well as accumulation of algesic agents leading to pain, although findings are inconsistent (Lund et al., 1991). Pain induced experimentally has been shown to increase (Del Santo et al., 2007; Sessle, 1999), decrease (Del Santo et al., 2007; Farina et al., 2005) or maintain (Farina et al., 2004; Matre et al., 1999; Schulte et al., 2004) muscle activity during task completion. However, there is a wealth of evidence to suggest that muscle activity (recorded using electromyography) decreases in the presence of pain during a range of tasks (Svensson et al., 1995); isometric (Schulte et al., 2004), dynamic (Arendt-Nielsen et al., 1996) and where task complexity is the primary performance measure (Graven-Nielsen et al., 1997) suggesting that there may not be a uniform increase in muscular activity as suggested by the vicious cycle theory.

2.3.8 PAIN ADAPTATION MODEL

As with the vicious cycle theory, the pain adaptation model (Lund et al., 1991) makes the underlying assumption that there is a uniform response to pain; in this case inhibition of motor drive to muscles experiencing pain or involved in painful movement (Hodges & Tucker, 2011). In conflict to the vicious cycle theory, the pain adaptation model suggests that agonist and antagonist muscles react to limit movement during pain. Agonists reduce activity whilst antagonists increase activity as a means of slowing or stopping the movement being undertaken and thereby limiting potential damage from occurring (Lund et al., 1991). Whilst this is supported through experimental pain research (Arendt-Nielsen et al., 1996; Stohler et al., 1988) there is also evidence to the contrary (Sessle, 1999) where no uniform response has been detected.

The mechanisms underpinning pain-related reductions in muscular performance have been suggested to include information processing components (Price, 2000) which may mean that information being processed concurrently (such as task demands) are reduced in priority

in the presence of pain; however exact mechanisms by which this may occur remain unknown. Muscle afferents identified to be sensitive to nociceptive stimuli are the group III and IV afferents (Falla and Farina 2008) which can be experimentally stimulated in order to allow for study of pain responses. In animal studies both excitatory and inhibitory postsynaptic potentials have been recorded in motoneuron membrane properties as a response to input from group III and IV nociceptive muscle afferents (Kniffki et al., 1981) supporting the existence of changes as suggested by the pain adaptation model. In human studies there have been inconsistent findings related to the pain adaptation model (Hodges et al., 2003). Patterns of electromypgraphic (EMG) activity relating to the model have been supported in a number of studies (Arendt-Nielsen et al., 1996; Stohler et al., 1988; Svensson et al., 1997; Graven-Nielsen et al., 1997), however others have found that whilst a reduction in agonist EMG activity is commonly found to occur, there is not always an increase in antagonist EMG activity, rather, there is a similar reduction in antagonist EMG activity detected (Ervilha et al., 2004(Birch, Christensen et al. 2000; Falla, Farina et al. 2007).

Challenges to the pain adaptation model come as a result of changes observed in motor unit discharge behaviour (Tucker, Butler et al. 2009), which has been found to decrease during force matched contractions in the presence of pain (Sohn, Graven-Nielsen et al. 2000; Ervilha, Farina et al. 2005) despite the fact that discharge rate is directly related to force production (Stuart and Enoka 1983) and therefore there should be a corresponding decrease in force which was not evident. When investigating force maintenance under painful conditions most studies have found a decrease in motor unit discharge rate where force is maintained (Sohn, Graven-Nielsen et al. 2000; Farina, Arendt-Nielsen et al. 2005; Hodges, Ervilha et al. 2008). However, equivocal data exist where Birch et al. (2000) reported no change in discharge rate looking at the whole motor pool, which was explained by recruitment of new motor units and increasing discharge rates in units not measured (Tucker et al., 2009). Given the equivocal reports in the literature and inconsistent changes in antagonist activity identified that could account for force maintenance (Tucker, Larsson et al. 2009), the findings related to uniform inhibition of painful muscles do not consistently support the pain adaptation model (Tucker, Butler et al. 2009).

Some aspects of the pain adaptation model have been identified to occur consistently between individuals (Hodges et al., 1996, 2003; MacDonald et al., 2009), whilst in other muscles there appears to be unique response unaccounted for by the model (Hodges et al., 1996; van Dieen & Selen., 2003) most commonly found to occur in the trunk which comprises of numerous muscles all able to achieve similar goals and therefore able to experience redundancy during movement (Hodges et al., 2003; van Dieen & Selen, 2003). This inter-individual variability is not accounted for within the pain adaptation model (Hodges & Tucker, 2011). Similarly, the

pain adaptation model ignores changes in postural control and makes predictions purely based on voluntary movements (Hodges & Tucker, 2011) which is based on the assumption that pain does not cause postural changes (Lund et al., 1991). However, literature increasingly refutes this with evidence that pain alters balance (Byle & Sinnott, 1991; Mok et al., 2004) as well as changes to whole muscle behaviour in postural mechanisms during pain (Hodges et al., 1996, 2003; MacDonald et al., 2010; Magnusson et al., 1996). This would provide rationale for assuming that pain does change the ability of an individual to control muscular functioning and hence may suggest that performance may be increased through training or interventions.

2.3.9 ADAPTATION TO PAIN MODEL

Hodges & Tucker (2011) suggest the 'adaptation to pain' theory in which pain is thought to stimulate a protective mechanism to minimise risk to the individual in a similar way to the pain adaptation model proposed by Lund et al. (1991). However, rather than suggesting one uniform response to pain, they propose a more flexible solution involving redistribution of muscle activity both within and between muscles, changes in normal mechanical behaviour and changes at multiple levels of the motor system (Hodges and Tucker) all of which are dependent on task and muscle region. Hodges & Tucker (2011) propose that when pain is present there is a redistribution of activity which may occur in conjunction with a change in recruitment order, thereby explaining decreased motoneuron discharge rate (Sohn, Graven-Nielsen et al. 2000; Farina, Arendt-Nielsen et al. 2004; Hodges, Ervilha et al. 2008). Alternatively a change in recruitment order may be presumed to occur incorrectly due to the fact that larger motor units may be recruited at a lower force (to enhance the rate of force development as part of the flight/fight response), or in a different direction (to preferentially activate muscle fibres with a specific force direction to change load distribution on the painful structure) (Hodges & Tucker, 2011).

There are inconsistencies with studies reporting recruitment patterns under conditions of pain which have been explained by Hodges & Tucker (2011), for example, spatial redistribution is difficult to detect by a single pair of surface electrodes placed over the whole muscle which could account for the variability reported between studies (Arendt-Nielsen 1996; Birch, Christensen et al. 2000; Schulte, Ciubotariu et al. 2004; Farina, Arendt-Nielsen et al. 2005; Falla, Farina et al. 2007). There is evidence to show that discharge rate can be maintained during pain thought to be through recruitment of a new population of units that were not active before pain which could not occur with uniform inhibition of the whole motoneuron pool (Tucker, Butler et al. 2009; Tucker and Hodges 2009). This theory advocates a non-stereotypical response to pain where the nervous system is thought to have a range of options

at its disposal in order to achieve protection of the painful area. This may involve increased, decreased or redistributed activity involving more complex neural processes than those proposed by the existing theories (Hodges and Tucker) but which are currently unexplained and require further investigation. It is not clear what the mechanism is that causes there to be a reduction in muscular performance when pain is present despite the wealth of theories proposed.

2.3.10 SUMMARY OF THEORIES TO DATE

The theories discussed above explore the issue of pain from a number of theoretical standpoints and with different disciplines underpinning their explanations. The early theories focus upon physiological explanations of pain and how the individual understands the physical sensation of pain as 'painful' with the central premise of a 'pain pathway' (Weddell et al., 1948; Von Frey, 1894). Specificity and Pattern theories have since been criticised for their limited acknowledgement of the interaction between the environment and the individual (Marshall, 1894; Melzack, 2001) and the fact that advances in physiological techniques have found no direct wire (or pain pathway) to exist (Melzack & Wall, 1965), indicating that other mechanisms are likely responsible. This was counteracted by a proposal in which affective constructs were also thought to contribute to the pain experience and therefore the inclusion of psychology was perceived important for advocates of the affect theory (Marshall, 1894) and parallel processing model (Addison et al., 1998). The psychological elements of pain were believed to be equally important to the sensation experienced as a result of the physical stimulus. These theories moved away from trying to provide a simple direct wire explanation for pain and moved towards a more complex relationship between physical and psychological constructs; however they still referred to the premise of a pain pathway for which no empirical evidence has been provided (Melzack & Wall, 1965).

The GCT proposed by Melzack & Wall (1965) considered pain to be more than a simple pathway from stimulus to response and recognised that ascending and descending signals can interact to mediate the pain experienced. However, this theory has also received criticism due to its diagrammatic emphasis in which the role of the substantia gelantinosa and presynaptic mechanisms were highlighted with little consideration of other variables (Nathan, 1976), and as with many of the pain theories, it tends to concentrate on mechanism rather than outcome without any solid evidence to suggest that the process outlined does describe the phenomenon. In contrast, the neuromatrix (Melzack, 2001) did not focus on a single pathway, but instead suggested the large number of opportunities for modulation to occur in which modulation occurs and how this cannot be viewed distinct from the environment and individual factors.

However, there is still no definitive single mechanism for the production of pain experiences. Whilst this remains a question yet to be answered by any of the theories proposed to date, there are commonalities amongst the later theories suggesting that some level of agreement has been reached (Melzack, 2001) for example that modulation can occur to the stimulus during transmission (Melzack, 1978; Melzack & Wall, 1965; Noordenbos, 1959; Roland, 1986) and multiple factors make up the pain experience rather than a simple pain pathway (Addison et al., 1998).

The second group of theories presented here focused upon the effect of pain on human movement and postulated reasons for behavior change observed when an individual experiences pain. The vicious cycle theory (Roland, 1986), pain adaptation model (Lund et al., 1991) and adaptation to pain theory (Hodges & Tucker, 2011) all agree that pain is accountable for certain changes in muscular performance. However, the vicious cycle theory suggests a uniform increase in muscle activity in the presence of pain whilst the pain adaptation model argues against this and instead proposes that there is a uniform reduction in muscle activity of the agonist and increase in antagonist muscle activity in response to pain. Both theories can be supported through empirical evidence where muscle activity has been shown to react in the way hypothesised by the authors (Roland, 1986; Lund et al., 1991), however neither has been able to predict accurately the way in which an individual responds to pain in all situations, nor provide an explanation for why there is no consistent uniform response evident (Hodges & Tucker, 2011). For this reason the adaptation to pain model (Hodges & Tucker, 2011) does not propose a uniform prediction for the response to pain and instead suggests a non-stereotypical response to pain which is dependent on location, type and situation. This accounts for some of the discrepancies found between the vicious cycle and pain adaptation model, however it still does not provide a clear explanation of mechanisms involved and requires further development before it can be used as a predictive tool. It is evident that further investigations into the mechanisms underpinning the pain experience are required before a conclusive explanation can be given, and with the introduction of emerging technologies new techniques may be developed that allow for this to be achieved.

2.4 NEUROMUSCULAR PHYSIOLOGY

In order to understand the effect of pain on muscle performance, an appreciation of how the neuromuscular system functions normally is also needed so that comparisons can be drawn and possible mechanistic changes can be investigated. It is also important to develop an understanding for the mechanisms of pain and how a pain response is generated to understand how this may interact with the normal functioning of the neuromuscular system and provide an

explanation for any differences observed. The following section discusses some of the key physiological processes involved in muscle performance and how this contributes toward the pain experience.

2.4.1 TRANSMISSION OF STIMULI TO THE BRAIN

Sensation, transmission and perception of pain are a function of the nociceptive system. When a stimulus is detected in the periphery a message is sent to the brain as nociception; it is only when the message reaches the brain that it may be interpreted or defined as pain (Melzack & Wall, 1965). The process of a message reaching the brain can be divided into four phases; transduction, transmission, modulation and perception (Heil & Fine, 1999). At the nerve endings a stimuli is detected (which may be pressure, heat, damage or any number of stimuli) this is then translated into electrical activity to allow it to be transmitted through the sensory nervous system. This change from mechanical to electrical activity is labeled the process of transduction. Once the message is electrical, the impulses are transmitted through the sensory nervous system to the brain. At any point between the area at which the stimulus was detected and the brain, there is the opportunity for modulation to occur where the message is transmitted from one nerve axon to the next (Melzack, 2000). At the synapse there is opportunity for the message to be changed through central, cortical or peripheral influences (Melzack & Wall, 1965). Once the signal reaches the brain it can be given meaning, which is known as the process of perception; it is at this point that the cognitive-emotional experience of pain is generated (Heil & Fine, 1999).

2.4.2 AFFERENT NERVE FIBRES

The sensory nervous system contains three types of afferent nerve fibre; A-Beta (Aβ), A-Delta (Aδ) and C fibres; all of which are responsible for detecting and transmitting stimuli to the brain (Melzack & Wall, 1965). Each has a different threshold at which point it is stimulated and they are classified by size and suffix. The Aδ fibres and C-fibres are thought to be those responsible for transmission of noxious stimuli; however the types of stimuli they transmit differs due to the physiological makeup of the fibres (Wall, 1978). Aβ and Aδ fibres are both myelinated which allows for faster neural transmission, however the larger diameter of the Aβ fibres means that they are much faster than the Aδ fibres at transmitting electrical impulses (Devor & Wall, 1978). Sensations such as touch and proprioception stimulate the Aβ fibres whilst heat and pinprick type sensations stimulate the Aδ fibres which connect to high threshold mechanoreceptors. The major physiological difference between A and C fibres is that the C-fibres are unmyelinated meaning that neural transmission is slower. C-Fibres have a small diameter and are responsible

for the transmission of ache type pain stimuli (Torebjork & Hallin, 1974). These fibres connect to polymodal nociceptors.

Nociceptors have a threshold too high to be stimulated by normal innocuous stimuli; their role is to detect noxious stimuli which generate a higher level of excitation (Charman, 1994). Aβ fibres detect these high threshold stimuli and transmit the information very quickly through the nociceptive system which ensures a rapid and non-conscious response (Eccles, 1964). This quick response to pain is what informed Decartes (1664) first major theory to pain mechanisms in which it was believed that pain transmission occurred through a single channel from the periphery to the brain triggering a reflex response. However clinically it has been demonstrated that humans do not have a direct wire linking the skin to the brain; each neural transmission has to pass through a number of nerves before reaching the brain (Melzack & Wall, 1965). Every synapse between nerve axons allows opportunity for modulation to occur at which point the meaning or interpretation of the stimulus may be altered (Melzack, 2000).

Pain is thought to activate large (Aβ and Aδ) and small fibres (Aδ and C-fibres) simultaneously (Noordenbos, 1959). The Aβ fibres ascend directly in the dorsal columns allowing for a quick response. The Aδ and C-fibres terminate in the dorsal horn, located in the spinal cord. Here the stimuli synapse with interneurones within the dorsal horn, predominantly in Lissauer's Tract but also deeper in the SG. At this point there is opportunity for the simultaneous messages being sent by the large and small fibres to interact with one another and even inhibit the signal that each is sending which then alters the message that is received by the brain (Melzack & Wall, 1965). This is important to note as during sports performance the individual may experience pain and this could mediate the normal functioning of the neuromuscular system thereby inhibiting performance (Scott & Gijsbers, 1981). Alternatively, it could provide valuable opportunity to help individuals learn to deal with pain and reduce the magnitude of effect it has on performance which could inform practitioners working in performance environments.

2.4.3 MUSCLE PERFORMANCE

The major function of a muscle is to produce force in order to facilitate movement (Jing et al., 2002). Force production contains many processes and mechanisms that need to be understood when investigating performance in tasks where force is an outcome measure. This includes identification of how the muscle contracts and the biomechanical relationships concerning force production; the length-tension relationship and the force-velocity relationship. When movement is required a message is sent from the brain via the central nervous system (CNS) and triggers muscle contraction. This happens through a series of events described within the

sliding filament theory (Huxley et al., 1953) for which muscle structure plays an important role. Several important functional components can be identified in Figure 2.1.

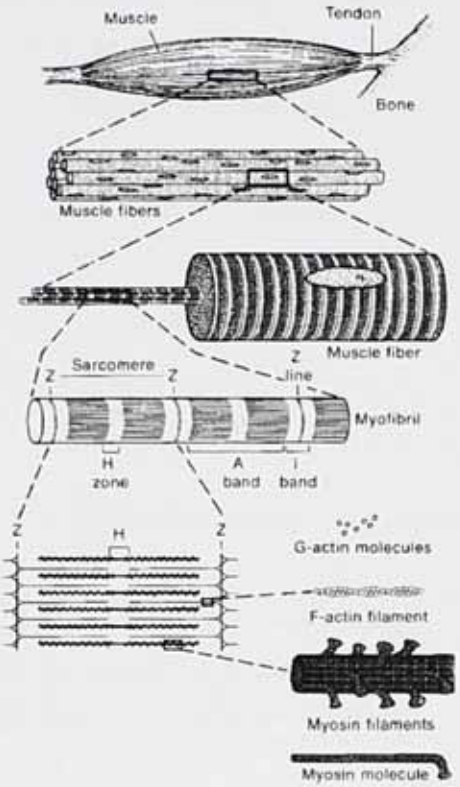


Figure 2.1. Hierarchical organization of muscle (adapted from Bloom & Fawcett, 1968).

The range of force a muscle can produced is largely determined by how many sarcomeres there are in a series and the magnitude of the force a muscle is able to produce is determined by the number of muscle fibres arranged in parallel (Meijer et al., 1998). These muscle fibres are organised into motor units in which all muscle fibres are of the same type and have the same metabolic profile, so that when they are activated they all behave in the same manner (Liu et al., 2002). In order to initiate voluntary movement command signals must be sent from the CNS via efferent nerves to the motor units. As the command is transmitted down the spinal cord there is opportunity for modulation as a result of sensory feedback sent via afferent neural fibres from the mechanoreceptors, muscles and soft tissue which may inhibit or potentiate the activation of the particular motoneurons (Enoka, 2008).

If the stimulus that reaches the neuromuscular junction exceeds the threshold required for that muscle, it will trigger action potentials of the motor units and all the muscle fibres of that motor unit will contract simultaneously (Ganong, 1971). According to Ohms law (I = V/R)

the rheobase current (I) is determined by the required voltage change to produce an action potential (V) and the input resistance of a motoneuron (R). As resting potential and the voltage at which an action potential is generated do not tend to change, it suggests that input resistance is the major determinant of activation (Gardiner, 2001). This is thought to be inversely proportional to motoneuron size (MacIntosh et al., 2006), so smaller motoneurones have a lower activation threshold as a smaller rheobase current is required to generate the action potential (Henneman et al., 1965). In order to generate more or less force, the number of motor units recruited must change; movements requiring low force recruit only few motor units, those requiring high levels of force recruit greater numbers (McArdle et al., 1996). If one considers that the signal to initiate movement can be mediated as suggested by the GCT and Neuromatrix theories then this is a point at which it may be proposed pain could alter performance of the muscle as a result of modulation from either the pain stimulus itself or the resulting experience (Melzack & Wall, 1965; Melzack, 2000).

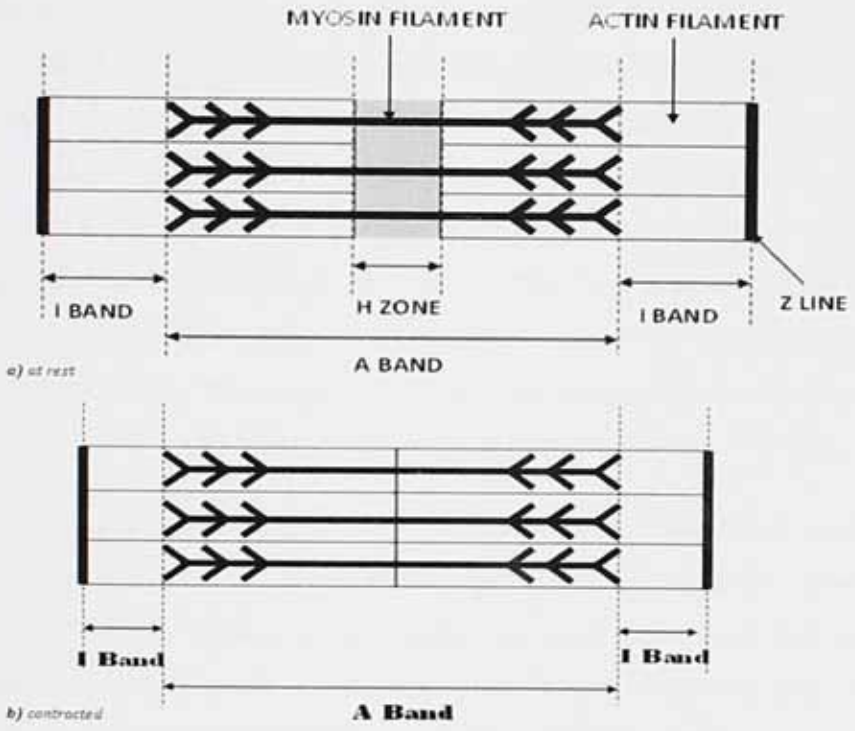


Figure 2.2. A simplified diagram of a single sarcomere within a myofibril a) at rest and b) contracted illustrating the process of the Sliding Filament Theory (Huxley et al., 1953).

Figure 2.2 depicts a sarcomere made up of myofibrils containing both actin and myosin filaments organized in a lattice formation (Horowitz & Podolski, 1987a). At rest, the thick myosin filament overlaps the thin actin filaments leaving a gap between the actin filaments known as the H Zone (Figure 2.2a). During contraction cross-bridges form and thereby cause the myosin and actin filaments to overlap, shortening overall muscle length without any of the

myofibrils themselves shortening (Figure 2.2b). When a muscle fibre is contracted, all sarcomeres contract simultaneously so the contraction is uniform across the sarcomeres (Horowitz & Podolski, 1987a). This suggests that if an impulse is sent by the brain for a muscle to contract, it will contract unless the message does not reach the intended recipient(s). Modulation of the message could occur to prevent signals reaching all motor units within the muscle group which would therefore decrease force output and hence limit performance.

The mechanism for muscle contraction is stable and relies on certain chemical changes allowing cross-bridges to form and ATP to bind to the free binding sites (Horowitz & Podolski, 1987b; Huxley, 1953). This happens whenever a movement is desired as a consequence of neural stimulation. However, level of force produced is determined by numerous factors, such as level of agonist activation (de Ruiter et al., 2004, 2006, 2007), intrinsic contractile properties of the muscle (Andersen et al., 2006), type of contraction (Hill, 1938) and muscle length and joint angle (Rassier et al., 1999). An inverted-U relationship has been found between length of the muscle and tension produced, with the greatest force evident at mid-range of motion in the contractile elements. At shorter lengths than resting length, the actin and myosin cross bridges are thought to interfere with one another meaning reduced force production (Abernethy et al., 1992). At lengths greater than resting rate there are not enough binding sites, meaning that muscle tension cannot be produced. In contrast, the elastic elements have no influence on force production until the muscle length is greater than resting length. When this occurs, the elastic fibres stretch and produce tension in the muscle. The total length-tension relationship is the sum of the two. Isometric tasks provide a situation in which the muscle length and joint angle remain constant and therefore allows for neural and mechanical determinants of force to be investigated more reliably which is why this type of task was selected for the current study.

The length and direction of movement also influences force production which is depicted as a hyperbolic curve describing the dependence of force on velocity of movement; implying that a) velocity of movement is inversely proportional to load, b) large force cannot be exerted in rapid movements, and c) conditions of low loading create the greatest velocities (Hill, 1953). The force-velocity relationship makes the assumption that at a given velocity, the muscles are generating maximum force (Beck et al., 2008) with the likely source of the relationship being the fact that when speed of cross-bridge cycling increases, there are fewer cross bridges formed to develop force (Gulch, 1994) thereby reducing output over a given velocity. In studies in which pain has been induced by injection of saline into the muscle (Arendt-Nielsen & Graven-Nielsen, 2008; Ervilha et al., 2004; Farina et al., 2004), the physiological properties of the muscle may be compromised and therefore alter processes involved in contraction. Within the current work, the use of a gross pressure device which is

non-invasive and induces pain remote to the target muscle ensures that any changes observed are as a consequent of the pain rather than physiological interference with normal processing.

Muscle performance in sport can take many forms, be that power output, force production or muscular endurance. However, within this study a task that would be relatively novel to all participants and would allow the controlled study of both force output and muscle activity was chosen. EMG can be used to capture and measure electrical activity produced by skeletal muscles during movement through detecting the action potential that is generated by those muscle cells when they are neurologically activated (MacIntosh et al., 2006; Robertson, 2004). Amplitude of EMG signal increases with increased motor unit recruitment and firing frequency, making it a useful measure of neural activation (De Luca, 1997; Farina et al., 2002) provided that methodological and physiological factors that can alter the signal are accounted for, such as crosstalk from other muscles, amount of subcutaneous tissue, blood flow, electrode placement (De Luca, 1997) and equipment noise (Turker, 1993). In small muscles the relationship between EMG and force tends to be linear, whilst bigger muscles that require greater motor recruitment tend not to demonstrate such a linear relationship as amplitude variations do not correspond directly to force variations (Basmajian et al., 2004). However a positive curvilinear relationship exists which is thought to support the use of EMG as a global indicator of neural activation (Alkner et al., 2000; Kooistra et al., 2007).

There are two types of EMG analysis; surface and intramuscular (fine wire), which can be used for diagnostic purposes (Nigg et al., 1999) and to assess tension developed in muscle (Petrofsky & Laymon, 2005) or the degree of fatigue during exercise (Gerdle et al., 1990; Basmajian & De Luca, 2004). Surface EMG records the sum of the electrical contributions made by active motor units which are detected by two electrodes placed over the muscle belly and is considered to be a global measure of motor unit activity (Farina et al., 2004). Intramuscular EMG can measure the activation timing and firing rate of individual motor units, and due to the fact that the electrode is inserted directly into the muscle belly being measured makes this suitable for muscle located deep within the body (Turker, 1993). However, it is inaccurate during strong contractions when impulse patterns become too dense (MacIntosh et al., 2006) and limited to measuring only one motor unit therefore for larger muscle groups surface EMG is more appropriate.

When a muscle starts to fatigue the power produced by the individual muscle fibres is reduced (Fitts & Holloszy, 1976) which is reflected in reduced EMG signal recorded. However, a number of factors have been identified to alter EMG amplitude and frequency including proportion of fibre types (fast or slow) (Petrofsky, 2001), thickness of subcutaneous fat

(Bilodeau et al., 1990) and placement of electrodes (Gerdle et al., 1990, 2001) which all need to be taken into account when selecting location of measurement site and task performed. This study used surface EMG to analyse muscular activity during an isometric task as it allowed for investigation of changes to muscle electric potential and muscle synergies in specific movement patterns (Farina & Merletti, 2000). The initial signal that is recorded is the collective action potential of the muscle fibres in the motor unit being measured, all of which work together as they are innervated by the same motor neuron (Basmajian & De Luca, 2004).

When analysing EMG recordings it is common to use the root mean square (RMS) value which quantifies the electrical signal through reflecting physiological activity in the motor unit during a contraction (De Luca, 1997) and applying mathematical treatments that quantify the intensity and duration of several events of the EMG signal (Farina & Merletti, 2000). As there are so many factors that can influence EMG signal between individuals, it is common for EMG signal to be normalised to maximum isometric contraction (Burden et al., 1999; Merletti et al., 1999; Kaplanis et al., 2000) in order that comparison can be made across participants. Using this technique, coupled with data about the level of force being produced allows for intra-individual differences to be observed which can be used to help explain the influence of pain on muscle performance and be applied to the sport setting.

2.4.4 THE INFLUENCE OF PAIN ON NEUROMUSCULAR FUNCTIONING

In elite sport it is essential that muscle performance is optimal in order to achieve the desired results; therefore any factors compromising this are detrimental to the athlete. Muscle pain has been shown to influence control of movement via numerous reflex and central mechanisms (Arendt-Nielsen and Graven-Nielsen 2008) with experimental pain induced within a muscle shown to reduce activation of the painful muscle (Farina, Arendt-Nielsen et al. 2004; Falla, Farina et al. 2007). This is thought to be reflective of a decreased neural drive from spinal cord to muscle (Sohn, Graven-Nielsen et al. 2000; Farina, Arendt-Nielsen et al. 2004). The inhibition in this case is postulated to be a result of a combination of reflex mechanisms which are mediated by small diameter muscle afferents and reduced supra-spinal drive to the muscle (Falla, Arendt-Nielsen et al. 2009) however the mechanisms for this are yet to be fully identified.

In an investigation of participants with neck and shoulder disorders, researchers observed changes in the activation of the upper trapezius (Arendt-Nielsen and Falla 2009; Falla, Arendt-Nielsen et al. 2009) including reduced muscle activation during a repetitive upper limb task in participants experiencing chronic neck pain (Falla et al., 2004), increased muscle activity during computer work (Szeto, Straker et al. 2005), reduced ability to relax the upper trapezius muscles following voluntary activation (Falla et al., 2004) and reduced rest periods of the upper

trapezius muscle during repetitive tasks (Veiersted, Westgaard et al. 1990). This was investigated further with regard to location of pain stimulus in the trapezius showing that location (caudal, cranial, or both simultaneously) caused the same activation changes, demonstrating that the activation pattern of inhibition was unaltered by site of stimulation (Falla, Arendt-Nielsen et al. 2009) and perhaps indicating a more centralised response to pain.

Experimentally induced pain has also been used to study the effect of pain on muscle performance and the possible mechanisms by which changes occur. In a swimming task inducing ischaemia there was no performance decrement detected in high level swimmers (Scott & Gijsbers, 1981). This contradicted the original hypothesis that pain could take control of an organism and therefore inhibit motor performance in suggesting that those at elite levels were able to perform without any significant inhibition. In contrast, Brewer et al. (1990) found that pressure pain did inhibit motor performance when task complexity was investigated. To explain this, the authors suggested that the pain stimulus induces a state of over arousal, which when considered relative to the inverted-U theory of arousal (Yerkes and Dodson 1908) would provide rationale for performance decrements to occur; for every task there is an optimal level of arousal and values above or below this cause performance to decrease (LeUnes 2008). In the case of pain, it could lead to increases in arousal levels and thereby take the participant outside of the "optimal" performance zone leading to a decrease in task performance. Similar findings were reported by Birch et al. (2000) who concluded that when exposed to pain during low precision tasks, muscle activity would decrease, whereas in high precision tasks there was no effect of pain suggesting that there may be a higher level neurological element able to override the influence of pain when examining task accuracy. The fact that in the Scott & Gijsbers (1981) study there were no decrements found in elite swimmers could be indicative of the fact that high level performers are able to train to deal with the consequences of pain and have therefore developed coping strategies allowing them to maintain performance (Jaremko et al., 1981; Ryan & Foster, 1967) or have reached elite levels as a result of being able to cope with pain (Pen & Fisher, 1994). However, a causal relationship has not been shown to date.

A significant development in the study of pain theories came with the proposition of the pain adaptation model (Lund et al., 1991) which attempted to explain the relationship between pain and muscular performance stating that pain affects muscle activation through inhibition of agonistic muscle and excitation of antagonistic muscle, resulting in a reduced force production and range of motion (Bonifazi et al., 2004). The activation of the agonistic and antagonistic muscle in synchrony with one another, referred to as co-contraction (Hammond et al., 1988) is thought to be a strategy providing a way of adapting the limb to external forces or those arising from multijoint dynamics (Gribble & Ostry, 1998). If a stressor is placed on a limb it is identified

by the body as a potential risk and in reducing muscular force in the agonist and increasing tension in the antagonist, the range of motion reduces, hence limiting the potential damage that can occur.

Patterns of EMG activity relating to the pain adaptation model have been supported in a number of studies (Arendt-Nielsen et al., 1996; Stohler et al., 1988) where pain has been found to decrease EMG activity in the agonist and increase EMG activity of the antagonist within a plantar flexion (Graven-Nielsen et al., 1997) and an elbow flexion (Ervilha et al., 2004) task. However, there is some contradictory evidence to show that whilst a reduction in agonist EMG activity usually occurs there is not always a corresponding increase in antagonist EMG activity, rather a similar reduction in antagonist EMG activity is also detected (Ervilha et al., 2004). Another finding common among studies looking at EMG response and pain is that the level of force produced during a maximum voluntary contraction (MVC) is decreased during both experimentally induced muscle pain and non-experimentally induced muscle pain (Graven-Nielsen et al., 1997; Backman et al., 1988; Suzuki & Endo, 1983).

Whilst EMG activity and force level were found to reduce during pain conditions in studies using MVCs (Graven-Nielsen et al., 1997; Backman et al., 1988; Suzuki & Endo, 1983), studies using sub-maximal contractions found opposing results. Ashton Miller et al. (1990) found that during sub-maximal isometric contractions there were no changes to either EMG activity or force. What was found to change however was the endurance time of participants in the pain condition compared to those in the no pain condition. These findings were later supported by Graven-Nielsen et al. (1997, 2002) who concluded that pain has an inhibitory effect of isometric contractions when the muscle activity rises above 70% of the MVC but not before. In dynamic tasks there is again a change in the findings reported from isometric MVC trials. Within dynamic tasks it is suggested that pain modulates voluntary activation of the muscle either through increasing the EMG activity in phases of contraction where it is normally silent, or through decreasing EMG activity in phases where it is normally activated (Arendt-Nielsen et al., 1996; Graven-Nielsen et al., 1997; Svensson et al., 1998; Zedka et al., 1999) thus not showing a net increase or decrease in EMG activity generally, but instead altering the patterns of contraction.

There is a relatively stable motor unit recruitment order for most behaviours which is based upon factors such as motoneuron size and common drive to the motoneuron pool (Heckman and Binder 1990; Mendell 2005) where it is predicted that recruitment of units extends from smallest to largest (Desmedt and Godaux 1977). However, changes in recruitment order have been identified as occurring as a result of non-physiological stimuli, such as

electrical stimulation (Garnett and Stephens 1981; Semmler and TÃ¼rker 1994), and in some voluntary tasks where muscles have multiple physiologic functions (Thomas, Schmidt et al. 1978; ter Haar Romeny, van der Gon et al. 1982; Riek and Bawa 1992; Butler, McKenzie et al. 1999). More contemporary research centers upon the changes occurring to muscle recruitment patterns in the presence of pain (Tucker and Hodges; Cowan, Bennell et al. 2001; Tucker, Butler et al. 2009) demonstrating that when knee pain is induced experimentally, there is a redistribution of active motor units within the quadriceps muscle which changes the direction of knee extension force a few degrees medial or lateral to that in the pain free trials (Tucker and Hodges). Beyond direction of force exerted, pain is also found to change relative timing of the activation of medial and lateral heads of the quadriceps in a stair stepping exercise with patients with patellofemoral pain (Cowan, Bennell et al. 2001; Hodges, Ervilha et al. 2008) and a reduction in synchronicity of discharge of motoneurons is found to occur within these muscle heads in the presence of anterior knee pain (Mellor and Hodges 2005). It is clear that pain can cause complex adaptations to motor strategy, which in turn can lead to impairment of the generation and control of steady force production thought to be linked to muscle architecture (Salomoni and Graven-Nielsen).

Neural mechanisms for the inhibition of muscle performance linked to pain also need to be considered. Pain is thought to decrease net excitatory drive to the motor neuron (Sohn, Graven-Nielsen et al. 2000; Wang, Arima et al. 2000; Farina, Arendt-Nielsen et al. 2004) and the general pattern is for the initial reduction in drive to take place in motor units that have a higher threshold for activation (Falla, Arendt-Nielsen et al. 2009). De-recruitment of motor neurons with a higher threshold for activation and a lower discharge rate at a given force occurs first via decreased synaptic input, therefore pain is found to induce a change in spatial distribution of muscle activity for muscles characterised by non-uniform spatial recruitment of motor units, such as the upper trapezius (Falla, Arendt-Nielsen et al. 2009). When pain was induced via injection of hypertonic saline within the cranial region of the trapezius muscle there was found to be a greater reduction in EMG amplitude in the cranial (over caudal) region of the muscle during static tasks (Madeleine, Leclerc et al. 2006) which is consistent with the above hypothesis as the cranial region has a higher threshold for activation (Falla, Arendt-Nielsen et al. 2009). This suggests that the neural mechanisms associated to the pain experience are important contributors to any decrements observed. However still no conclusive evidence exists to demonstrate a mechanism for pain and associated performance change in muscles.

The use of surface EMG has provided valuable insight into the activation of muscle groups within a movement (Graven-Nielsen et al., 1997; Backman et al., 1988; Suzuki & Endo, 1983) however cannot give accurate detail about which motor units are activated at certain points.

Fine wire EMG data recordings have been used to show a decrease and changed recruitment in 25% and 40% of quadricep and flexor pollucis longus recording zones measured, with the new units recruited found to be those at the next highest threshold in some cases, but not all the time (Tucker, Butler et al. 2009). These findings are significant as they suggest that it is not the painful region of the muscle that is always most protected, instead there is a more consistent change in motor strategy which prioritises motor units with the highest activation thresholds and therefore inhibits these first (Falla, Arendt-Nielsen et al. 2009). By way of explaining the mechanisms responsible for this Luscher et al. (1979), suggests that inhibitory input to the motoneuron pool will generate larger inhibitory postsynaptic potentials in the smaller motoneurones, thereby overcoming the excitation to those motoneurones first (De Luca 1985). De-recruitment of low threshold motor units would consequently result in higher levels of central drive and a subsequent recruitment of new, higher threshold units to maintain the force that is generated at pre-pain levels (Tucker, Butler et al. 2009). A further possibility is that there is an inhibition of discharge of the smaller low-threshold motor units when larger units are recruited, thereby explaining the change in recruitment pattern (Ross, Cleveland et al. 1975; De Luca 1985). It could then be suggested that pain causes changes to both population of motor units recruited and the order in which they are recruited and this is what could account for performance decrements (Tucker, Butler et al. 2009).

2.5 THE ROLE OF PSYCHOLOGY IN PAIN RESPONSE

As discussed in section 2.4.1, pain is thought to stem from the detection of noxious stimuli which signifies potential risks to the brain allowing the individual to act upon this (Heil & Fine, 1999). There is however, little neurological evidence that noxious stimuli exist (Pen & Fisher, 1994), instead it is suggested that every stimulation is simply carried as an impulse along a certain neural pathway; it is only at the point of recognition or perception that these stimuli become noxious (Melzack, 1973). Pain could therefore be classified as a cortical phenomenon as it is not experienced as pain until it reaches the cortex and is identified as noxious. For this reason it must be studied with physiological and psychological components in mind. There is little literature published with a specific focus on mechanisms underpinning the pain experienced by athletes despite the evidence that there are differences in the way athletes and non-athletes respond to pain (Giesbrecht et al., 2005; Hall & Davies, 1991; Janal et al., 1994; Ryan & Kovacic, 1966; Straub et al., 2003).

There is a 'macho' image associated with sports participation which has been linked to a culture in which 'playing with pain' is widely accepted (Nixon, 1992, 1993) and peer pressure from team mates or the broader sports net can lead to individuals tolerating greater levels of

pain than they would otherwise (Frey, 1991). Social pressure has also been identified to alter pain response where physiological, subjective and social cues investigated were thought to interact to change the verbal reporting of a pain stimulus (Manell, 1980; Sternbach, 1978). Similarly social models may increase the ability of an individual to deal with pain (Craig & Weiss, 1971; Craig & Coren, 1975) so when seeing a significant other deal with a given stimulus an individual may feel pressure to be able to cope equally well with that stimulus and not show weakness in relation to peers. These are all factors suggesting that cognitive processes can alter pain response and are prevalent within the sporting culture. Therefore the psychological element of the experience must be considered carefully. It also demonstrates that it is possible to manipulate the perception of pain through controlling situational factors which suggests it may be possible for applied practitioners to design interventions intended to help athletic performance.

Ryan & Kovacic (1966) suggested that the different pain tolerances identified between athletes and non-athletes may be as a result of the way in which the pain stimulus is perceived. They proposed that the perceptual style of an athlete allowed them to reduce the perceptual stimulation and thereby tolerate greater levels of pain than non-athletes, which was developed further by Petrie (1978) who differentiated perceptual style into three categories; reducers, augmentors and moderators. The way in which the same stimuli are processed from the environment within the three categories differs. Reducers tend to decrease what is perceived, therefore allowing them to tolerate greater levels of pain than moderators. In contrast augmentors tend to amplify the stimuli perceived and therefore are not able to tolerate as much pain as moderators. This is consistent with findings from mainstream psychology where the effects of association and dissociation have been studied (Schomer, 1987), however has not been widely researched within the athletic population. However, Addison and colleagues (1998) attempted to bring together some of the existing research available within mainstream psychology and the more specific traits associated with those involved in high level sport which highlights a number of factors that should be considered within studies in which athletic pain response is investigated.

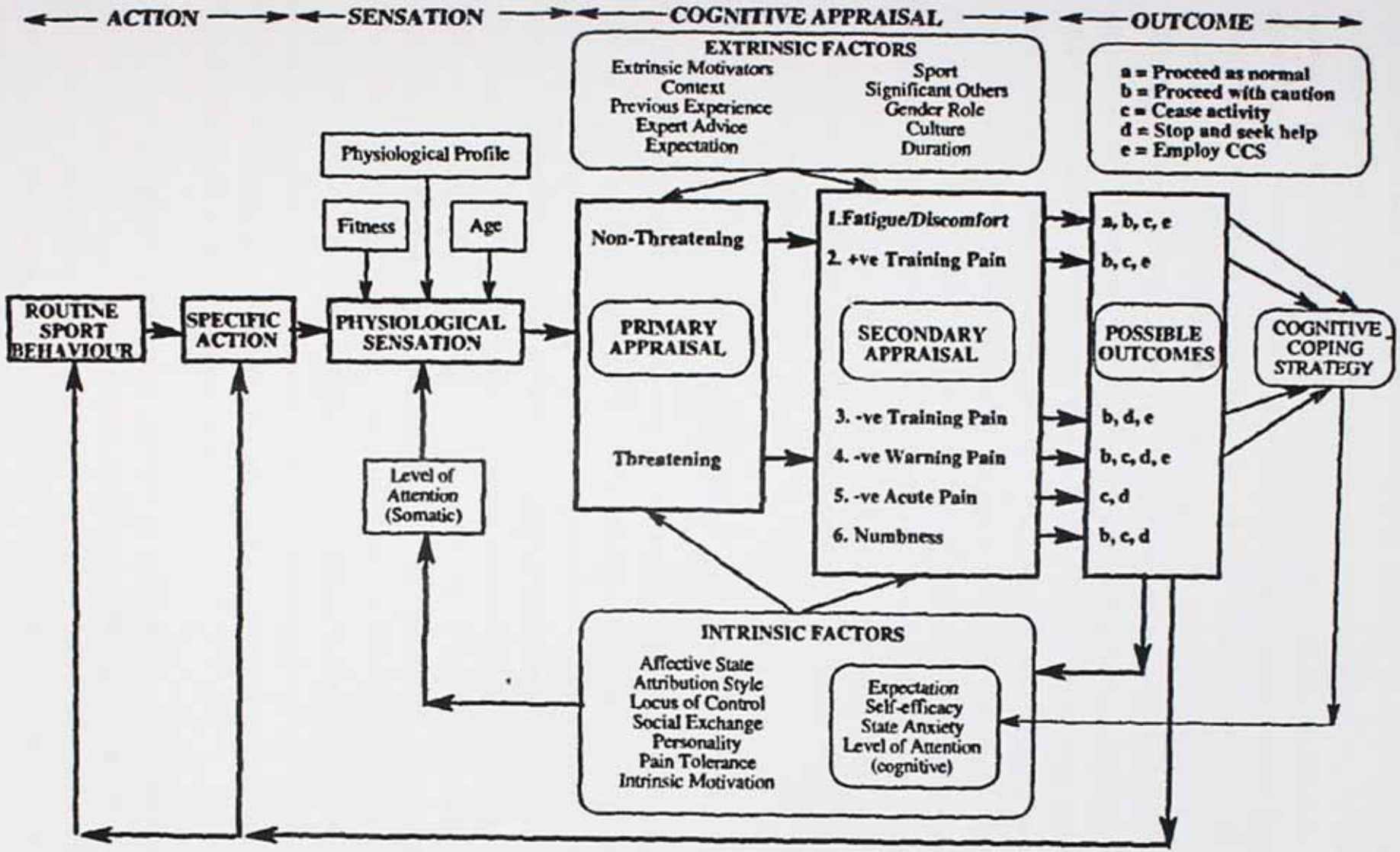


Figure 2.3. An integrative model of pain in sport (Addison et al., 1998).

Figure 2.3 summarises the work of Addison et al. (1998) whereby the highly complex processes involved in the athletic experience of, and response to pain are represented in a systematic manner. There are numerous variables referred to within the model that may help predict the way in which an athlete responds to a given pain stimulus, however the list is not exhaustive and should therefore form the basis for further research in order to help elaborate on what is already understood to be important. This model identifies the complicated underlying processes associated with pain during sports performance (such as physiological, psychological and behavioural characteristics) and suggests that they are closely intertwined to determine the outcome. Of particular note are the primary and secondary appraisals of pain given by athletes which categorise pain into threatening and non-threatening, with altered responses based upon the label attached to the pain experienced. With further development this model could be a valuable tool for practitioners to help guide athletes to deal with pain both during training and competition. For the current research this model is of interest as it suggests that intrinsic factors such as expectation may be able to influence the physiological sensation of pain which forms the basis for one of the experimental studies carried out and will be discussed further in the next section.

2.5.1 EXPECTATION OF PAIN

There is to date little known in relation to the role of expectation of pain and how that may influence muscle performance, however theories proposed in relation to pacing strategies in the heat can offer some indication of how and why anticipation of conditions may be an important factor for investigation (Noakes et al., 2004). Pacing strategies have been found to alter in hot climates where the athlete automatically, and subconsciously, selects an appropriate pace based upon the conditions (Tucker, 2006) and an expectation effect that determines athletic performance output based on the conditions has also been identified for pacing strategies in distance running (Noakes et al., 2004, 2005). However this is not caused by the conditions themselves, rather it is a response mechanism that reduces performance output based on anticipation of severity of environmental conditions. Although there is no existing research into this form of mechanism with regard to neuromuscular responses to pain, it could be argued that a similar central control mechanism exists.

Perception of effort or pain may positively or negatively affect fibre recruitment; for instance during the closing stages of a time trial athletes typically perform better and tolerate higher levels of pain (Tucker, 2006). Similarly anticipation of high ambient temperatures has been suggested to reduce muscle fibre recruitment thereby decreasing performance output (Noakes, 2004). In isometric contractions it has been found that by exposing a muscle to

experimentally induced muscle pain the motor unit firing rate decreases (Sohn et al., 2000) and Farina et al. (2004) found that the amount of nociceptive input was linearly correlated to inhibition, meaning the more pain that was detected, the more the contraction was limited. However, method of pain induction for both studies was injection of hypertonic saline which is highly invasive and difficult to control for level of pain.

Lund et al. (1992) suggest that pain causes the agonistic muscles to shut down in order to protect the organism from potential harm, whilst Hodges & Tucker (2011) argue for a non-uniform response in which muscle recruitment patterns are altered, which also serves to protect the region. What is unknown, however, is whether it is the physical stimulus of pain that causes the muscle to reduce its force output, or whether the expectation of receiving a pain stimulus would be sufficient to cause performance decrements. As explained earlier, there is opportunity for modulation of a pain stimulus at numerous points as it ascends to the brain (Melzack & Wall, 1965). Descending pathways may interact with the ascending message to modulate the end resulting pain experience, and where an individual is expecting high dose pain the descending messages may then interfere with the ascending message to create a pain perception higher than the actual pain stimulus that is applied. Alternatively, if a pain stimulus higher than the expected level of pain is applied, then the modulation from the descending pathways may create a pain perception that is lower than the actual stimulus warrants (Melzack & Wall, 1965).

Descending pathways can have an influential role in the pain experience by modulating the signal that eventually arrives at the brain. Placebo response research has demonstrated that responses to a stimulus are partially mediated by simple cue-outcome contingencies (Brown et al., 2008). However, when induced by verbal cues these responses are found to be particularly significant as the perception of pain can be increased and decreased through manipulation of the verbal cues hence altering the level of uncertainty (Benedetti et al., 2003; Fields & Price, 2005; Watson et al., 2006). There are environmental factors that have been found to make pain symptoms more relavant and hence change the perception of pain that a participant experiences. For example, being warned that a procedure may be painful, combined with a prior experience of pain can lead to a schema whereby pain or other symptoms are more carefully monitored and attended to (Bayer, 1998; Leventhal & Everhart, 1979) suggesting that pain expectancy may have a strong influence on pain perception (Price, 1999; Fields, 2000; Wager, 2005; Vase et al., 2005).

Expectation of pain has been shown to alter attention (Hudson et al., 2006) and information processing demands (Van Damme et al., 2004) but how this may alter muscle

performance has not been established. There are different components of the pain experience that should be considered which include the cognitive, affective and sensory-discriminatory aspects (Finniss, 2005). If a substance is believed to increase pain (nocebo expectation) it may alter expectations (cognitive), but it may also lead to anticipatory anxiety (emotional). A cognitive modulation of pain therefore may be assumed to comprise of an emotional response, and the influence emotions have on pain may include cognitive operations, so they should be considered as interdependent constructs (Anderson & Pennebaker, 1980; Weich, 2009). A study using fMRI activation patterns to identify differences between control and placebo analgesia supported this by suggesting that placebo analgesia affected all three dimensions of the pain experience (Wager et al., 2004). A heightened fear of pain, which may be induced by verbal cues or expectations of pain, has also been shown to up-regulate the sensitivity in regions of the brain encoding the emotional aspects of pain (Weich, 2009) which may explain the altered responses to pain due to expectation and support the hypothesis that anticipation may lead to altered pain responses.

2.5.2 PLACEBO AND NOCEBO EFFECTS

A placebo can be defined as an inert substance or procedure that alters one's physiological and psychological response (Stewart-Williams, 2004). Research on the effects of placebos suggests an important link between physiology and psychology (Geers et al., 2007) as they are believed to produce an effect in patients that results from intent rather than specific physical or chemical properties (Arnstein, 2003). Placebo's have been held partially responsible for bringing about treatment outcomes in many areas of medicine and play a major role in the clinical practices of both medicine and research (Geers et al., 2005). The placebo and nocebo effect may be useful in determining the psychological and physiological involvement in pain responses, however even within the placebo response it is unclear whether changes can be attributed to physiological and psychological factors. In pain research there has been a focus on endogenous opioid mechanisms and their involvement in placebo analgesia. However other non-opioid mechanisms (such as seretonin and other hormone secretion) have been suggested, which may be possible physiological explanations for the influence of pain on performance (Benedetti et al., 2003b). There is no definitive explanation for the mechanisms associated with the placebo response, however the major neurobiological mechanisms that have recieved considerable attention have been conscious expectation and unconscious behavioural conditioning (Pacheco-Lopez et al., 2006).

The conditioning mechanism has been based upon Pavlov's theory of classical conditioning (1927) where it is suggested that a previously neutral stimulus (such as the

environment), when coupled with an unconditioned stimulus (such as a drug), is able to elicit a response (Siegel, 2002; Stewart-Williams & Podd, 2004). Therefore it could be suggested that by giving an individual expectation for a certain level of pain, this could produce a conditioned response based upon their previous exposure to pain and thereby alter their performance. In an attempt to explain the placebo response through physiological mechanisms, de la Fuente-Fernandez et al. (2004) proposed that when an interaction creates a reward possibility (such as positive verbal suggestion), certain cortical neurons are activated in relation to reward probability. These neurons send direct excitatory glutamergic inputs to dopaminergic cell bodies along with indirect inhibitory gamma amino butyric acid inputs. The combination of these signals arriving at the dopaminergic neurons contributes to the probability of tonic activation. In further research the tonic activation during reward expectant conditions was examined and it was reported that neurons in the prefrontal cortex, nucleus accumbens and the caudate-putamen displayed tonic activation when the participant was expectant of recieving a reward (Schultz, 1998). This would suggest that there are chemical changes associated to the expectation of an event occurring, so therefore it is not simply a psychological phenomenon.

As discussed in section 2.4.1 when a message is sent to the brain there is opportunity for modulation of the signal to occur and once in the brain this opportunity is still existant. All thoughts come from communication between nerve cells (Benson, 1994) and when you consider that within the brain there are in the region of 5000 to 500 000 connections between nerve cells, and any of these connections offers opportunity for modulation of a message this provides huge opportunity for modulation to occur. As suggested by the neuromatrix explanation, there are some aspects of human perception that are genetically programmed, whilst others are brought about through environmental factors and learning (Melzack, 2001). Every event that occurs within our lifetime becomes stored as a schema and is interpreted based upon previous events which suggests that whilst there are physiological events that must occur in order for us to generate thoughts, we must also regard the psychological aspects of the process in which messages are interpreted.

Numerous psychological factors have been found to influence the placebo response (Pacheco-Lopez et al., 2006). The strength of the placebo response has been found to alter based upon motivational processes (Geers et al., 2005), concern (Todd, 1987), anxiety (Melzack, 1988), feeling uncomfortable (Todd, 1987), consciousness (Benedetti et al., 2003), expectations (Benedetti et al., 2003), optimism (Geers et al., 2007), pessimism (Geers et al., 2005) and the character of pain (Hauor, 2005). All of these factors can mediate the effect of a message that is being sent to the brain via interactions occurring in between ascending and descending pathways. If a person has optimistic traits then they may modulate the message accordingly

and be less prone to reporting pain (Geers et al., 2007). The same principle could apply to any psychological variable that may have the opportunity to modulate ascending messages, thereby inferring that there is possibility that some are born with innate abilities to tolerate pain better (Pen & Pisher, 1994), but also individuals can learn to deal with the negative effects of pain in sports performance (Benedetti et al., 2003; Geers et al., 2007).

Nocebo was a term originally used to define negative symptoms due to a placebo (Geers et al., 2005), for example, negative meanings that patients attach to verbally induced expectations (Benson, 1997; Hahn, 1997; Speigel, 1997). Whilst referred to seperately from placebo effects, the underlying mechanisms behind nocebo effects are considered to be the same (Hahn, 1997). At its most extreme, in the Framingham Study (Eaker et al., 1992), nocebo effects have been attributed to causing death; in these cases it is thought to be the belief that you are going to die that will increase the risk of death. In less extreme cases, researchers have found that the search for pain, or expectation towards pain, is what leads to altered pain reporting behaviour rather than any actual experience of pain (Bayer et al., 1998). If a substance is believed to increase pain, then it seems feasable to believe that this may change expectations and also anticipatory anxiety (Weich, 2009). This is not limited to the use of substaces to bring about changes to pain perception. In a study by Bayer et al. (1998) participants were lead to believe that a sham stimulator would be passing electrical current through their heads and that they may experience a head ache as a result of this. Consistent with studies using substance ingestion, it was found that in the absence of a painful stimulus, participants still reported pain demonstrating the powerful influence of expectation. If the role of expectation of pain can be better understood, it can be applied to the elite sport environment in order to best help athletes deal with the pain of performance, or alternatively help with rehabilitation from injury in athletic and non-athletic populations.

2.6 AIMS

The literature currently offers a wide range of conflicting theories attempting to identify a simple pain pathway (Marshall, 1894; Von Frey, 1894; Weddell et al., 1948), or lack thereof (Addison et al., 1998; Melzack, 2001; Melzack & Wall, 1965) and how this may influence muscle performance ((Hodges and Tucker); Lund et al., 1991; Roland, 1986) without any one definitive explanation being supported. The development of new technologies may allow for this to be understood better in the coming years, however regardless of mechanisms, what is of value to applied practitioners is an understanding of how the pain experience influences behaviour and what, if anything, can be done to prevent or minimise negative consequences of pain. In order to achieve this there needs to be a clearer understanding of the role of psychological and

physiological influences upon pain and sports performance which has received limited attention to date. The current research sought to further understand the relative influence of psychological and physiological factors in performance when an individual experiences pain. Each chapter focusses upon achieving these aims with regard to specific hypotheses; however the general aims of the work were addressed through the following questions:

1. Does pain perception threshold induced via GPD alter muscle performance?
2. Does the relationship between pain and muscle performance alter based upon pain severity?
3. What role, if any, does expectation of pain severity play in muscle performance?

3 PILOT WORK

3.1 INTRODUCTION

In order to study the effects of pain a number of experimental methods have been developed including electric shock (Neddermeyer et al., 2008; Tursky, 1972), hypertonic saline injections (Arendt-Nielsen et al., 1996; Ervilha et al., 2005), delayed onset muscle soreness (DOMS) (Dannecker et al., 2008) and thermal pain stimuli (Dannecker et al., 2008; Hatayama & Shimizu, 1993; Neddermeyer et al., 2008). These methods have been validated for use in the studies in which they were used, however they have limitations in their application for investigating mechanisms by which pain alters performance and also tend to be highly invasive (Wing et al., 2011a). A method used by Ryan & Kovacic (1966) enabled the experimenters to induce pain in a non-invasive manner. The gross pressure device (GPD) induces pressure pain to the tibia using a blood pressure monitor to carefully control amount of pressure exerted. In the Ryan & Kovacic study (1966) pain tolerance levels were examined in relation to athletic versus non-athletic groups, rather than investigating muscle performance; however the benefits of this method are that it allows pain to be induced away from the muscles, thereby not interfering with normal muscular functioning in the area being measured for performance. This device has however not been validated for use at pain perception threshold, the lowest stimulus recognised to be painful (Loeser & Treede, 2008). Therefore, in order to establish the optimal methods for this research project, a number of pilot studies were conducted to establish validity, reliability and positioning of the GPD constructed.

The use of the GPD in inducing controlled pain at tolerance level has been validated in previous research (Ryan & Kovacic, 1966) and it has been suggested that the ability to tolerate pain induced by the GPD is a good indicator of the ability to tolerate pain generally (Bartholomew, 1998). Pain is thought to consist of two components: sensory and emotional (Melzack & Torgerson, 1971). In asking a participant to identify pain perception threshold (the earliest perception of pain) as will be used in this study, there is little threat to the emotional element. Conversely measuring pain tolerance levels (defined as the maximum amount of pain an individual can withstand) may induce a fear component which may exaggerate the pain response. Previous studies have tested pain tolerance (Ryan & Kovacic, 1966) and for this reason, whilst the GPD was considered a valid measure within the pain tolerance studies, this cannot be generalised to pain perception threshold as well, as the two constructs are working within different components of pain.

3.1.1 THE GROSS PRESSURE DEVICE

A prototype GPD was developed for use in this research based on the work of Ryan and Kovacic (1966). It consists of a modified sphygmomanometer cuff (model DM304, Mercurial sphygmomanometer Bigger Case, China) which was extended to allow it to fit around the girth of a lower leg, a curved plastic shin pad (107 x 175 mm) with a removable semicircular metal cleat (80mm length, 23mm radius) and Velcro attachment to the inner surface of the shin pad positioned over the tibia.

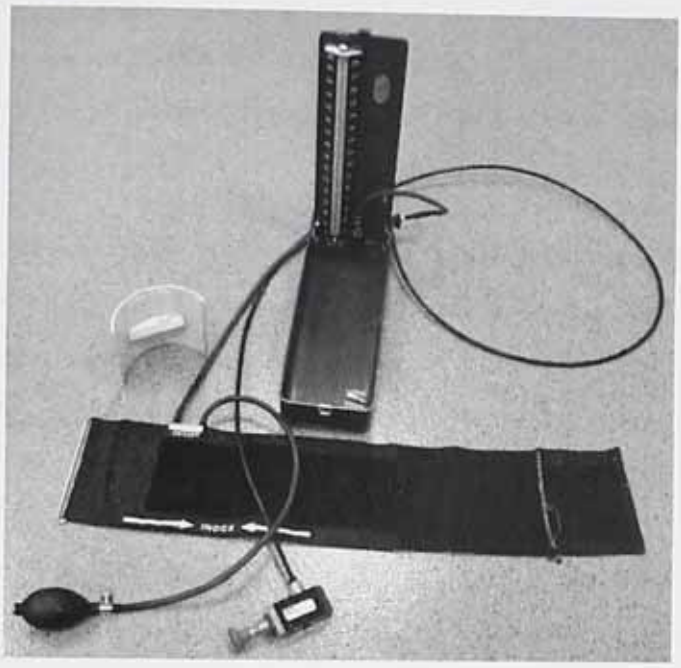


Plate 3.1. Gross Pressure Device consisting of a modified sphygmomanometer cuff and blood pressure monitor, safety release valve for the participant to hold during the trials, curved shin pad and metal plated cleat.

3.2 POSITIONING OF THE CLEAT FOR RELIABLY INDUCING PAIN PERCEPTION THRESHOLD ON THE TIBIA

3.2.1 INTRODUCTION

Previous research using the GPD has not specified precisely where on the limb pain is induced (Ryan & Kovacic, 1966). The anatomy of the lower leg would suggest that there may be differences in an individuals' sensitivity for pain based upon location of cleat relative to the skin surface, number of nerves, amount of fatty deposits, curvature of the bone and muscular coverage of the bone. Consequently placement of the GPD is important to ensure that the position chosen is that which elicits the earliest pain response from the GPD, thereby ensuring inflation of the cuff is minimal and therefore likelihood of confounding variables related to ischaemia reduced.

An examination of the muscular anatomy of the lower leg shows that in the upper portion of the lower leg the tibialis anterior, sartorius and extensor hallucis longus muscles cover some of the medial subcutaneous surface of the tibia which means there is some protection offered to the tibia. In the lower third of the lower leg there is also protection offered to the bone surface in the form of the superior extensor retinaculum, extensor digitorum longus and extensor hallucis longus which all merge towards the ankle joint forming a muscular layer over the bone as illustrated by Plate 3.2. An axial view of the structure of the lower limb indicates that there is little soft tissue protection offered to the surface of the tibia in some regions of the lower leg. The posterior border and medial subcutaneous surface of the tibia are most exposed across the mid portion of the lower leg which means that there is little protection of the bone from the cleat of the GPD. The periosteum that surrounds all bone isolates the bone from surrounding tissue, as well as providing a channel for nervous supply to the lower limb (Martini, 2004). This would suggest pain sensitivity will be greatest over the bony surface and the mid region of the lower leg, therefore 50% from the medial malleoulus would face an increased level of pain sensitivity due to the bones exposure.

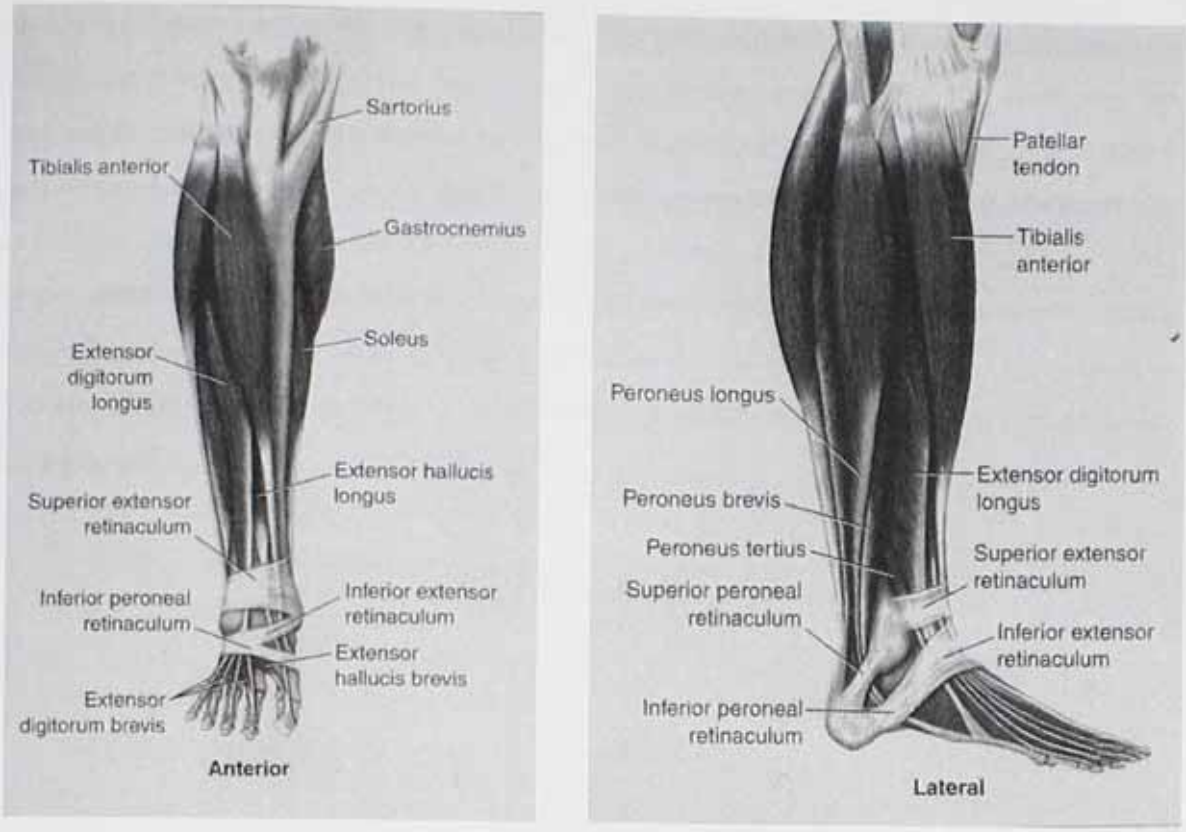


Plate 3.2. The anterior and lateral muscles of the ankle and foot (Behnke, 2006).

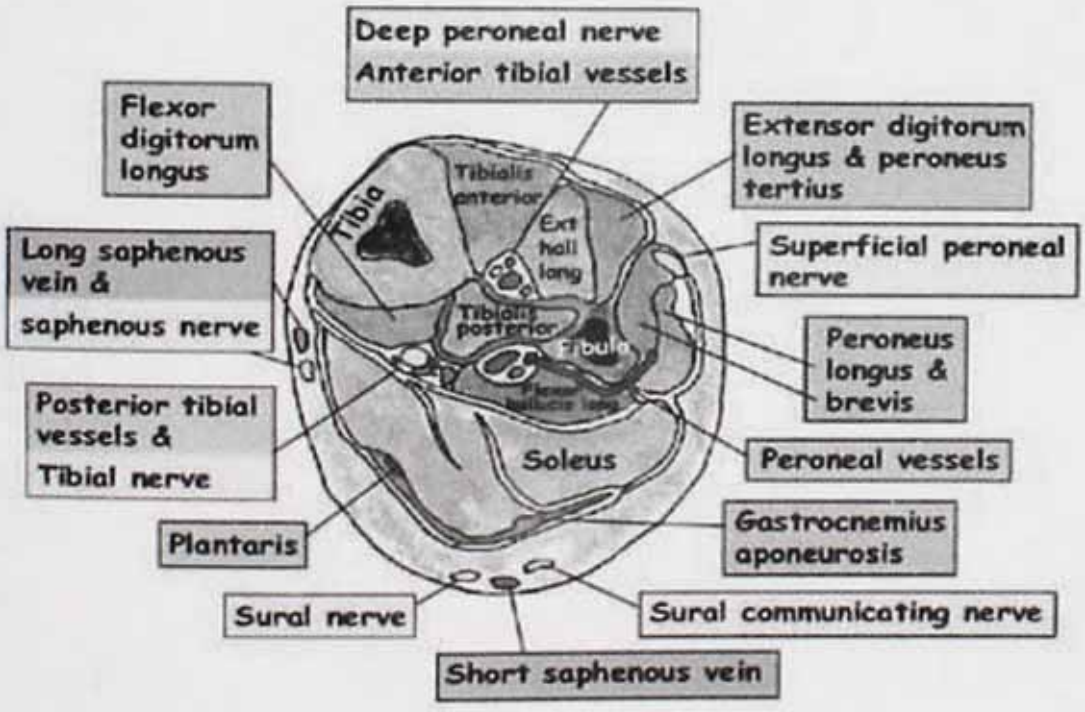


Plate 3.3. Cross sectional axial view of the lower limb (Whitaker, 2010).

The location of major nerves within the lower leg also suggests pain sensitivity may be increased in the mid portion of the lower leg. The nerves surrounding the tibia are the saphenous nerve and superficial peroneal nerve; both located to the side of the tibia and hence should not interact with the GPD (see Plate 3.3). The deep peroneal nerve also runs beneath the extensor hallucis longus and so this may be detected on some of the sites. The main nerve that may interact with the GPD is the middle cutaneous nerve of the calf which runs directly down the centre of the lower leg and hence is quite exposed as can be seen in Plate 3.4. The exposure of the nerves along the mid-section of the lower leg could indicate a possible reason for increased pain sensitivity.

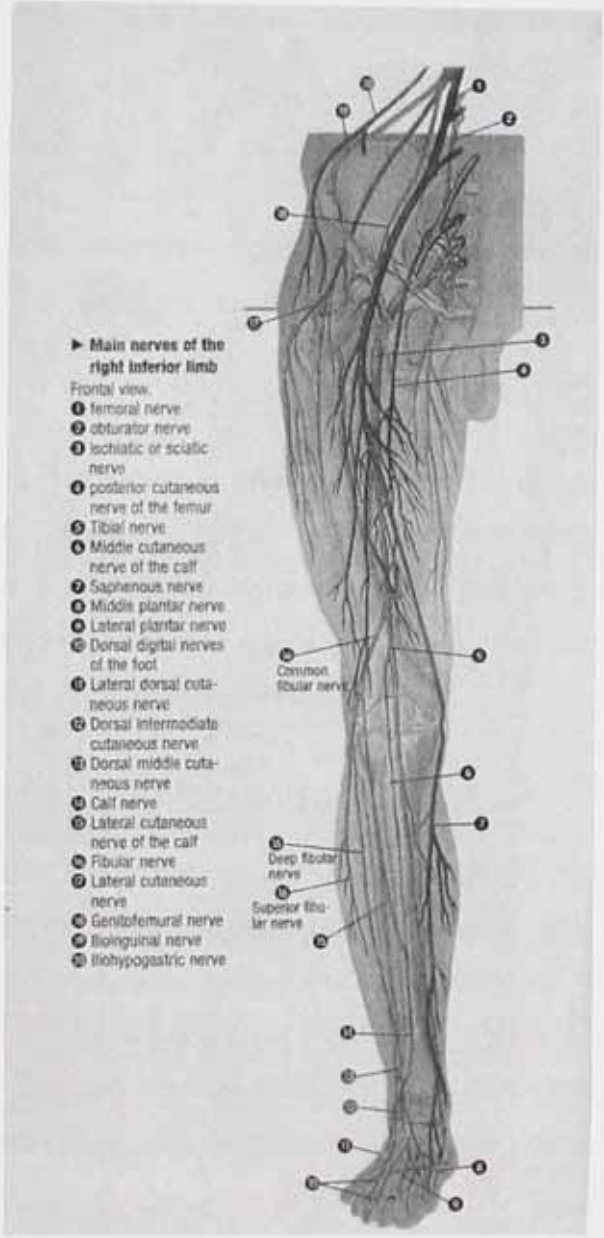


Plate 3.4. Nerves of the lower leg (Behnke, 2006).

A further consideration to cleat placement is the soft tissue structure of the lower leg. Whilst individual difference accounts for a large amount of variation within the exact distribution of fat on the body, there is limited space available beneath the skin surface on the anterior surface of the lower leg and this prohibits gross fat formation. Individuals are more likely to have fatty deposits on the upper leg where there is more soft tissue or on the posterior side of the lower leg. Within the anterior side of the lower limb itself, there appears to be more soft tissue coverage of the bone at the upper and lower extremities of the lower limb. This includes tendons and muscular coverage, but also fatty deposits. At these sites it would be expected that lower perceptions of pain will be prevalent due to increased coverage of the bone. If an individual has large fatty deposits on the lower limb this may cushion the effects of the GPD and hence increase that person's pain tolerance which should be a consideration in participant suitability.

3.2.2 AIM

The aim of this study is to determine the optimal placement of the GPD on the lower limb to induce the lowest pain perception threshold.

3.2.3 PARTICIPANTS

Twenty healthy males volunteered to take part in the study having given written informed consent (see Appendix 1) (mean ±SD; age = 23.9 ± 5.9 yrs, height = 1.7 ± 0.05m, mass = 77.6 ± 0.9kg). All participants were recreationally active and free from illness or injury that would interfere with the study results. Ethical approval was obtained from the Moulton College Ethics Committee in accordance with the declaration of Helsinki.

3.2.4 PROCEDURE

Participants sat with their knee held at a 90° angle. The GPD was placed on the participant's self-selected dominant leg. The five selected sites were marked along the tibia with a permanent marker and measured with an anthropometric tape measure (Body Care, Warwickshire, UK). The shin pad and cleat were held in place using the sphygmomanometer cuff but this remained deflated until immediately prior to each experimental condition. The equipment was set up as illustrated in Plate 3.5. The cuff was inflated at a constant rate of ~5mmHg on each inflation to put pressure on the cleat.

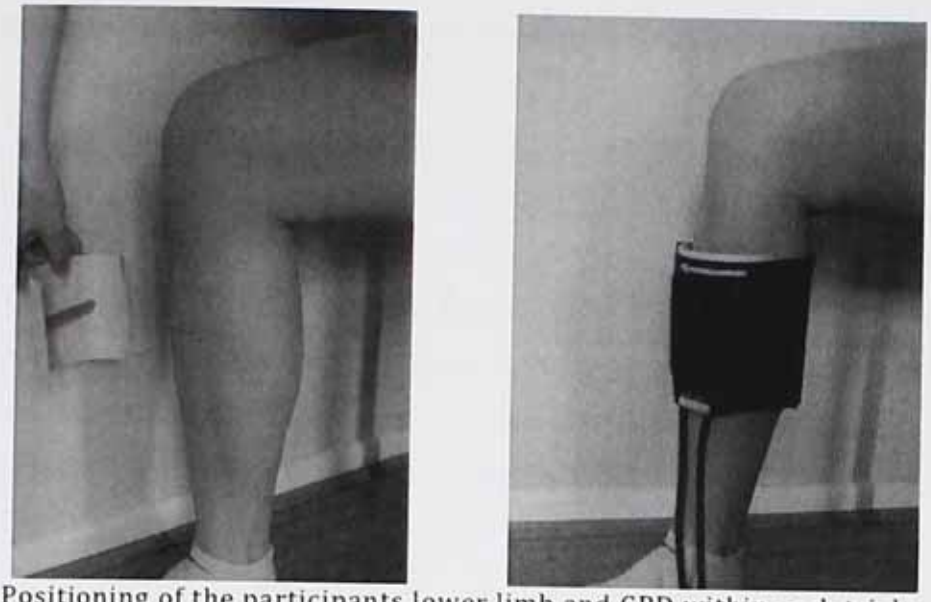


Plate 3.5. Positioning of the participants lower limb and GPD within each trial

Participants were given instructions as to what pain perception threshold was, and that there was no merit in exceeding this. Trials took place in a room away from others and the participant had no knowledge of their own or other participants' results, thereby limiting the potential for confounding variables. Pain was induced at all five sites with a two minute interval between tests. This was carried out in a randomised, counterbalanced and cross-over manner to prevent likelihood of order or familiarisation effects.

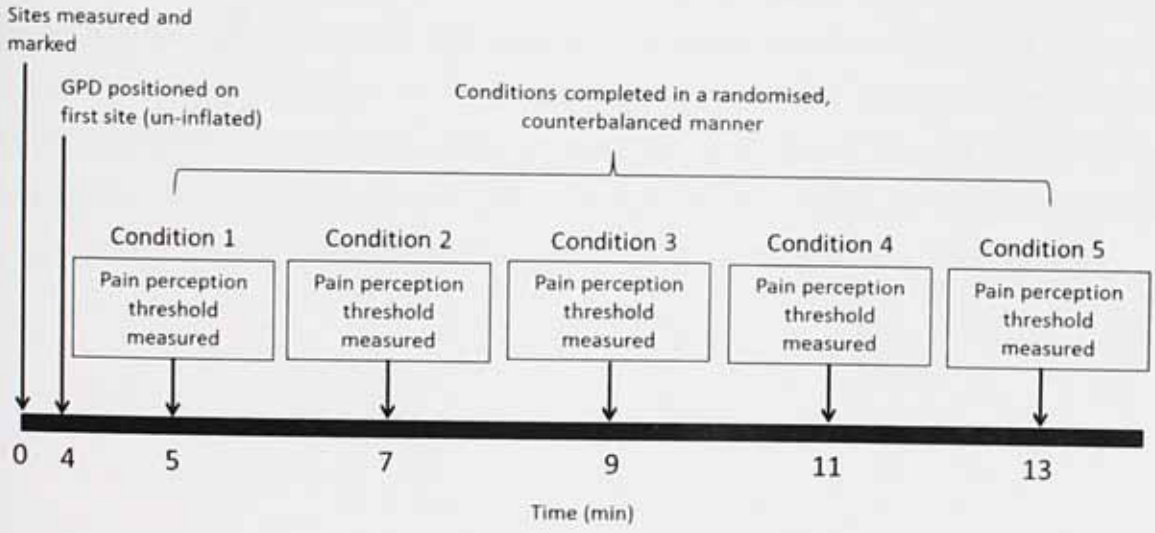


Figure 3.1. Timeline of protocol used to establish positioning of GPD

3.2.5 EXPERIMENTAL CONDITIONS

Five measurement sites were selected along the lower leg in order to cover the largest possible scale. The upper and lower 25% of the lower leg were excluded due to practical considerations; the GPD could not be secured to the leg in those areas. Five sites were measured along the

anterior surface of the lower leg at 25%, 33%, 50%, 66%, 75% from medial malleolus to medial condyle.

3.2.6 DATA ANALYSIS

Data were analysed using the SPSS statistics software package (version 17.0). Data within each condition were examined for normality using the Kolmogorov-Smirnov test. Where the raw data were not normally distributed appropriate data transformation was used. Where no suitable transformation could be found, non-parametric tests were used. All data are reported as mean ± SD where normally distributed, and as median, IQR and range where non-parametric. Statistical significance was accepted at $p < 0.05$.

A one way ANOVA was carried out comparing values for all five sites. A Bonferroni post-hoc t-test was carried out to establish difference between sites and a correction factor of ten was used. This ensured that likelihood of type I error was not inflated as multiple comparisons had been accounted for. An alpha value of $p < 0.005$ was set as the acceptable limit for these tests.

3.2.7 RESULTS

Data were found not to be normally distributed at the 25%, $D(17) = .20$, $p < 0.05$, 33%, $D(17) = .18$, $p < 0.05$, and 66%, $D(17) = .18$, $p < 0.05$, sites. Whilst all other sites were found to be normally distributed no suitable data transforms could be found, and therefore non-parametric tests were used for analysis. A Friedmans ANOVA revealed a significant difference in pain perception thresholds at the five different test sites on the lower leg, $\chi^2(4) = 38.613$, $p < 0.05$ (see Figure 3.2). Wilcoxon tests revealed a significant difference ($p < 0.05$) to lie between all sites except between 25%-75% ($z = -0.91$, $p > 0.05$) and 33%-66% ($z = -1.95$, $p > 0.05$).

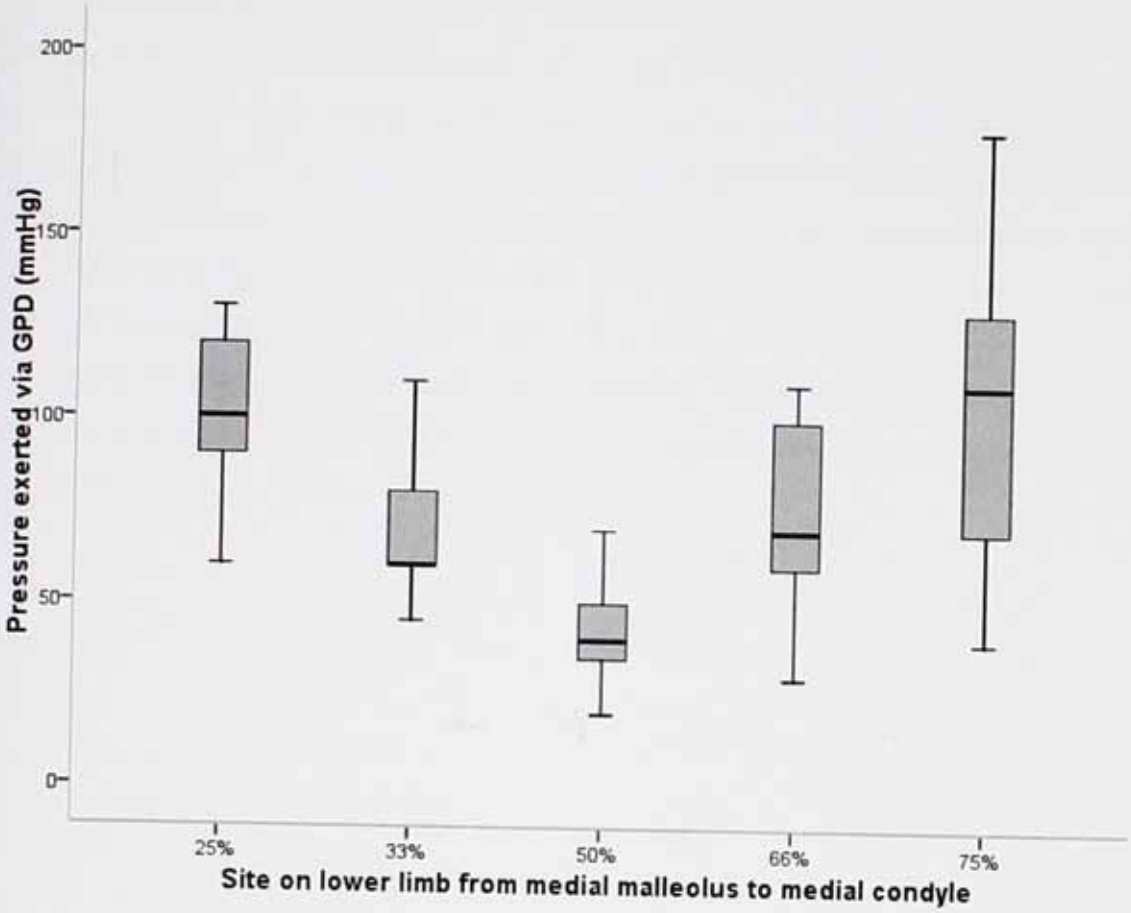


Figure 3.2. Median pain perception threshold measured across five sites on the lower limb between medial malleolus and medial condyle. Data presented as median, IQR and range.

A Friedmans ANOVA showed that there was a difference in pain perception threshold between sites and post-hoc t-tests with Bonferroni correction establishing that at 50% from the medial malleolus to medial condyle median pain perception threshold was significantly lower ($p < 0.005$) than at all other sites. This was also the site at which the range was smallest (median = 40.0, IQR = 25.0, range = 80.0mmHg) indicating higher reliability than at the other sites.

3.2.8 DISCUSSION

The results of this study support the use of the GPD within pain perception threshold testing and further to this that optimal placement is at 50% from medial malleolus and medial condyle. Determining a site on the lower limb that produces the highest pain perception from the lowest pressure stimulus reduces the need to inflate the cuff and in doing so limits the possibility of confounding variables such as ischaemia. This suggests less variance at this site and therefore more homogeneity between participants which may have implications for validity and reliability. There is no documented evidence that the positioning of the GPD affects pain response, however this study suggests that 50% between the medial malleolus and medial condyle is the optimal placement to induce early pain perception threshold responses.

An explanation for this heightened sensitivity at the 50% mark may lie in the muscular anatomy of the lower limb as discussed previously, in which the bone surface of the tibia and the free nerve endings are exposed at the mid-point of the lower leg. Aside from the anatomical makeup of the lower leg, it is also important to consider other variables that may affect pain response. As reported in a study using DOMS as a measure of pain tolerance, there were higher pain ratings after the first bout of tests than after the second, suggesting that desensitisation occurred (Dannecker et al., 2008). This study also used multiple trials and therefore a desensitisation effect may have occurred. However a crossover and counterbalanced design was used which eliminates familiarity effects and suggests that results are valid regardless of order in which the sites were tested.

3.3 RELIABILITY OF INDUCING PRESSURE PAIN VIA GPD ON CONSECUTIVE DAYS

3.3.1 INTRODUCTION

The initial pilot work conducted in this research determined the position at which the gross pressure device was perceived to be most painful (50% from medial malleolus to medial condyle). This study aimed to determine the reliability of the GPD at pain perception threshold level when induced on consecutive days to determine the ability of the GPD to be used in studies where testing is not carried out within one session. In previous research the GPD has been found to have a .95 test-retest reliability coefficient when measuring pain tolerance (Brewer et al., 1990; Ryan & Kovacic, 1966) which means it is highly likely that on two separate tests the same level would be reached. In order to be a useful tool in studying the effects of experimentally induced pain, the GPD needs to be able to induce pain repeatedly and illicit the same response from the participant. However within this study the equipment has been modified slightly and therefore the results cannot simply be generalised across. The study also differs from previous research in that it seeks to measure pain perception threshold rather than pain tolerance, therefore again any previous research using the GPD cannot be presumed to have the same reliability and for that reason a series of pilot studies were carried out to determine its suitability.

A study investigating pain perception threshold using algometry to induce pressure pain on the torso and upper extremities found there to be good reliability across the four days tested with highly consistent intraclass correlations (Jones et al., 2007). When pressure pain threshold was tested in measures taken a week apart using an algometer this finding was also supported with no significant difference being found between measures (Brennum et al., 1989). Pain is thought to have a sensory and emotional component (Melzack & Torgerson, 1971) and as a result of this emotional attachment it may be that familiarisation to a given stimulus will reduce sensitivity to it and therefore reduce reliability. Algometry studies show pain perception threshold to be reliable across different days and weeks (Brennum et al., 1989; Jones et al., 2007; Orbach & Gale, 1989) however a significant finding from Jones et al. (2007) reported that on day four readings were significantly lower than the baseline measure taken on day one suggesting that there may be familiarisation effects evident when the participant is exposed to repeated trials. Jones et al. (2007) argued that this was as a result of a reduced fear element attached to the stimulus when the participant was familiar with it. This finding was however in contrast to the findings of Orbach & Gale (1989) who found no significant differences. This pilot study will aim to determine if the GPD induces pressure pain reliably over consecutive days

which will show whether or not the device can be reliably used to test participants on separate days during the experimental trials.

3.3.2 AIM

To determine the reliability of the GPD to induce pain perception threshold on consecutive weekdays.

3.3.3 PARTICIPANTS

Seven healthy male volunteers (mean ± SD; age = 24.4 ± 10.7, height = 1.8m ± 0.03, mass = 78.9kg ± 3.5) volunteered for the study. The participants all completed an informed consent form prior to commencement of the study confirming that they had no injury or illness that may exclude them from taking part (see Appendix 1). Ethical approval for this study was granted by the Moulton College Ethics Committee.

3.3.4 PROCEDURE

The participants each completed ten trials on ten consecutive weekdays at the same time of day (± 1 hr) to avoid circadian influences. In each trial the participant was asked to indicate pain perception threshold once using the GPD at the previously established site on the lower leg. The protocol for using the GPD was established in the first pilot study (see chapter 3.2).

3.3.5 DATA ANALYSIS

Data within each condition were examined for normality using the Kolmogorov-Smirnov test. Where the raw data were not normally distributed appropriate data transformation was used. Where no suitable transformation could be found, non-parametric tests were used. All data are reported as mean ± SD where normally distributed, and as median, IQR and range where non-parametric. Statistical significance was accepted at $p < 0.05$. Mean data were analysed comparing week one with week two using a paired t-test. Further analysis using repeated measures ANOVA were conducted to investigate the effects of time. Statistical significance was accepted at an alpha value of $p < 0.05$. Furthermore coefficient variation (percentage) was calculated using the equation

$$\frac{\text{standard deviation}}{\text{mean}} \times 100$$

3.3.6 RESULTS

Data were found to be normally distributed for week 1, ($D(7) = .17$, $p > 0.05$), and week 2, ($D(7) = .21$, $p > 0.05$). A paired samples t-test was conducted to compare the mean (± SD) pain perception threshold reported in week one (52.57 ± 10.0) and week two (60.21 ± 15.45) and determined that there was no significant difference between groups, ($t(6) = -1.58$, $p > 0.05$) (two-tailed) (see figure 3.3).

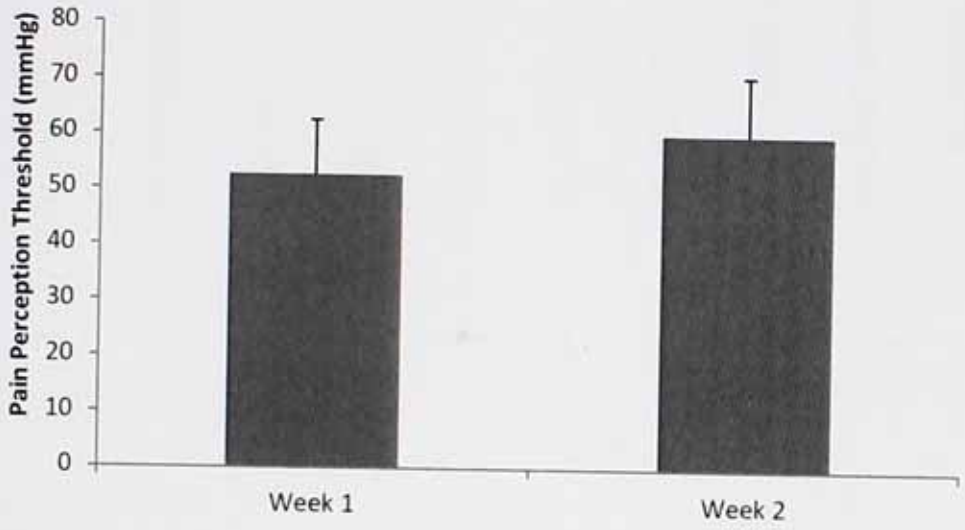


Figure 3.3. Pain perception threshold (mmHg) measured on consecutive weekdays. No significant difference ($p<0.05$) was found between measurement in week one and week two.

In comparing individual days, data from three days of the study were reported to violate assumptions of normality and no suitable transforms could be found; day 3, ($D(5) = .39$, $p < 0.05$), day 9, ($D(5) = .36$, $p < 0.05$), and day 10, ($D(5) = .36$, $p < 0.05$). Data from all other days were found to be normally distributed; however as the sample has to be considered as a whole, non-parametric tests were used. A Friedman's ANOVA revealed there to be no significant difference in pain perception threshold reported between days, ($\chi^2(9) = 10.228$, $p > 0.05$) (see Figure 3.4).

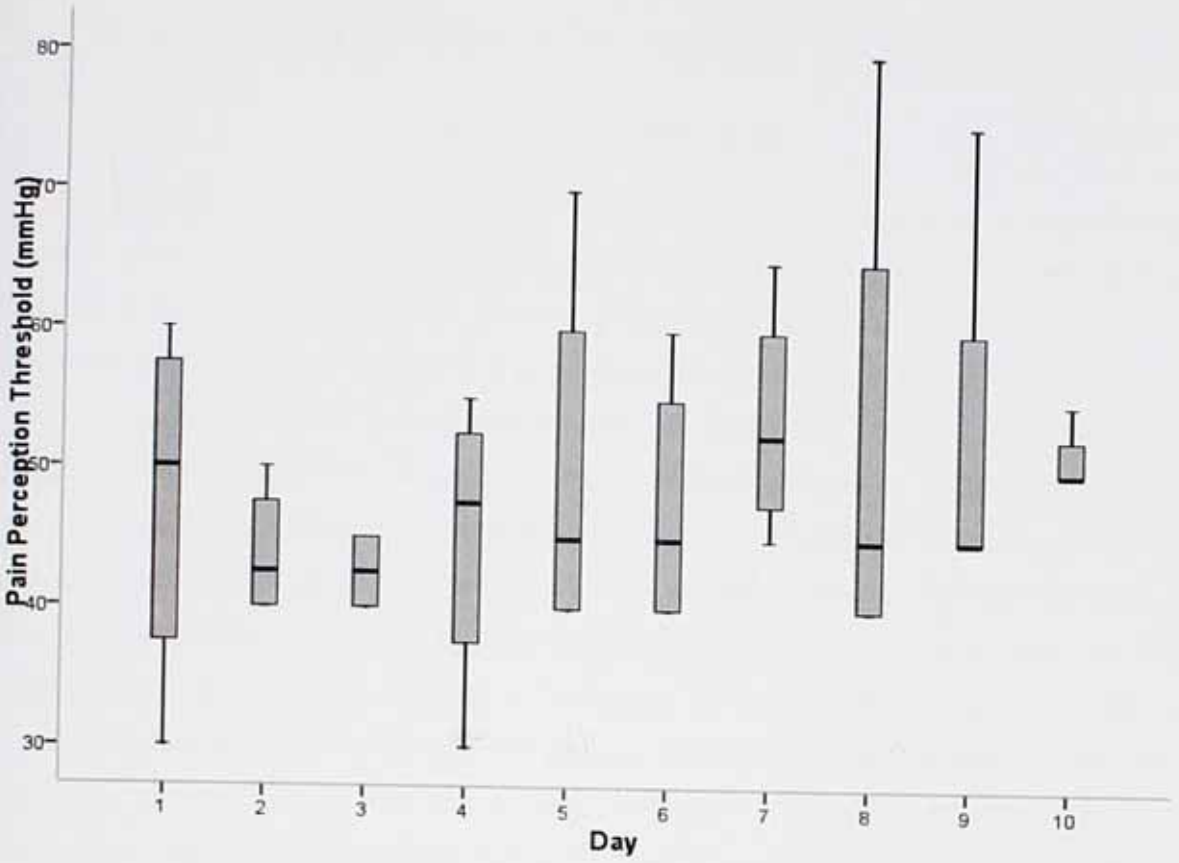


Figure 3.4. A comparison of pain perception threshold (mmHg) reported on consecutive weekdays over the course of two weeks. Data presented as median, IQR and range (n = 7). No significant difference ($p < 0.05$) was found between measures.

3.3.7 DISCUSSION

There were no significant differences found between the measurements taken in week one and week two and no trends were identified between the measurements taken on each day of the week. Further analysis established that the data reported in this study had a coefficient of variation of 17.5% and intraclass correlation coefficient of 0.98. This suggests that whilst there is some variation, the GPD is reliable across measurements taken on consecutive days with no desensitisation or familiarisation effects found to occur. In contrast to Jones et al. (2007) there was no significant difference found between the last measurement and the baseline measure as there were no differences detected between the days of the tests. It is also suggested by this study that the two day interval between week one and week two testing did not defamiliarise or influence the pain perception threshold of the individuals as no significant differences were detected between measurements ($p > 0.05$). This suggests that the GPD is suitable for use in studies where participants will be exposed to the stimulus on multiple days over a period of time, whether this be on consecutive days or more widely interspersed. What remains to be determined is whether this is also true of repeated tests on the same day.

3.4 INTRA-SESSION RELIABILITY OF INDUCING PRESSURE PAIN VIA GPD

3.4.1 INTRODUCTION

The previous pilot study established reliability of the GPD on measures of pain perception threshold taken across consecutive days; however, there is no data reported stating the reliability of the GPD across intra- session measures which is fundamental to the current research project as each participant will be required to complete multiple tests within the same session. There have been no reports of any lasting damage caused by the GPD where pain was induced at pain tolerance (Ryan & Kovacic, 1966), or within pilot work reported in this research testing pain perception threshold (chapter 3). It is therefore unlikely that there would be any physical implications to inducing pain at perception threshold level. However if the same site is being tested repeatedly in a short time period it may be that there is some transient damage caused that will increase the sensitivity of the testing site after multiple trials. The anatomy of the lower leg offers little protection to the surface of the tibia at the site being tested (Behnke, 2006) therefore any damage that may be caused would be confined to skin, bone, nerves and blood vessels. The likelihood of bone being damaged is remote due to the low pressures being exerted by the GPD, however the skin and blood vessels may be damaged through the pressure and bruising may result. This was not found to occur in previous pilot work, however in these tests the trials were separated by a minimum of 24 hours. This does not eliminate the possibility of heightened or reduced sensitivity resulting from later trials within a session which is what this current study examined.

3.4.2 AIM

The aim of this study is to determine the intra session reliability of the GPD.

3.4.3 PARTICIPANTS

A total of seven participants completed the study (mean ± SD; age = 26.2 ± 4.9yrs, height = 1.8 ± 0.09m, mass = 78. 3 ± 10.1kg). The participants gave written, informed consent prior to commencement of the study confirming that they had no injury or illness that may exclude them from taking part (see Appendix 1). Ethical approval for this study was granted by the Moulton College ethics committee.

3.4.4 PROCEDURE

The participants each completed six trials with a two minute interval between trials. In each trial the participant was asked to indicate pain perception threshold once using the GPD. The

GPD was then deflated for the remainder of the two minute interval and then inflated immediately prior to the next trial. The site at which the cleat was positioned was marked with a permanent marker. Between trials the GPD remained fastened but deflated to ensure the reliability of positioning in each trial. The protocol for using the GPD was established in the first pilot study (see chapter 3.2).

3.4.5 DATA ANALYSIS

Data within each condition were examined for normality using the Kolmogorov-Smirnov test. Where the raw data were not normally distributed appropriate data transformation was used. Where no suitable transformation could be found, non-parametric tests were used. All data are reported as mean ± SD where normally distributed, and as median, IQR and range where non-parametric. Statistical significance was accepted at $p < 0.05$.

3.4.6 RESULTS

Data were not found to be normally distributed in 2 of the 6 trials; trial 3, ($D(7) = .29$, $p < 0.05$), and trial 5, ($D(7) = .29$, $p < 0.05$). No suitable transforms were found and therefore a Friedman's ANOVA was used to examine if there was a significant difference between trials. Statistical significance was accepted at an alpha value of $p < 0.05$. There was no significant difference between mean pain perception threshold reported across the ten time points, ($\chi^2(5) = 2.598$, $p > 0.05$) (see figure 3.5). Further analysis established that the data reported in this study had a coefficient of variation of 12.2% and intraclass correlation coefficient of 0.84.

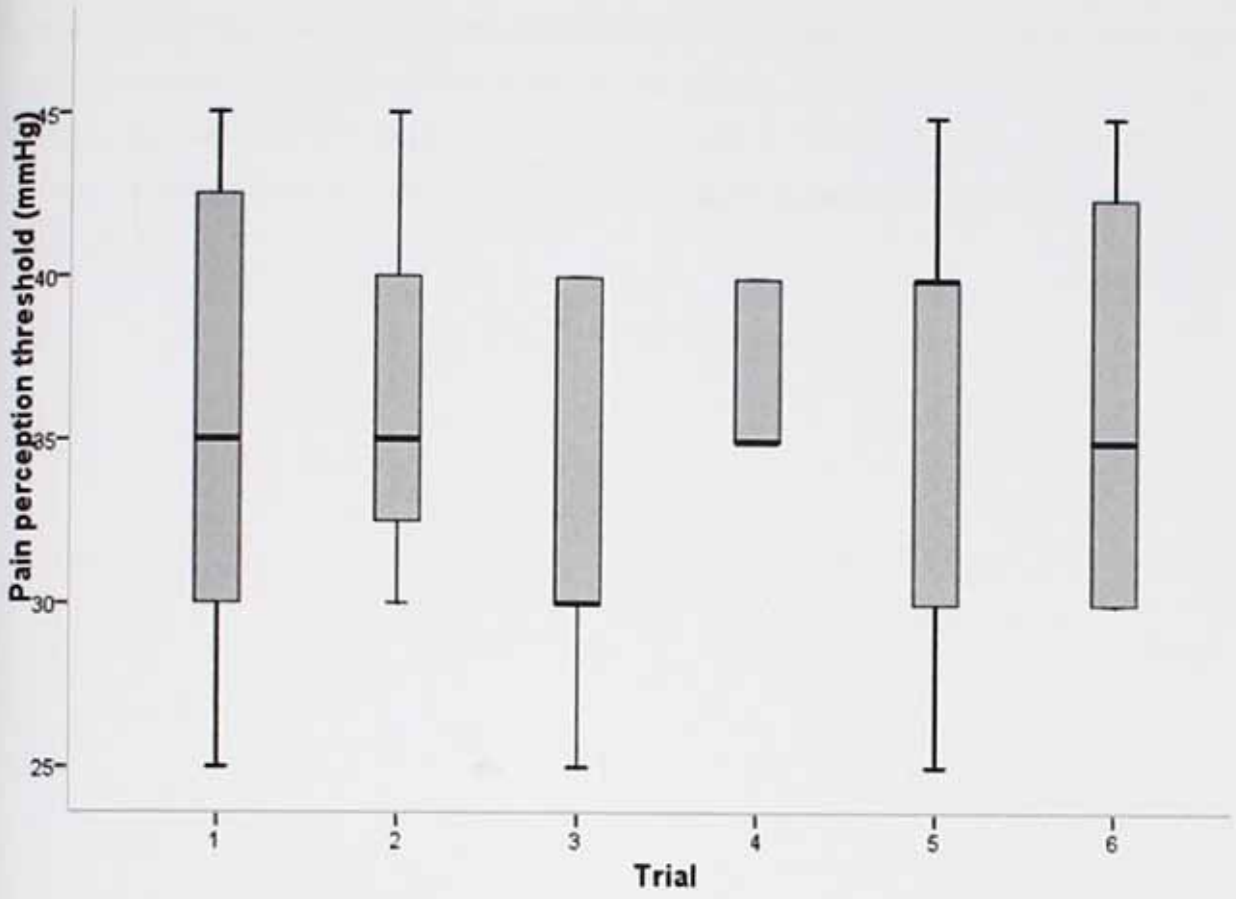


Figure 3.5. Pain perception threshold (mmHg) measured across 6 trials with 2 minute intervals. Data presented as median, IQR and range (n = 7). No significant differences ($p < 0.05$) were found between trials.

3.4.7 DISCUSSION

There were no significant differences ($p > 0.05$) found in intra session measurements suggesting that the GPD is a reliable method of pain induction for studies in which comparisons between individual scores in different conditions are required. There were no familiarisation or order effects apparent and no suggestion of increased sensitivity as trials continued. The anatomy of the lower limb suggests that there is little protection offered to the tibia when the GPD cleat is pressed against it (Behnke, 2006) which means that there is little soft tissue that can be bruised through use of the GPD. This may explain the findings of this study in showing no significant differences between the trials.

Jones et al. (2007) found that there were familiarisation effects evident in their study using algometry across a number of days, however in this study trials were conducted within the same session and such familiarisation effects were not evident. This could be due to the fact that this familiarisation effect was thought to occur as a result of cognitions attaching meaning to the pain stimulus and thereby rationalising it to be as a result of an experimental trial where no harm will result (Melzack & Torgeson, 1971). In this study there were only 2 minutes

between each trial meaning less time for the individual to think about the experience and it appears that this potentially resulted in maintenance of the emotional component of pain rather than any increase or reduction (Melzack & Torgeson, 1971). The results reported imply that the GPD is a reliable method of pain induction where multiple measurements are required within a single session.

3.5 ESTABLISHING THE SEVERITY OF PAIN THAT CAN BE TOLERATED USING THE GPD

3.5.1 INTRODUCTION

The previous pilot studies have focused upon the need to reliably induce pain at pain perception threshold level using the GPD. The reason the GPD was selected as the method of pain induction is due to the highly controllable nature of the device and the fact that it can be used to induce different severities of pain. However, all that is known to date is that this can be used to establish pain tolerance (Brewer et al., 1990; Ryan & Kovacic, 1966) and pain perception threshold (pilot work), but how much pain between these two threshold that can be comfortably withstood by an individual so that it can be repeatedly induced is unknown.

3.5.2 AIM

The aim of this study is to examine the pressure that can be induced via GPD and tolerated by individuals before pain tolerance is reached.

3.5.3 PARTICIPANTS

A total of ten participants completed the study (mean ± SD; age = 24.9yrs ± 10.1, height = 1.8m ± 0.06, mass = 79.2kg ± 4.0). The participants gave written, informed consent prior to commencement of the study confirming that they had no injury or illness that may exclude them from taking part (see Appendix 1). Ethical approval for this study was granted by the Moulton College ethic committee.

3.5.4 PROCEDURE

Upon arrival in the lab pain perception threshold was established for each participant. This then formed the baseline measure for each individual. The participants each completed as many trials as they were able to withstand. There were two minute intervals between trials. On the first trial pain perception threshold (as previously determined) was induced and then the cuff was deflated. In the subsequent trials pain perception threshold was multiplied by two, three, four etc. until the participant could no longer withstand the pressure induced. Participants were asked to indicate how painful each trial was using a VAS immediately following each trial (see Appendix 2). The site at which the cleat was positioned was marked with a permanent marker following the protocol established in chapter 3.2. Between trials the GPD remained fastened but deflated to ensure the reliability of positioning in each trial. A successful trial was considered to be any trial at which the predetermined pressure was

exerted. As soon as a participant indicated they wished to stop this ended their involvement in further trials and established their maximal pain tolerance.

3.5.5 DATA ANALYSIS

There were two variables measures in the present study; amount of pressure (mmHg) withstood, and pain perception indicated in each trial. Descriptive data of number of trials successfully completed by each participant is presented as there was not considered a benefit to statistical analysis. Trials are presented as a percentage of pain perception thresholds. The mean percentage of pain perceived by participants as indicated via VAS was examined for normality using the Kolmogorov-Smirnov test and repeated measures ANOVA used to determine any statistical difference present in the conditions where all participants successfully completed the trial. Significance was accepted at an alpha value of 0.05. Post-hoc t-tests with Bonferroni correction were used where appropriate to determine where any difference lay.

3.5.6 RESULTS

All ten participants successfully completed the 100%, 200% and 300% conditions before drop out occurred (see Table 3.1). No participants successfully completed 700% pain perception threshold, however over 50% of participant were able to withstand 500% pain perception threshold without the pain being considered unbearable. Figure 3.6 indicates that the pain perceived by participants increased in a near linear fashion in line with pressure exerted via GPD.

Table 3.1. Number of participants successfully completing each of the conditions and mean pain perception indicated via VAS.

Condition	Number of participants completing trial	Mean pain perception (%)
100%	10	12.4
200%	10	27.5
300%	10	49.3
400%	8	70.75
500%	6	83.5
600%	2	95
700%	0	N/A

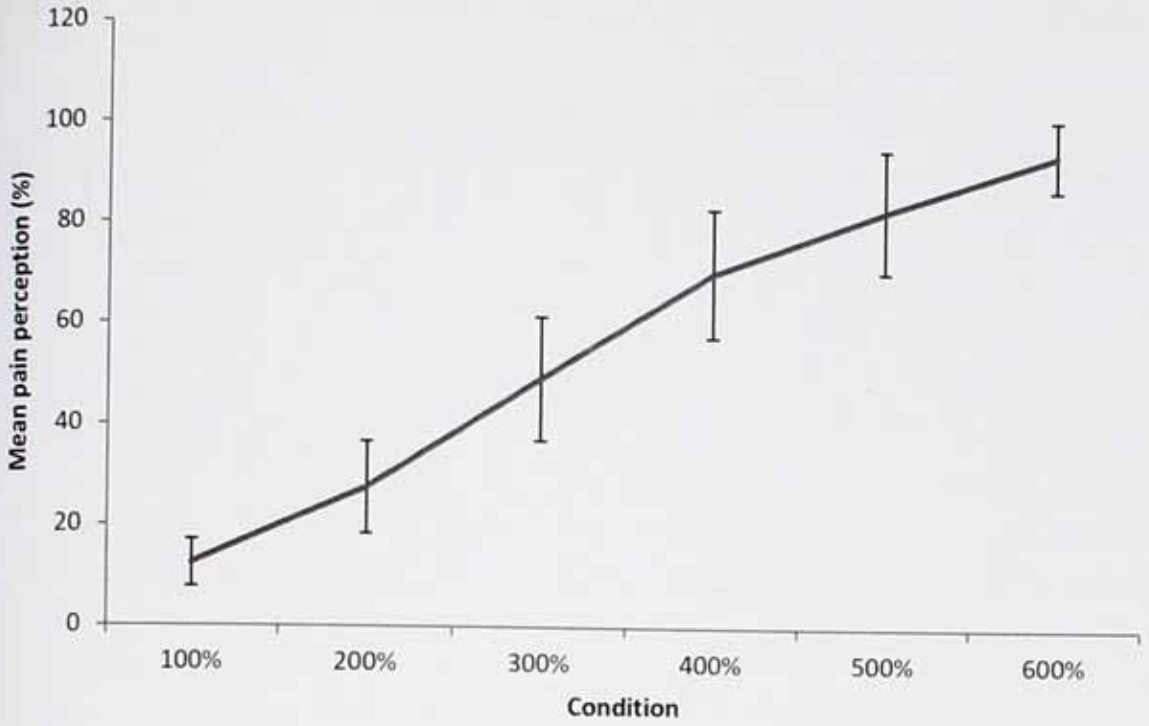


Figure 3.6. Mean pain perception indicated via VAS immediately following each trial.

Further analysis of the 100%-300% conditions was conducted as these were the three conditions that all participants were able to complete. Data were found to be normally distributed in the 100%, ($D(10) = .22$, $p > 0.05$), 200%, ($D(10) = .20$, $p > 0.05$) and 300% condition, ($D(10) = .16$, $p > 0.05$). Therefore a repeated measures ANOVA was used to determine whether there was a significant difference between the three conditions. There was a significant difference in mean pain perception found between conditions ($F = 60.0$, $p < 0.001$). Post-hoc t-tests with Bonferroni correction indicated that there was a significant increase in pain perceived between 100% - 200%, 100%-300% and 200%-300% ($p < 0.05$).

The value (mmHg) indicated to be pain perception threshold was examined in relation to how many trials the participant was able to complete. Table 3.2 indicates that the pain perception thresholds of the ten participants ranged between 25-50mmHg and the maximum value tolerated was 200mmHg. The lowest pressure tolerated as maximum was 120mmHg which was 300% of that participant's pain perception threshold. Participants with the higher pain perception thresholds tended not to be able to continue through as many trials as those with the lower pain perception thresholds.

Table 3.2. Pain perception threshold (mmHg) and maximal pressure exerted for each participant within the study.

Condition successfully completed	Pain perception threshold (mmHg)	Maximal pressure exerted via GPD (mmHg)
300%	40	120
300%	45	135
400%	35	140
400%	50	200
500%	30	150
500%	30	150
500%	25	125
500%	35	175
600%	25	150
600%	30	180

3.5.7 DISCUSSION

The data presented in this study suggest that the GPD can be used to induce pressure pain up to three times the individuals' pain perception threshold before participants are unable to tolerate the stimulus. The VAS data suggests that participants are able to distinguish between the different levels of pain induced and that there appears to be a linear relationship between pressure exerted via GPD and severity of pain perceived by the individual. This data would suggest that 300% pain perception should be the highest severity of pain induced for a study examining the effect of pain severity on muscle performance as this would decrease likelihood of dropout from participants and therefore ensure that data could be collected for all individuals taking part in the study. The VAS data showed that statistically significant differences ($p < 0.05$) existed between the 100-300% conditions. This indicates that these severities of pain are perceived differently enough by participants to be used within the main studies where a variety of levels are required. Another important finding of this study was that the average pressure induced at 300% pain perception threshold was between 75-150mmHg for the ten participants tested. This means that the maximum pressure required for a study examining this level of pain is likely not to exceed 200mmHg which is the pressure at which Bostock et al. (1991) suggested ischaemia can occur. This means that any differences detected in performance would likely result from pain rather than extraneous variables caused by the pressure cuff.

3.6 CHAPTER SUMMARY

The pilot work presented in this chapter determined that the GPD is a reliable method for inducing pain at perception threshold levels both on consecutive days and within a session. Testing found it to be highly reliable at identifying pain perception threshold (ICC = 0.84), which is consistent with Ryan & Kovacic (1966) who also found it to be reliable at inducing pain tolerance (ICC = 0.95). This indicates that the GPD may be an appropriate method of reliable pain induction that can be used for inducing pain at varying intensities (pain perception threshold, present data; pain tolerance, Ryan & Kovacic, 1966), whilst having the additional benefit of being non-invasive and therefore not interfering with the mechanical or physiological functioning of the limb on which pain is induced.

It was decided that only males would be selected for participation due to the influence of the menstrual cycle on pain thresholds (Rollman et al., 2000) which would therefore allow for repeat testing to be conducted should this be required in a later study. The study population consisted of recreational males, defined as those participating in physical activity at least three times a week. This was the sample group chosen as they would have some understanding of their physical capabilities and the novel task would therefore not be too difficult for them, but as this was the first study investigating the effects of pain using the GPD in this way it wasn't considered necessary to use an athlete population who are harder to recruit. Pilot testing found these participants to be able to distinguish pain perception threshold level and withstand reasonably high levels of pain which makes them suitable for studies involving pressures higher than pain perception threshold.

The pilot studies conducted have found that the modified GPD is reliable both intra- and inter-session which means that it can be used in studies that require repeat visits to the lab, or multiple trials within a session. This makes it a highly flexible tool in future research and increases the potential scope of investigations that can utilise it. It has also been determined that the optimal positioning of the cleat of the GPD is mid-way between medial condyle and medial malleolus as this is the most sensitive part of the lower limb. Using the GPD at this point reduces the need to inflate the cuff to high pressures and thereby reduces risk of confounding variables associated with ischeamia. A further finding from the pilot work was that pain could successfully be induced at varying intensity, with 300% of pain perception threshold determined to be the highest pressure that all participants could withstand. The studies conducted within this chapter will form the basis for the protocol used throughout this thesis.

4 THE INFLUENCE OF PAIN PERCEPTION THRESHOLD INDUCED VIA GROSS PRESSURE DEVICE ON NEUROMUSCULAR PERFORMANCE IN A MAXIMAL ISOMETRIC KNEE EXTENSION TASK

4.1 INTRODUCTION

The mechanisms underpinning pain-related reductions in muscular performance have been suggested to include information processing components where pain sensation is given a high supra-spinal priority due to it being an implicit survival mechanism (Price, 2000). This may result in a loss of performance when more than one stimulus is being processed concurrently (e.g. task demands) as the pain sensation will be afforded greater priority than the task being completed. In an effort to explain how this could influence muscle performance specifically, Lund et al. (1991) proposed the pain adaptation model whereby pain was thought to cause a decrease in muscle force or velocity. It was suggested that EMG activity of the agonist reduced and EMG activity of the antagonist increased by way of a protective response limiting movement around a joint (Bonifazi et al., 2004).

Patterns of EMG activity found in a number of studies have supported the pain adaptation model (Arendt-Nielsen et al., 1996; Stohler et al., 1988); however evidence for this is equivocal. Some studies have found that a reduction in agonist EMG activity may not necessarily be accompanied by an associated increase in antagonist EMG activity, instead there is a reduction in antagonist EMG activity detected (Ervilha et al., 2004). This suggests that the relationship between pain and muscle performance is more complex and therefore greater flexibility is required when trying to establish mechanisms. To address this issue, Hodges & Tucker (2011) proposed a theory in which a non-stereotypical response to pain was advocated. Within the adaptation to pain theory the nervous system is thought to have a range of options at its disposal in order to achieve protection of the painful area which may involve increased, decreased or redistributed activity involving more complex neural processes than those proposed by existing theories (Hodges & Tucker, 2011) but which currently remain unexplained and require further investigation (see Chapter 2).

The literature suggests that when a pain stimulus is present, muscular recruitment and therefore performance can be compromised (Arendt-Nielsen et al., 1996; Bonifazi et al., 2004; Ervilha et al., 2004, 2005; Hodges & Tucker, 2011; Stohler et al., 1988) however, the method for inducing pain has been highly invasive via saline injections that do not enable the level of pain induced to be easily controlled. Furthermore, injecting fluid into the muscle may compromise normal biomechanical properties and physiological processes important to force production.

Pilot work outlined in Chapter 3 established that the use of a GPD is an instrument that can be used to reliably induce pain in consecutive trials at perception threshold level (CV = 12.5; ICC = 0.84) and at varying intensity. It was shown not to cause any damage to the site at which pain was induced, making it a safe tool for use in research particularly where multiple trials are required. Therefore, the aim of this study was to determine whether pain induced via GPD at pain perception threshold affects muscular performance during an isometric knee extension task.

4.2 RESEARCH QUESTION

Does pain induced via GPD at perception threshold level influence neuromuscular performance in a maximal isometric knee extension task?

4.3 METHOD

4.3.1 PARTICIPANTS

In order to ensure an adequate participant population size for the study to reach statistical power, variance was calculated from the first 20 participants of this sample group. A number of variables will be measured through this study (force output, EMG activity of the vastus lateralis and semitendinosus), however the primary research question is whether there is a difference between control and pain in relation to force produced. The first 20 participants were used as a pilot in order to determine the mean difference and standard deviation of difference in order to calculate the sample size required for a power of 90%. For the first 20 participants an estimate of mean difference was 10.0 and SD of difference 16.7. This would give a required number of cases of 31 participants to achieve a 90% power. This means that 90% of studies would be expected to yield a statistically significant effect assuming it exists, rejecting the null hypothesis that the population mean difference is 0.00. Based upon this, thirty three recreationally active male participants (mean ± SD; age = 31.9 ± 12.8 yr, height = 1.8 ± 0.07 m, mass = 85.4 ± 12.4 kg) were recruited via poster (see Appendix 3) and email (see Appendix 4) advertisement. As was the case in all studies, participants received a participant information sheet (see Appendix 5) at least 24 hours prior to the study and were required to give written informed consent (see Appendix 1) prior to participation. The participants were free from any lower limb injury and were asked to refrain from any intense exercise or stimulant use for 24 hrs prior to testing. Ethical approval was granted from the School of Health's Ethics Committee in accordance with the declaration of Helsinki.

4.3.2 EQUIPMENT AND MEASURES

An isokinetic dynamometer (Biodex System 3 Pro, IPRS, Suffolk, UK) was used to measure isometric knee extensor moment, while skin-mounted bi-polar double differentiated active electrodes (model MP-2A, Linton, Norfolk, UK) measured EMG activity of the vastus lateralis (VL) and semitendinosus (ST) muscles. Joint moment and EMG data were directed to a high-level transducer (model HLT100C, Biopac, Goleta, CA) before analog-to-digital conversion (model MP150 Data Acquisition, Biopac). The data were then directed to and stored on a personal computer running AcqKnowledge software (version 4.0, Biopac). To induce pain, a gross pressure device was designed using a modified sphygmomanometer cuff (model DM304, Mercurial sphygmomanometer Bigger Case, China), a curved plastic shin pad (107 x 175 mm) with a removable semicircular metal cleat (80mm length, 23mm radius) and Velcro attachment to the inner surface of the shin pad positioned over the tibia (see Chapter 3).

4.3.3 PROCEDURE

Participants visited the laboratory on two separate occasions completing a familiarisation trial at least 24 hours prior to the experimental trial. On both occasions, the participants performed a five minute warm up on a Monark cycle (824E) at 60 rpm against a 1 kg load producing a power output of 60 W. The familiarisation trial included measuring and establishing pain perception threshold and practicing explosive and ramped MVC's on the isokinetic dynamometer until each MVC performance was consistently within 5% to ensure that maximum values were being achieved within each trial. The ramped contraction, where MVC was achieved through a gradual increase over 5 seconds, was used to determine the EMG-joint moment relationship (Kay & Blazevich, 2009), while the explosive, rapid MVC performed in the shortest time was used as a measure of maximal muscular performance. Physiological research has shown that the level of force produced during a maximum voluntary contraction (MVC) decreases during both experimentally induced muscle pain (Graven-Nielsen et al., 1997; Ervilha et al., 2005) and non-experimentally induced muscle pain (Backman et al., 1988; Suzuki & Endo, 1983), however during sub maximal isometric contractions pain was not found to be a limiting factor in performance (Ashton-Miller et al., 1990), therefore MVCs would be used throughout the current research.

During the experimental trial, pain perception threshold was measured and recorded prior to participants performing a ramped isometric MVC. Participants then underwent one of three experimental conditions (pain, cuff or control) where they performed three explosive isometric MVC's on an isokinetic dynamometer with a two minute rest period between contractions (see figure 4.1). All three conditions were performed during the trial in a

randomised, counterbalanced manner. The peak isometric knee extensor moment (force) and the corresponding peak EMG activity from the vastus lateralis and semitendinosus from the three trials were used for analysis. The peak values were used as it was felt that an average value across the three trials in each condition may not be reflective of the participant's maximal ability and may cause a skewing of data if the participant under- or over- performed in any of the trials. In order to avoid skewing as far as possible, when analysing the results any trials that was more than 5% different from the other trials in that condition was excluded from analysis as this was considered to be an outlier and not a true MVC.

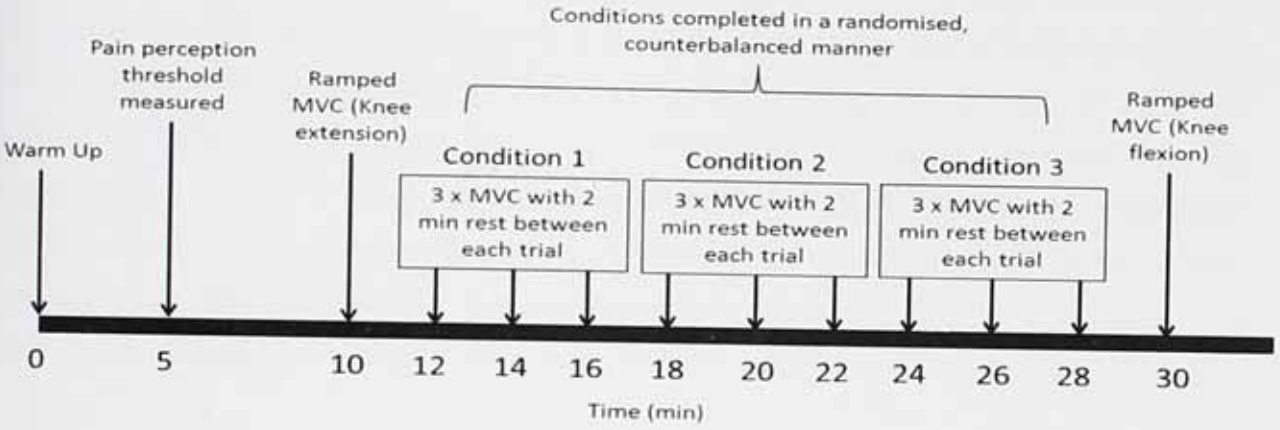


Figure 4.1. Timeline of protocol undertaken by each of the participants during the experimental trials.

4.3.4 EXPERIMENTAL CONDITIONS

Three conditions were performed within the trial; pain, cuff and control. During the pain condition, the GPD was strapped to the lower limb with the cleat positioned over the tibia one third from medial tibial epicondyle to medial malleolus. Pilot testing revealed that this site was the most sensitive area on the tibia accessible when the leg was positioned on the isokinetic dynamometer (see chapter 4). The GPD was then inflated at 5 $mmHg \cdot s^{-1}$ to the previously established pain threshold, considered as the lowest experience of pain that a participant would recognise (Loeser & Treede, 2008). The participant was then instructed to perform the MVC trials whilst the cuff remained inflated. Immediately following each MVC, pressure from the cuff was released; however the GPD remained in position between each trial to ensure reliability of the position of the device. The cuff condition was identical with the exception that the cleat was removed from the GPD, therefore no pain was induced, while the control condition followed the same protocol but without use of any parts of the GPD.

4.3.5 ISOMETRIC JOINT MOMENT

Participants sat upright in the chair of an isokinetic dynamometer (Biodex System 3 Pro) with the knee positioned at a 90° angle (180° full extension) with the lateral femoral epicondyle aligned to the centre of rotation of the dynamometer. The foot and shank were supported by Velcro strapping attached to the lever arm to hold the lower limb in position (see Plate 4.1). The upper body was also strapped into position across the chest, pelvis and femur to ensure reliable subject positioning. Participants were instructed to sit with their hands behind their backs for each trial as pilot testing indicated this position resulted in the most reliable joint moment data (see Plate 4.2). Participants were given a safety release valve to hold during the trials which when activated would deflate the GPD. During the MVC trials, joint moment data were exported from the dynamometer to a high-level transducer (model HLT100C, Biopac) before analogue-to-digital conversion at a 1000-Hz sampling rate (model MP150 Data Acquisition, Biopac). The data were then directed to a personal computer running AcqKnowledge software (version 4.0, Biopac) and filtered with a double-pass 6-Hz Butterworth low-pass filter. Data were normalised as a percentage of the peak isometric joint moment recorded during the ramped MVC. The normalised joint moment (%MVC) from the trials that produced the greatest joint moment were used as a measure of muscular performance.

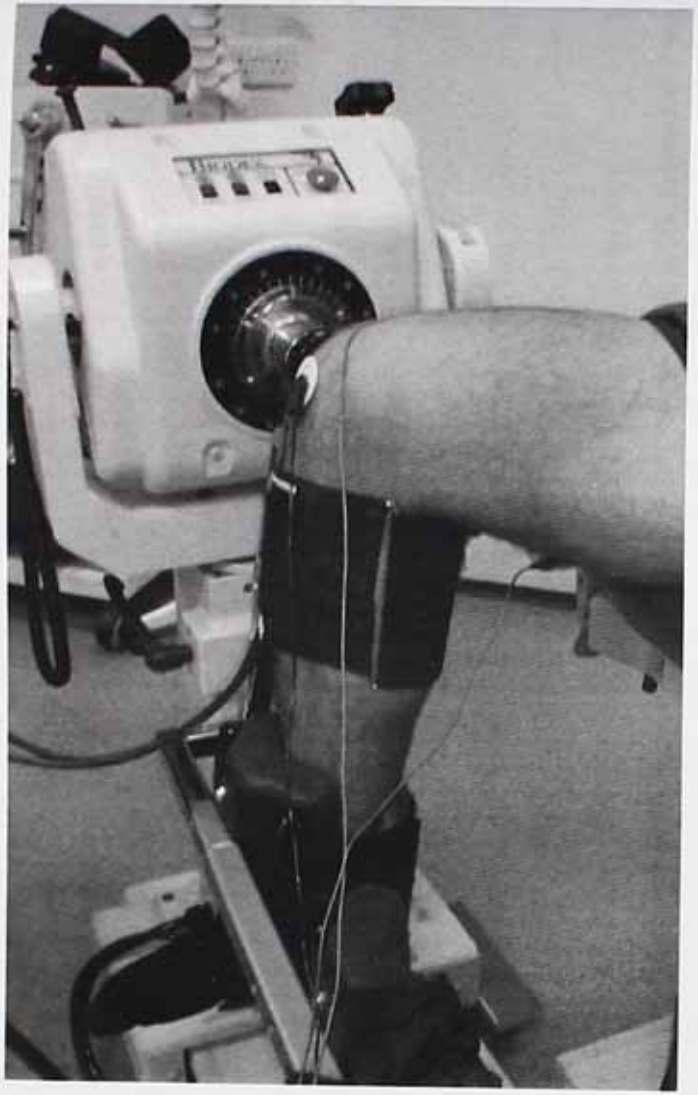


Plate 4.1. Knee positioning during the MVC trials on the isokinetic dynamometer, with GPD positioned one third from medial malleolus to medial epicondyle.

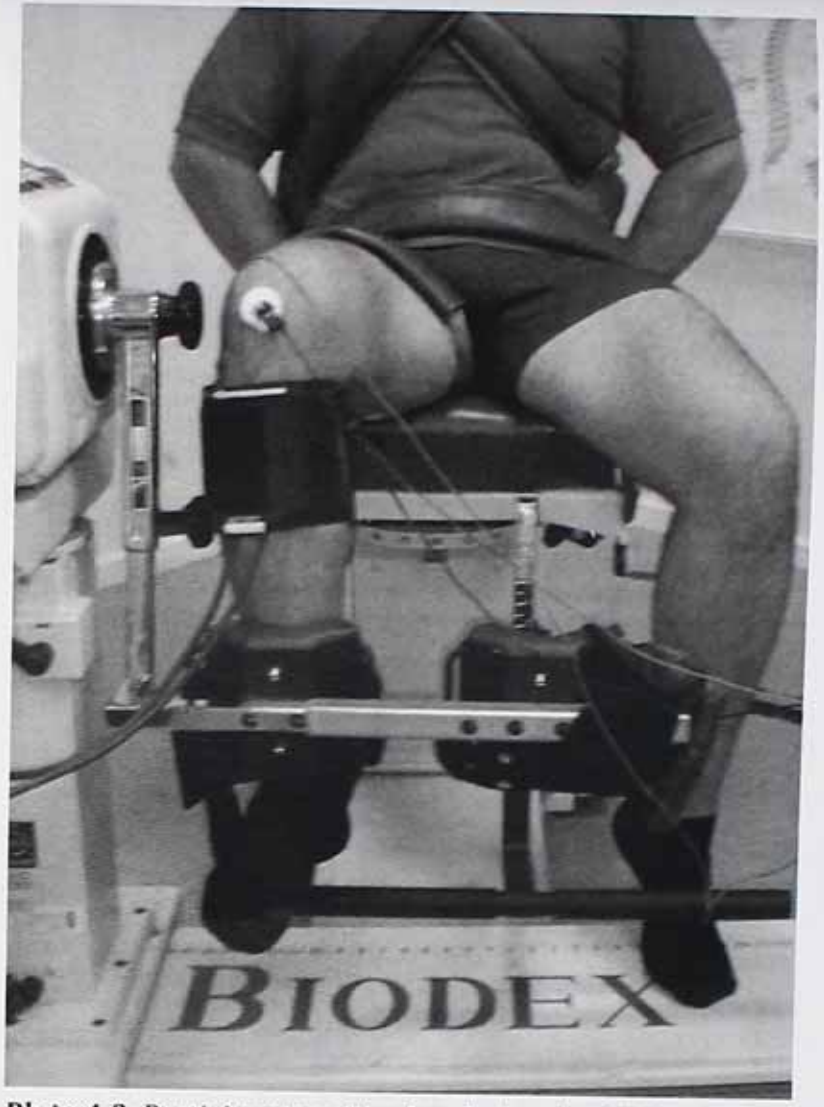


Plate 4.2. Participant positioning during the MVC trials. Dominant leg is positioned as shown in plate 4.1. Whilst upper body is strapped in place and hands held behind the participants back.

4.3.6 EMG RECORDING & PROCESSING

The skin over each electrode site was shaved, lightly abraded and then cleansed with ethanol in order to reduce skin resistance and to minimise the risk of infection (De Luca, 1997). Skin-mounted bi-polar double-differentiated active electrodes (model MP-2A, Linton) with a 12 mm diameter, a fixed inter-electrode distance of 18 mm and central bar of 12 mm by 3 mm, were then positioned over the central portion of the muscle bellies of the VL and ST. The positioning of the electrode on each muscle followed the general direction of the muscle fibres. A ground electrode was positioned over the medial aspect of the patella of the same limb. During the MVC trials, EMG activity was amplified (gain = 300, input impedance = 10 GΩ, common mode rejection ratio ≥100 dB at 65 Hz) and directed to a high-level transducer (model HLT100C, Biopac) before analog-to-digital conversion at a 1000-Hz sampling rate (model MP150 Data Acquisition, Biopac). The signal was then directed to a personal computer running AcqKnowledge software (version 3.8.2), where it was filtered using a 20- to 500-Hz band-pass filter. The filtered signal was converted to root-mean-squared (RMS) EMG with a 1000-ms sample window and normalised as a percentage of the peak amplitude recorded during the ramped MVC. The normalised peak EMG amplitude (%MVC) from the trials that produced the greatest joint moment was used as a measure of neuromuscular activity.

4.3.7 DATA ANALYSIS

Data were analysed using the SPSS statistics software package (version 17.0) and reported as mean ± SD where normally distributed, and as median, IQR and range where non-parametric. All data were initially examined for normality using the Kolmogorov-Smirnov test before further analyses were performed. A repeated measures design was used for this study, therefore either a one way repeated measures ANOVA, or Friedman's ANOVA would be used for data analysis. The ANOVA is a robust statistical test when used with equal sample sizes (Field, 2009) thereby reducing the likelihood of Type I error. It does assume that distributions within groups are normal and that variances within experimental conditions are fairly similar, which they would be expected to be in this study.

Post-hoc t-tests with Bonferroni correction were used, where statistical significance was established. Statistical significance was accepted at $p < 0.05$. In each condition the peak MVC trial was selected for analysis. All values were normalised to the ramped contractions and reported as percentages. Initial analysis of the data from the 33 participants revealed an outlier in the EMG activity for one participant in the latter two of the three conditions, which suggests that the electrode position or adhesion to the skin may have been compromised and the data

were unreliable and were therefore removed from subsequent analysis leaving 32 subjects' data for analysis.

4.4 RESULTS

Due to the number of hypotheses being examined within this study, the results have been divided up for ease of understanding into; isometric knee extensor moment, EMG of the vastus lateralis, and EMG of the semitendinosus. Each section first reports normality of the data being tested (using the Kolmogorov-Smirnov test) and then any significant differences ($p < 0.05$) found as well as showing where those differences lie where appropriate.

4.4.1 ISOMETRIC KNEE EXTENSOR MOMENT (FORCE)

Normality was confirmed in all three conditions; pain, ($D(32) = 0.66$, $p > 0.05$), cuff, ($D(32) = 0.89$, $p > 0.05$), and control, ($D(32) = 0.89$, $p > 0.05$). An analysis of variance (ANOVA) with repeated measures was therefore used to test the following:

H1: There is a difference in knee extensor moment in an isometric knee extension task when pain perception threshold is induced via GPD.

H0: There is no difference in knee extensor moment in an isometric knee extension task when pain perception threshold is induced via GPD.

A significant difference in force produced between the three conditions was found ($F = 7.96$; $p < 0.05$), with Bonferroni-adjusted post-hoc t-tests indicating that there was a significant difference between the pain and cuff condition, and pain and control ($p < 0.05$), but no significant difference between cuff and control ($p > 0.05$). Mean force (see Figure 4.2) was 8.9% lower in the pain condition (101.0 ± 2.5% MVC) than control (110.9 ± 2.5% MVC) and 7.2% lower than the cuff condition (108.9 ± 3.1% MVC).

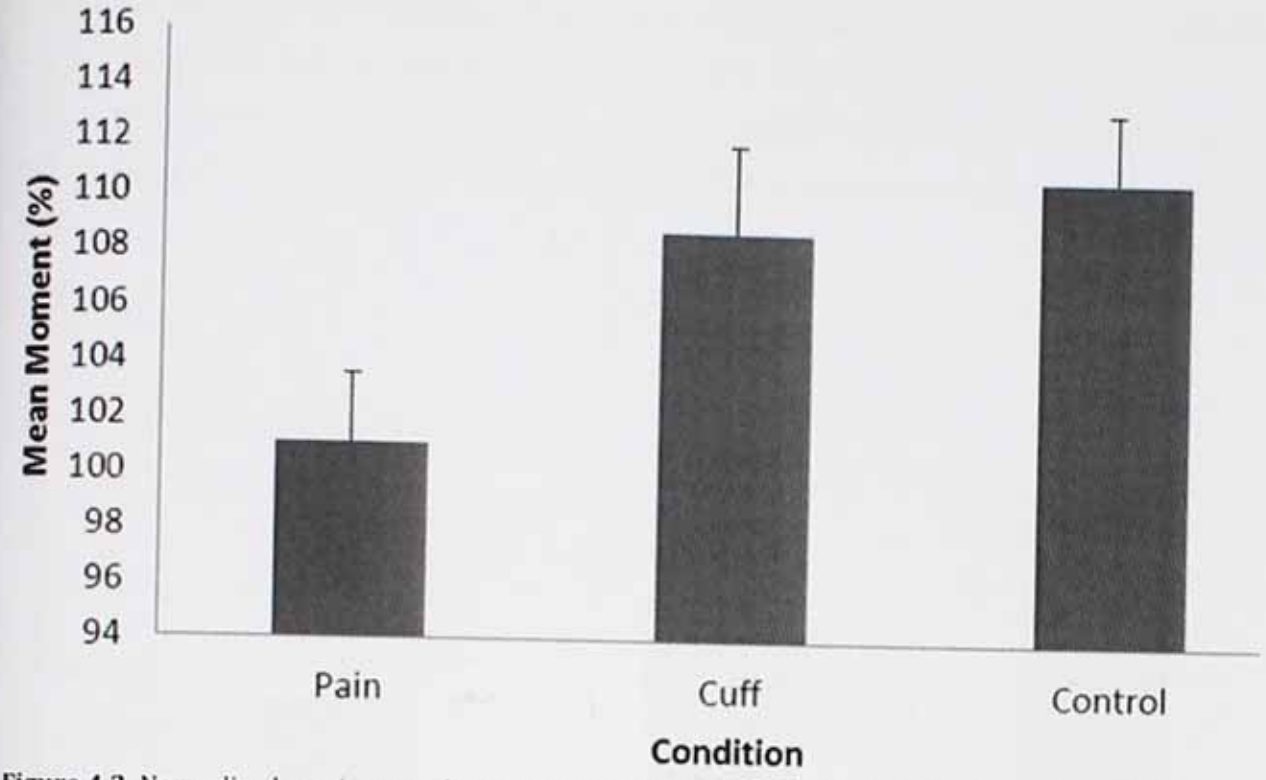


Figure 4.2. Normalised maximum voluntary isometric contractions produced in the pain, cuff and control condition. Data are presented as mean ± SD (n=32).

Table 4.1. Statistical differences in maximum voluntary isometric contractions produced between the three experimental conditions where significance is accepted at $p < 0.05$.

	Pain	Cuff	Control
Pain	-	p = 0.017	p = 0.002
Cuff	p = 0.017	-	NS
Control	p = 0.002	NS	-

NS = No significant difference

4.4.2 EMG VASTUS LATERALIS

Data were found to be normally distributed in the pain, ($D(32) = 0.08$, $p > 0.05$), cuff, ($D(32) = 0.06$, $p > 0.05$) and control condition, ($D(32) = 0.06$, $p > 0.05$). An analysis of variance with repeated measures (ANOVA) was therefore used to test the following:

H1: There is a difference in VL EMG activity in an isometric knee extension task when pain perception threshold is induced via GPD.

H0: There is no difference in VL EMG activity in an isometric knee extension task when pain perception threshold is induced via GPD.

A repeated measures ANOVA revealed a significant difference in VL EMG activity between the three conditions ($F = 6.73$, $p < 0.05$) with post-hoc t-tests identifying that the difference was

located between pain and control ($p < 0.05$). No difference was found between pain and cuff ($p > 0.05$) and control and cuff ($p > 0.05$). In the presence of pain there was a 12.8% reduction in EMG activity (see Figure 4.3) between pain (97.08 ± 3.92% MVC) versus control (111.37 ± 5.15 % MVC), and a 9.7% reduction during the pain condition in comparison to cuff (103.43 ± 4.69 % MVC).

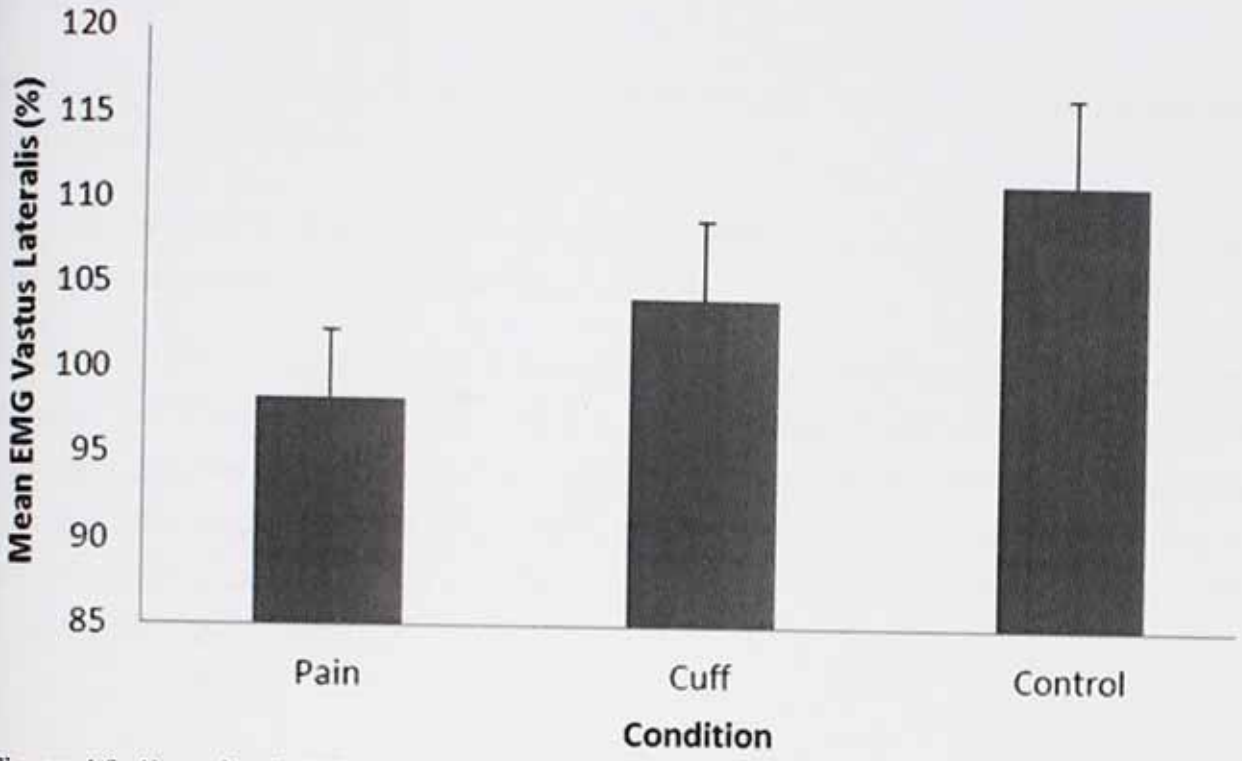


Figure 4.3. Normalised EMG activity recorded in vastus lateralis during the pain, cuff and control condition. EMG recordings corresponding to the peak MVC produced over three trials in each condition were selected. Data are presented as mean ± SD (n=32).

Table 4.2. Statistical differences in normalised EMG activity of the vastus lateralis recorded between the three experimental conditions where significance is accepted at $p < 0.05$.

	Pain	Cuff	Control
Pain	-	NS	p = 0.002
Cuff	NS	-	NS
Control	p = 0.002	NS	-

NS = No significant difference

4.4.3 EMG SEMITENDINOSUS

The assumptions of normal distribution were violated in the cuff ($D(32) = 0.20$, $p < 0.05$), and control ($D(32) = 0.19$, $p < 0.05$), conditions. A log transformation was used, whereby normality was confirmed in pain, ($D(32) = 0.10$, $p > 0.05$), cuff, ($D(32) = 0.10$, $p > 0.05$), and control, ($D(32) = 0.11$, $p > 0.05$). An analysis of variance (ANOVA) with repeated measures was therefore used to test the following:

H1: There is a difference in ST EMG activity in an isometric knee extension task when pain perception threshold is induced via GPD.

H0: There is no difference in ST EMG activity in an isometric knee extension task when pain perception threshold is induced via GPD.

Repeated measures ANOVA found there to be a significant difference in ST EMG activity between the three conditions ($F = 7.03$, $p < 0.05$). Post-hoc t-tests revealed a significant difference between pain and control ($p < 0.05$) but no significant difference between pain and cuff ($p > 0.05$) and cuff and control ($p > 0.05$). In the presence of pain there was found to be a 22.3% reduction in EMG activity (see Figure 4.4) (6.84 ± 0.76% MVC) in comparison to the control (8.80 ± 0.95% MVC).

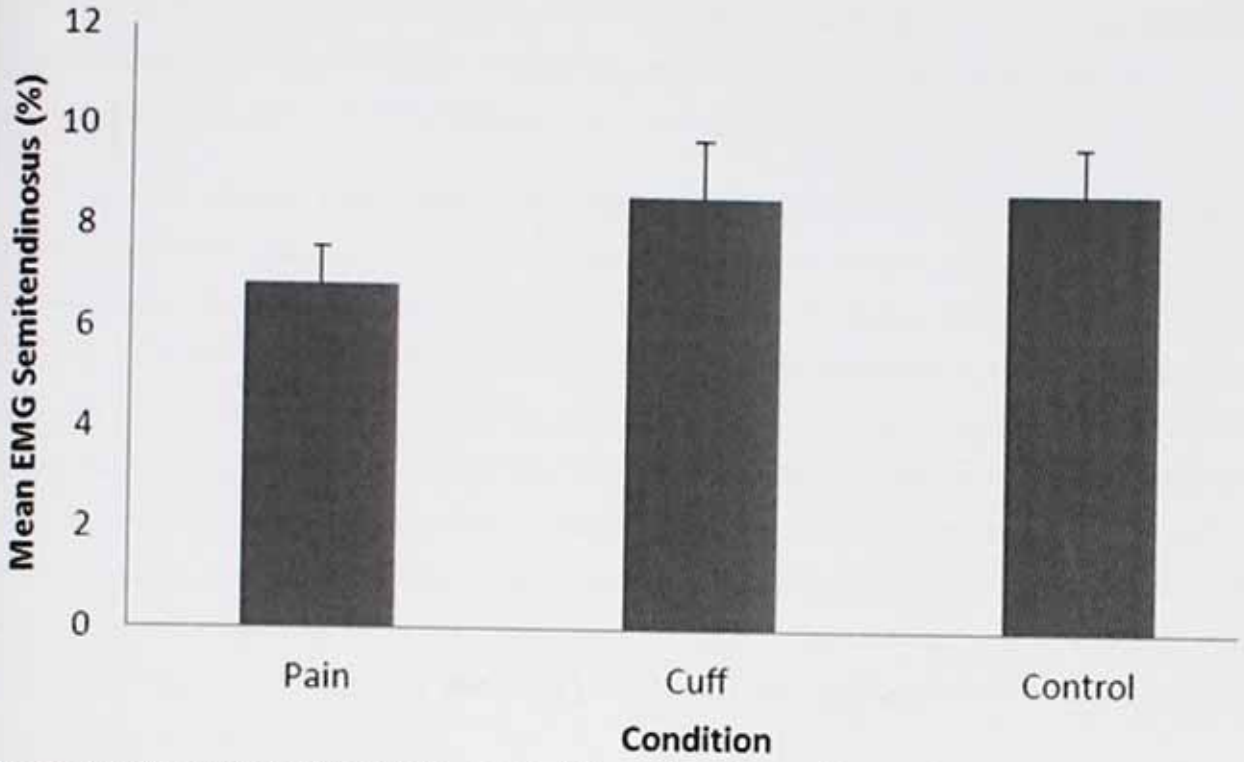


Figure 4.4. Normalised EMG activity recorded in semitendinosus during the pain, cuff and control condition. EMG recordings corresponding to the peak MVC produced over three trials in each condition were selected. Data are presented as mean ± SD (n=32).

Table 4.3. Statistical differences in normalised EMG activity of the semitendinosus between the three experimental conditions where significance is accepted at $p < 0.05$.

	Pain	Cuff	Control
Pain	-	NS	p = 0.002
Cuff	NS	-	NS
Control	p = 0.002	NS	-

NS = No significant difference

To establish the EMG:moment relationship, a bivariate correlation was conducted between EMG activity in the vastus lateralis and force produced during the ramped contraction trial for 10 participants. Strong correlations were established between mean EMG VL and mean joint moment ($r = 0.992$; $p < 0.01$) with individual participants also showing strong correlations ($r = 0.703 - 0.999$; $p < 0.01$). The ratio of EMG activity from VL to ST was also examined. A repeated measures ANOVA showed that there was no significant difference between conditions ($F = 1.44$, $p < 0.05$).

4.5 DISCUSSION

The major finding from this study was that pressure pain induced by the GPD significantly reduced muscular performance during an isometric knee extension task as a reduction in peak

force (8.9%) during the MVC was detected. This is in agreement with previous studies that also reported reductions in performance following pain induced via hypertonic saline injections (Farina et al., 2004, 2005; Graven-Nielsen et al., 1997; Ervilha et al., 2004, 2005).

Several possible mechanisms could explain the reduced force output in these studies including reduced neuronal activity, ischaemia or changes to normal physiological processes following saline injection to the target muscle. The methods and data from the cuff condition in the present study have removed ischaemia as a possible mechanism. A concern with using pressure pain induced with a cuff inflated around the limb was that this may result in ischaemia with potential implications for force production. However, this is unlikely for several reasons: a) the cuff was distal to the target muscle and therefore unlikely to compromise blood flow; b) while ischaemia can be induced at around 200mmHg (Bostock et al., 1991), the mean pressure exerted during trials was far below this intensity (58.8 ± 33.8 mmHg); and c) the cuff was inflated for only a short duration (5-10s). This makes it very unlikely that any losses in force could be due to ischaemia.

This study also found that, unlike the previous studies employing saline injections to induce pain, changes to normal physiological processes are unlikely as the pain stimulus was non-invasive and removed from the target muscle. Interestingly the present results also detected a concomitant reduction in agonist EMG activity, which would suggest that the reduction in force might be attributed to reduced neuromuscular activity. A correlation in reductions between peak VL EMG and moment was detected ($r = 0.387$, $p < 0.05$) when pain was induced. This is in agreement with previous research using dynamic tasks where EMG amplitude and MVC force decreased with a pain stimulus (Backman et al., 1988; Graven-Nielsen et al., 1997; Suzuki & Endo, 1983) thereby suggesting that reductions in neuromuscular performance are likely to be as a result of neuronal inhibition.

The pain adaptation model (Lund et al., 1991) attempts to explain reductions of force in the presence of a pain stimulus through a reordering of neuromuscular agonist-antagonist activity, which suggests a reduction in agonist activity and an increase in antagonist activity. The present findings support this model as concomitant reductions in force and agonist EMG activity were detected coupled with increases in EMG activity reported in the ST. The decrease in force output and concomitant reduction in agonist muscle activity during the pain condition, suggests that there may be central processes limiting performance. In order for movement to occur a message needs to be sent from the brain to the relevant muscles to instigate that movement. In the presence of pain the level of activation of the motor units within the muscles may be influenced either at a central or spinal level. There could be an interaction effect

between descending neural drive and efferent signals which may account for limitations in motor output.

The mechanisms by which pain modulates behaviour are still not fully understood, however the Gate Control theory (Melzack & Wall, 1965) suggests the neural pathway by which a pain stimulus reaches the brain from the peripheral nerve to the spinal cord is subject to modulation by both intrinsic neurones and controls emanating from the brain (Dickensen, 2002). As every synapse allows opportunity for modulation this means that at any given point between the peripheral site at which the stimulus was detected, right up until it is translated in the brain, there can be any number of factors that can influence it. Alternatively, the presence of a painful stimulus may alter the message sent from the brain, thereby limiting motor output at a higher level. An explanation for why this may happen lies in the high supra-spinal priority that pain is afforded as this is thought to be implicit in order to survive (Price, 2000). As a consequence, other information processing requirements occurring at the same time (for example, motor output) may be reduced in priority and performance changes may result (Lorenz et al., 1997b; Lorenz & Bromm, 1997).

Melzack (2001) put forward the idea of a 'neuromatrix' suggesting that pain is an integrative experience. In contrast to previous thinking, the neuromatrix contends that the loops allow parallel processing within which the cyclical processing and synthesis of nerve impulses develop a characteristic pattern; labelled the neurosignature. The neurosignature is determined by multiple influences and past experiences, not just somatic sensory input, therefore the context in which a person is being asked to perform a task may influence their reaction to the pain stimulus. In this case, performance in the isometric knee extension task was compromised as a result of a pain stimulus being induced in the lower leg. The nociceptive signals travelling from the site of the pain to the brain could in some way interact with the efferent signals controlling the task and therefore influence performance output on a spinal level, or there could have been a more complex process occurring within the brain in which past experience of pain dictated the level of activation being sent from the brain to the muscles; thereby limiting motor output at a higher level.

4.6 CHAPTER SUMMARY

This study has demonstrated that the presence of pain decreases the level of isometric force produced by the knee extensors with a concomitant reduction in EMG activity of the VL muscle. Unlike previous studies where saline injections induced pain in the target muscle, the non-invasive pressure cuff employed in the present study enabled both a reliable intensity of pain to

be administered and the pain to be remote from the target muscle in which muscular performance was measured. This indicates that the presence of pain compromises normal muscular functioning despite the target muscle remaining intact. The major benefit of this method of pain induction is that there are no influences upon muscle physiology or biomechanics as a result of the methods employed, removing these as possible mechanisms underpinning the losses in force. Higher level, spinal or supra-spinal control mechanisms may be compromised as a result of pain, which would explain the reduced EMG activity detected. However, the present methods do not enable the location of the impairment to be detected and further research could examine where this exists. Similarly, while pain perception threshold significantly reduces muscular performance, the severity of pain stimulus should be examined to determine whether intensity influences force losses.

5 THE INFLUENCE OF PRESSURE PAIN SEVERITY ON NEUROMUSCULAR PERFORMANCE IN A MAXIMAL ISOMETRIC KNEE EXTENSION TASK

5.1 INTRODUCTION

Experimental muscle pain has been found to decrease maximal muscular performance (Ervilha et al., 2004, 2005; Graven-Nielsen et al., 1997) even when severity of pain induced was at perception threshold level (see chapter 4). This suggests the presence of pain across varying levels influences performance, but the relationship between severity of pain and muscle performance has not received a great deal of attention. The previous chapters have demonstrated that pain can be induced reliably via GPD at pain perception threshold level (CV = 12.5: ICC = 0.84, Chapter 3) and is associated with a decrease in maximal muscular performance (chapter 4). This is in agreement with previous research in which pain has been found to have debilitating effects on muscular function and physical performance in day-to-day tasks (Young, 1993) with the association found to be greater as severity of pain increases (Onder et al., 2006). However, the literature focuses upon populations experiencing chronic pain, or groups where small losses of function are likely to have more damaging consequences (such as older adults) and as such there is little evidence of the effects of severity of experimental pain in a healthy sample.

Farina et al. (2004) conducted an experiment in which they administered repeated hypertonic saline injections to induce an increasing level of pain in a sub-maximal isometric task. Perceived pain intensity was found to be inversely proportional to motor unit firing rate, thereby supporting the pain adaptation model (Lund et al., 1991). However the method of pain induction was highly invasive and could not be controlled for severity in comparison to individual thresholds but could only be reported as a perception retrospectively (Wing et al., 2011a). The fact that pain severity could only be increased through accumulation of the hypertonic saline solution also means that conducting experiments in a randomised manner is problematic and thus limits the usefulness of the technique to investigate mechanisms of pain-related changes. The highly controllable nature of the GPD allows for baseline measures to be obtained and used to quantify different pain severities for the purpose of experimental trials. It was the aim of this study to examine the influence of the severity of pain induced by a non-invasive GPD on muscular performance in the lower limb.

5.2 RESEARCH QUESTION

Does severity of pain induced via a GPD influence neuromuscular performance in a maximal isometric knee extension task?

5.3 METHOD

5.3.1 PARTICIPANTS

Thirty-one recreationally active male participants (mean ± SD; age = 32.7 ± 12.3yrs, height = 180.2 ± 7.9m, mass = 85.3 ± 12.1kg) volunteered for the study after giving written informed consent (see Appendix 1). The participants were free from any lower limb injury and were asked to refrain from any intense exercise or stimulant use for 24 hrs prior to testing. Ethical approval was granted from the School of Health's Ethics Committee in accordance with the declaration of Helsinki.

5.3.2 PROCEDURE

Equipment, measures and experimental protocol were as described in chapter 4 with all participants visiting the lab on two separate occasions for a familiarisation session and then experimental trial. Each participant undertook three MVCs in three conditions in a counterbalanced and randomised trial. The peak MVC in each trial was selected for measurement.

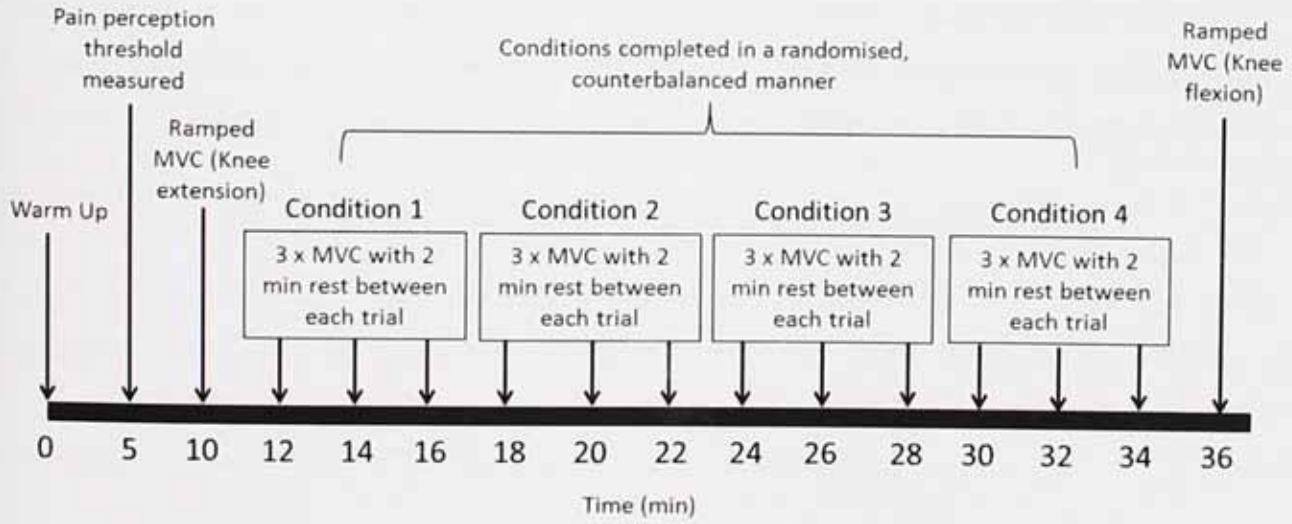


Figure 5.1. Timeline of protocol undertaken by each of the participants during the experimental trials.

5.3.3 EXPERIMENTAL CONDITIONS

Four conditions were performed within the trial in which a control, 100%, 200% or 300% of the participants' previously determined pain perception threshold was induced using the GPD. The participant was then instructed to perform the MVC trials. Immediately following each MVC, pressure from the cuff was released, while the GPD remained in position between each trial to ensure reliability of the position of the device.

5.3.4 DATA ANALYSIS

In each condition the peak MVC trial was selected for analysis. All values were normalised to the ramped contractions and reported as percentages; all data are reported as mean ± SD where normally distributed, and as median, IQR and range where non-parametric. Data within each condition were examined for normality using the Kolmogorov-Smirnov test. Where the raw data were not normally distributed appropriate data transformation was used. Where no suitable transformation could be found, non-parametric tests were used. Post-hoc t-tests with Bonferroni correction were used where statistical significance was established. Statistical significance was accepted at $p < 0.05$ (see chapter 4 for full explanation of data analysis).

5.4 RESULTS

Due to the number of hypotheses being examined within this study, the results have been divided up for ease of understanding into; isometric knee extensor moment, EMG of the vastus lateralis, EMG of the semitendinosus and pain perception (VAS). Each section first reports normality of the data being tested (using the Kolmogorov-Smirnov test) and then any significant differences ($p < 0.05$) found as well as showing where those differences lie where appropriate.

5.4.1 ISOMETRIC KNEE EXTENSOR MOMENT (FORCE)

Assumptions of normality were violated in the control, ($D(31) = 0.16$, $p < 0.05$), and 300% condition, ($D(31) = 0.17$, $p < 0.05$). Normality was confirmed in the 100%, ($D(31) = 0.10$, $p > 0.05$), and 200% condition, ($D(31) = 0.12$, $p > 0.05$). No suitable data transforms could be found; therefore a Friedman's ANOVA was used to test the following:

H1: There is a difference in knee extensor moment in an isometric knee extension task when pain of varying severity is induced via GPD.

H0: There is no difference in knee extensor moment in an isometric knee extension task when pain of varying severity is induced via GPD.

A significant difference in mean force output was found between all four conditions, ($\chi^2(3) = 41.28$, $p<0.05$). Wilcoxon tests were used to determine where this difference existed. A Bonferroni correction was applied and so all effects are reported at a .0125 level of significance. A significant difference was found between control and 100% ($z = -3.74$, $p = 0.000$), 200% ($z = -4.31$, $p = .000$), and 300% ($z = -4.53$, $p = .000$). Further significant differences were detected between 100%-200% ($z = -2.69$, $p = .007$) and 100%-300% ($z = -2.78$, $p = .005$). However, no significant difference was detected between 200%-300% ($z = -.61$, $p = .544$). In comparison to control (median = 111.6, IQR = 30.82, range = 118.3% MVC) median force was 6.2% lower in the 100% condition (median = 104.7, IQR = 72.8, range = 22.1% MVC), 9.5% lower in the 200% condition (median = 101.0, IQR = 24.8, range = 90.1% MVC) and 9.7% lower in the 300% condition (median = 100.8, IQR = 28.8, range = 74.8% MVC). In comparison to the 100% condition median force was reduced by 3.5% in the 200% condition, and 3.7% in the 300% condition (see Figure 5.2).

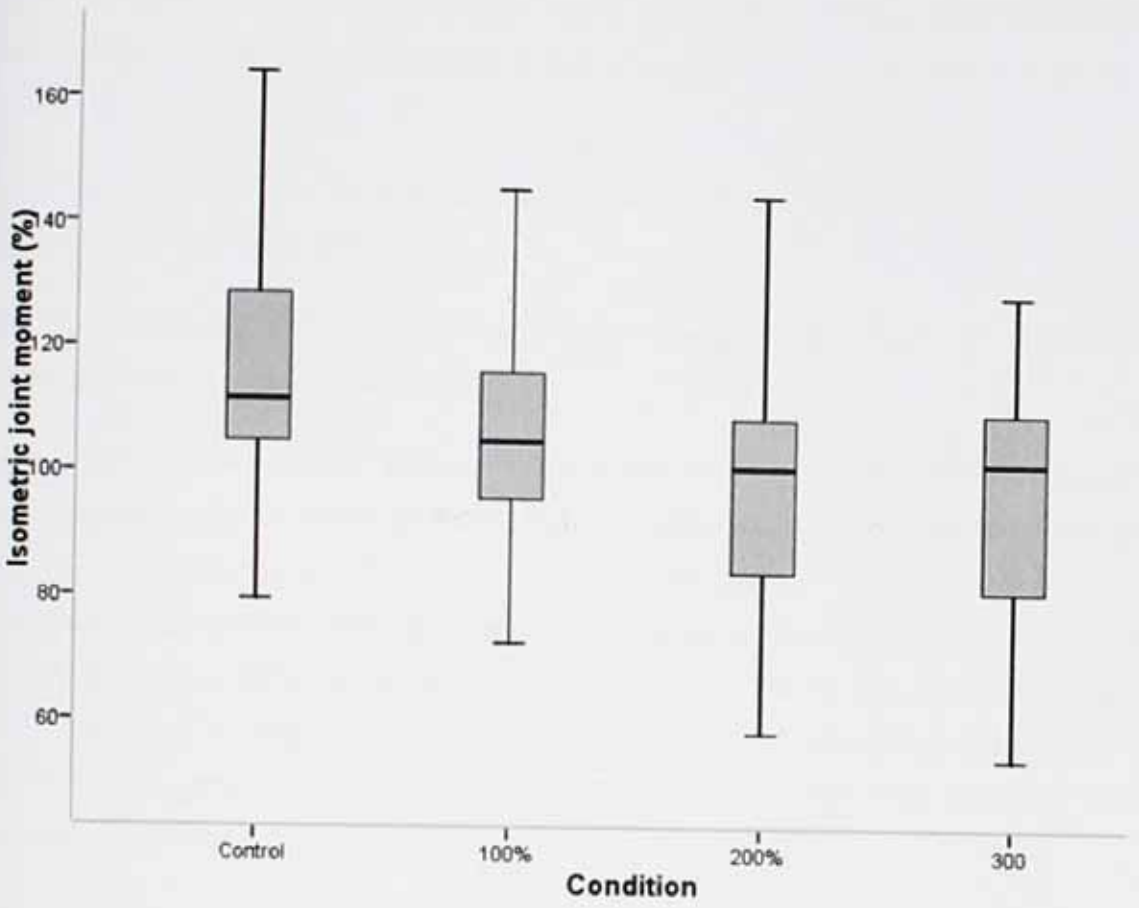


Figure 5.2. Normalised maximum voluntary isometric contractions produced in the control, 100%, 200% and 300% pain perception threshold conditions. Data are presented as median, IQR and range (n=31). The location of significant ($p > 0.05$) differences are denoted in Table 5.1.

Table 5.1. Statistical differences between mean maximal voluntary contractions produced in the four experimental conditions where significance is accepted at $p < 0.05$.

	Control	100%	200%	300%
Control	-	p = 0.000	p = 0.000	p = 0.000
100%	p = 0.000	-	p = 0.007	p = 0.005
200%	p = 0.000	p = 0.007	-	NS
300%	p = 0.000	p = 0.005	NS	-

NS = No significant difference

5.4.2 EMG VASTUS LATERALIS

Data were found to be normally distributed in the 100%, ($D(31) = 0.09$, $p > 0.05$), 200%, ($D(31) = 0.09$, $p > 0.05$), 300% ($D(31) = 0.10$, $p > 0.05$) and control condition, ($D(31) = 0.13$, $p > 0.05$). An analysis of variance with repeated measures (ANOVA) was therefore used to test the following:

H1: There is a difference in VL EMG activity in an isometric knee extension task when pain of varying severity is induced via GPD.

H0: There is no difference in VL EMG activity in an isometric knee extension task when pain of varying severity is induced via GPD.

A significant difference in VL EMG activity between the four conditions ($F = 10.933$, $p < 0.05$) was reported with post-hoc t-tests identifying a difference between all conditions and control ($p < 0.05$). In comparison to control (117.4 ± 29.8% MVC) mean EMG activity reduced by 13.6% in the 100% condition (101.79 ± 20.2 MVC), 16.2% in the 200% condition (98.4 ± 24.24% MVC) and 21.5% in the 300% condition (92.2 ± 22.2% MVC). There was a 9.5% reduction in the 300% condition in comparison to the 100% condition which was found to be significant ($p < 0.05$), but no other conditions significantly differed ($p > 0.05$) from 100% or 200% pain perception threshold conditions (see Figure 5.3).

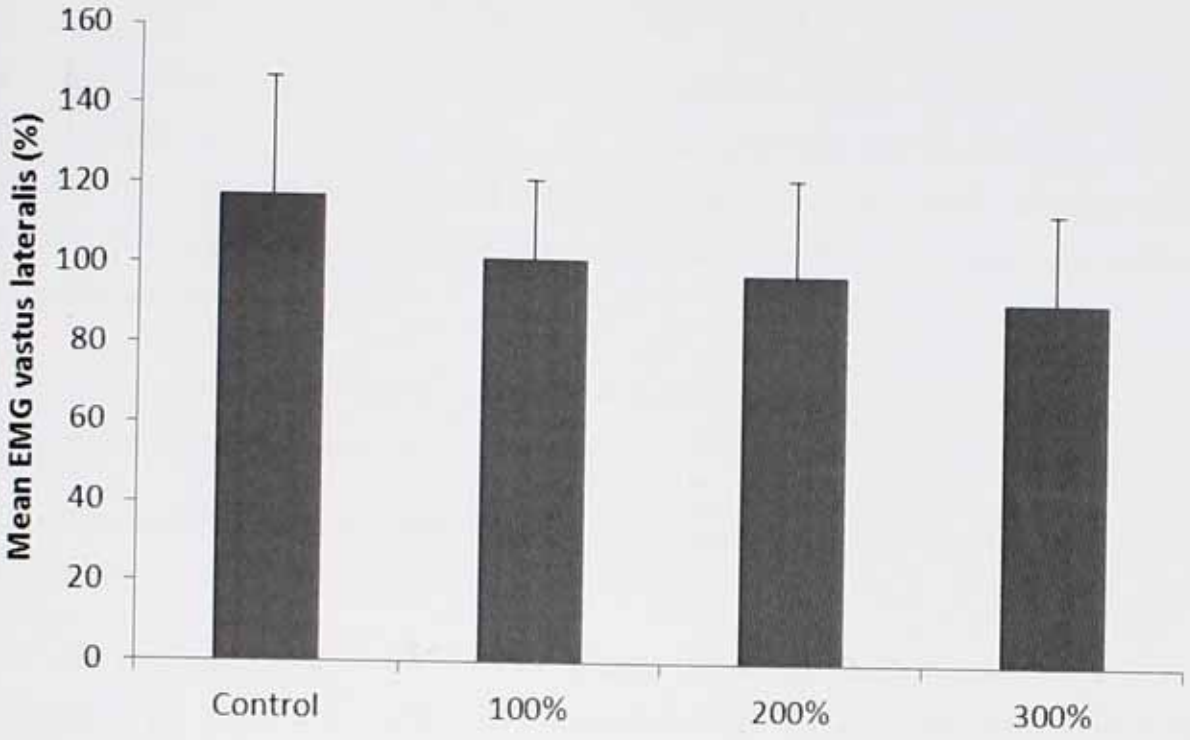


Figure 5.3. Normalised EMG activity recorded in vastus lateralis during the control, 100%, 200% and 300% pain perception threshold conditions. EMG recordings corresponding to the peak MVC produced over three trials in each condition were selected. Data are presented as mean ± SD (n=31). The location of significant differences is described in Table 5.2.

Table 5.2. Statistical differences in mean EMG activity of the VL produced between the four experimental conditions where significance is accepted at $p < 0.05$.

	Control	100%	200%	300%
Control	-	p = 0.042	p = 0.027	p = 0.000
100%	p = 0.042	-	NS	p = 0.019
200%	p = 0.027	NS	-	NS
300%	p = 0.000	p = 0.019	NS	-

NS = No significant difference

5.4.3 EMG SEMITENDINOSIS

The assumptions of normality were violated in the 100% condition, ($D(31) = .18$, $p < 0.05$), 200% condition, ($D(31) = .16$, $p < 0.05$), and control condition, ($D(31) = .002$, $p < 0.05$). Normality was confirmed within the 300% condition, ($D(31) = .11$, $p > 0.05$). No appropriate transforms were found for the other three conditions and therefore a Friedman's ANOVA was used to test the following:

H1: There is a difference in ST EMG activity in an isometric knee extension task when pain of varying severity is induced via GPD.

H0: There is no difference in ST EMG activity in an isometric knee extension task when pain of varying severity is induced via GPD.

There was a significant difference in EMG activity of the ST detected between the four conditions, ($\chi^2(3) = 13.88$, $p < 0.05$). Wilcoxon tests were used to determine where this difference existed. A Bonferroni correction was applied and so all effects are reported at a .0125 level of significance. A significant difference was found between control and 100% ($z = -2.82$, $p = 0.005$) but not between all other conditions ($p > 0.0125$). In comparison to control (median = 8.0, IQR = 5.7, range = 17.6% MVC) median EMG reduced by 22.5% in the 100% condition (median = 6.2, IQR = 6.2, range = 23.2% MVC), 12.5% in the 200% condition (median = 7.0, IQR = 5.9, range = 18.5% MVC) and 13.8% in the 300% condition (median = 6.9, IQR = 5.7, range = 17.6% MVC) (see Figure 5.3).

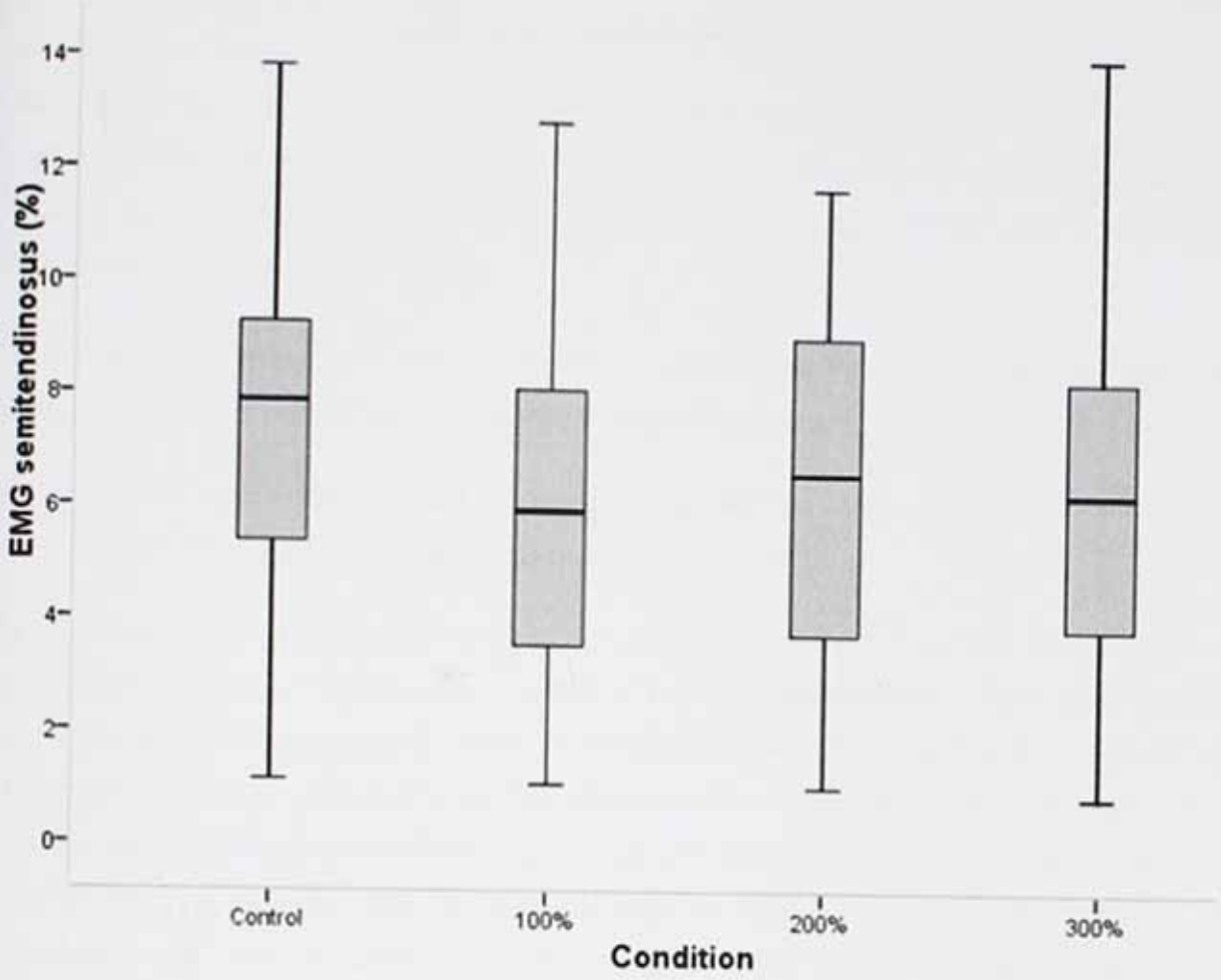


Figure 5.4. Normalised EMG activity recorded in semitendinosus during the control, 100%, 200% and 300% pain perception threshold conditions. EMG recordings corresponding to the peak MVC produced over three trials in each condition were selected. Data are presented as median, IQR and range (n=31).

Table 5.3. Statistical differences in mean EMG activity of the ST recorded between the four experimental conditions where significance is accepted at $p < 0.05$.

	Control	100%	200%	300%
Control	-	p = 0.005	NS	NS
100%	p = 0.005	-	NS	NS
200%	NS	NS	-	NS
300%	NS	NS	NS	-

NS = No significant difference

5.4.4 PAIN PERCEPTION (VAS)

The assumptions of normality were violated in the 100% condition, ($D(30) = .15$, $p < 0.05$), and 300% condition, ($D(30) = .17$, $p < 0.05$). Normality was confirmed within the 200% condition, ($D(30) = .11$, $p > 0.05$). No appropriate transforms were found and therefore a Friedman's ANOVA was used to test the following:

H1: There is a difference in pain perception reported via VAS during an isometric knee extension task when pain of varying severity is induced via GPD.

H0: There is no difference in pain perception reported via VAS during an isometric knee extension task when pain of varying severity is induced via GPD.

There was a significant difference in pain perception reported between the three conditions, ($\chi^2(2) = 44.60$, $p<0.05$). Wilcoxon tests were used to determine where this difference existed. A Bonferroni correction was applied and so all effects are reported at a .017 level of significance. There was a significant increase in pain perception between the 100%-200% conditions ($z = -4.39$, $p = .000$), 100%-300% conditions ($z = -4.78$, $p = .000$) and 200%-300% conditions ($z = -3.36$, $p = .001$). In comparison to the 100% condition (median = 24.9, IQR = 27.8, range = 82.3% MVC) there was a 122.1% increase in pain perception in the 200% condition (median = 55.3, IQR = 37.6, range = 88.7% MVC), and 198.4% increase in the 300% condition (median = 74.3, IQR = 32.4, range = 91.5% MVC). There was a 34.6% increase in pain perception in the 300% condition in comparison to the 200% condition (see Figure 5.4).

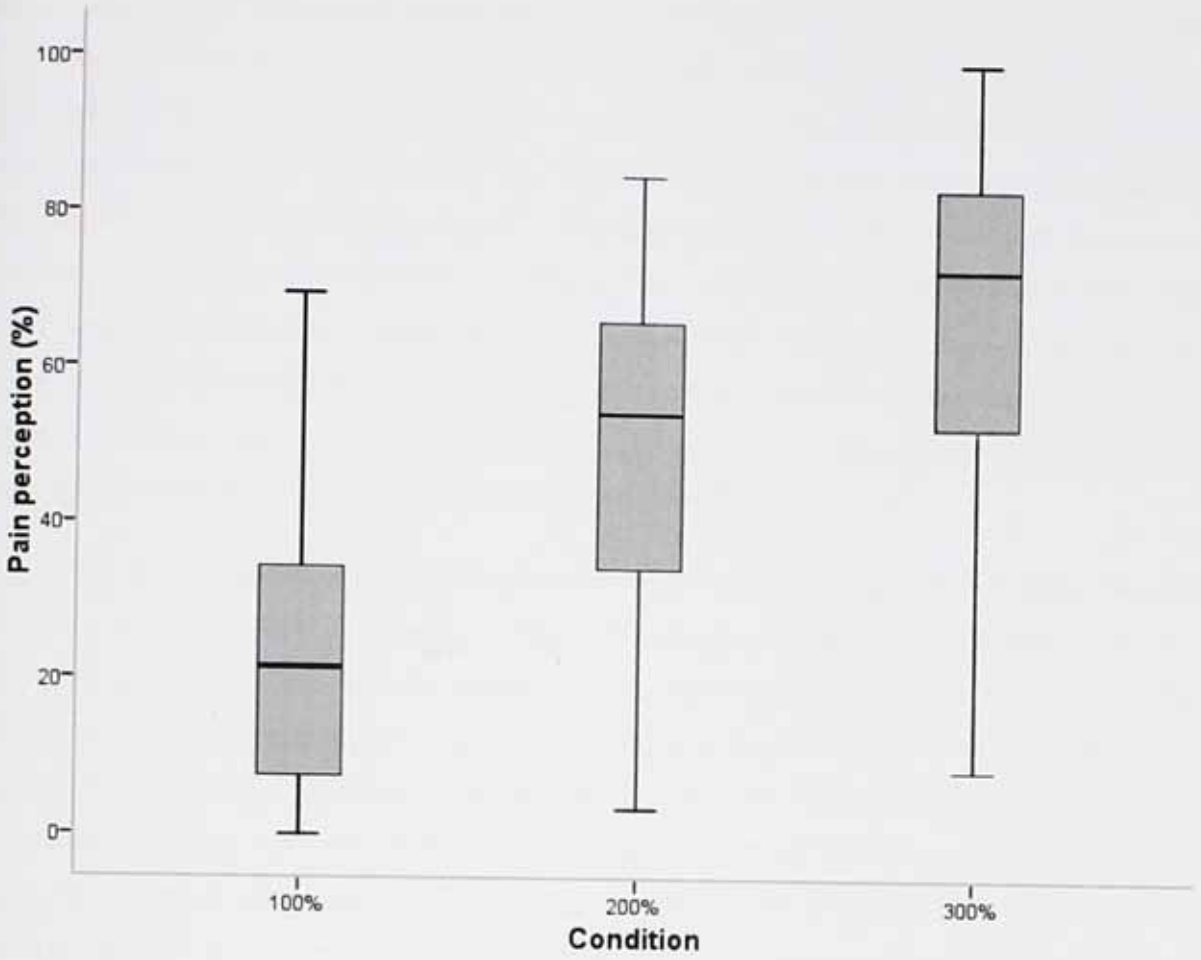


Figure 5.5. Pain perception as reported by participants on a visual analogue scale in the 100%, 200% and 300% conditions. Data are presented as median, IQR and range (n=31). The location of significant differences is described in Table 5.3.

Table 5.4. Statistical differences in mean pain perception reported via VAS in each of the three pain conditions where significance is accepted at $p < 0.05$.

	100%	200%	300%
100%	-	p = 0.000	p = 0.001
200%	p = 0.000	-	p = 0.000
300%	p = 0.001	p = 0.000	-

5.5 DISCUSSION

The major finding of this study was that muscular performance on a maximal isometric knee extension task decreased as pain intensity induced by GPD increased. This supports the findings of Farina et al. (2004) who determined a similar association between pain and muscle

performance where hypertonic saline injections were used to induce pain. The current study used a non-invasive method of pain induction whereby pain was induced remotely from the target muscle thereby limiting the potential confounding variables, such as changes to muscle physiology or mechanics as a result of fluid injections. This suggests neuronal inhibition is the likely cause of reductions in performance as discussed in Chapter 4, however does not allow for location of such impairment to be determined and this is something that requires further study. It has been suggested that in order to gain a better understanding of central muscle control strategies, more advanced techniques are required so that activity distribution can be traced at single motor unit level (Farina et al., 2002). This was not the purpose of the current investigation but should be considered for future research.

The most likely theory to explain the reductions in muscle performance identified is the adaptation to pain theory (Hodges & Tucker, 2011) as it proposes a non-uniform response to pain whereby a global decrease in muscle activity is one of three possible responses to pain. Whilst the pain adaptation model (Lund et al., 1991) also suggests a decrease in muscle activity, it specifically predicts a decrease in agonist EMG activity and associated increase in antagonist EMG activity which was not evident in this study, instead both agonist and antagonist decreased. Another adaptation response proposed by Hodges & Tucker (2011) is that muscle activity is redistributed to alternate muscles that are able to fulfill similar functions (Hodges & Tucker, 2011). However, this study only examined the VL (agonist) and ST (antagonist) and can therefore not state whether redistribution occurred in this case, but the fact that reductions in activity were identified whilst pain was induced remotely, coupled with concomitant reductions in force, suggests that neuronal inhibition was the likely cause rather than any mechanical or physiological mechanisms within the muscle. A further consideration is that this study did not specifically test athletes, instead recruited recreationally active males. There is a suggestion that athletic status increases an athlete's ability to tolerate pain (Tesarz et al., 2012) which may therefore make them more able to control their performance in the knee extension task even when experiencing higher levels of pain. This may result in different findings were the study to be replicated using athletes at a high level.

An interesting finding within the data collected for ST EMG activity was that there was a significant difference between the control condition and 100% condition. However, there was no significant difference found between control and 200% or 300% despite the fact that the median decrease from control to 200% (7.0%) and 300% (6.9%) was greater than the median difference from control to 100% (6.2%). This could be attributed to a number of factors. Firstly, the amount of activity required by the ST during this task was limited as the participant was strapped in to the dynamometer with their knee locked at 90° and was instructed to push

against the lever arm. Therefore involvement of an antagonist is minimal, and the perception that they were in a safe environment (little risk of injury) may have contributed to the lack of engagement of the ST. Figures recorded tended to be below 10% maximum and there were comparatively large deviations reported, indicating that this data is sensitive to distortion. Where dynamic tasks have been used (Arendt-Nielsen et al., 1996) there is a clearer increase in antagonist EMG, supporting the Lund et al. (1991) theory, as within dynamic tasks it can be assumed that the need for stabilisation and control around the joint is greater and therefore engagement will likely be higher. This may explain why the pain adaptation model was not supported in this study and lends further support to the adaptation to pain theory in suggesting that task demands may also contribute to the protective strategy adopted.

As was discussed in Chapter 4 the method of inducing pain via GPD allows for careful control of pain induced. The researcher was able to accurately measure absolute pressure being exerted (to within 5mmHg) and relate this to perception of pain reported by the participant in order to establish the experimental conditions. An interesting finding in this study is that although pressure exerted (absolute pain) increases incrementally from pain perception threshold, there was not a symmetrical increase in pain perception (as measured by VAS). No correlational analysis was carried out due to the different type of the data gathered between the variables (with VAS data being reported as absolute values and force data being normalised). However, researchers may wish to consider carrying out studies in the future that seek to explore the relationship between pain perception reported and force output in order to build up a better understanding of the mechanisms at work as in the current study some interesting findings emerged. Perception of pain appeared to plateau after 200% pain perception threshold was reached with a greater mean difference found between 100-200% (26.25%) than between 200-300% (12.99%) conditions. Interestingly this relationship was similar in the force data, with a plateau appearing to occur. The median difference in force between the control to 100% condition was 6.3%, but between 100 to 200% was only 3.5% and between 200 to 300% was 3.7%. The current study did not investigate beyond 300% pain perception threshold and therefore it cannot be assumed that this is a plateau and that no further increases would occur, but it does suggest that the association between pain perception and muscle performance may be closer than that between absolute pain and muscle performance. This adds further support to the suggestion that reductions in performance in the presence of pain are as a result of neuronal inhibition. However, to determine whether this is at spinal or supra-spinal level further investigation is required.

5.6 CHAPTER SUMMARY

This study showed that severity of pain induced via non-invasive GPD is inversely related to muscular performance in the lower limb. This supports previous work in which injection of hypertonic saline was used to induce pain of varying intensities. Concomitant reductions in agonist EMG and force data, combined with the non-invasive methods employed suggest a neuronal inhibition as the likely cause of reductions in force. In contrast to the Farina et al. (2004) study, this research was able to carefully control for severity of pain induced which was demonstrated through the VAS data collected and indicated non-linear relationship between absolute pain and perceived pain which may have consequences for future studies. Whilst this study was able to rule out mechanical or physiological changes to the muscle being a likely cause of performance differences, it cannot conclusively suggest a mechanism by which changes occur. It is likely that reductions in performance are as a result of neuronal inhibition, but location (spinal or supra-spinal) remains unknown and should be investigated further.

6 THE ROLE OF PERIPHERAL MECHANISMS IN REDUCTION OF NEUROMUSCULAR PERFORMANCE IN A MAXIMAL ISOMETRIC KNEE EXTENSION TASK DURING PAIN

6.1 INTRODUCTION

Pain has been identified as a factor that reduces muscular performance in a range of tasks (Ervilha et al., 2005; Farina et al., 2008; Sohn et al., 2004; Wing et al., 2011a, 2011b). However, to date a comprehensive understanding of the mechanisms by which pain reduces muscle performance is not available. Studies conducted within the current research have established that the presence of low level pain reduces muscle performance (see Chapter 4), and further to this that the severity of pain is inversely related to exercise performance (see Chapter 5) when pain is induced via GPD in the lower limb remote from the target muscle. Concomitant reductions in muscle activity and force produced by the participants, combined with the non-invasive methods employed to induce pain suggest a neuronal inhibition as the likely cause of reductions in performance but whether this was at the central or peripheral level could not be determined from these studies.

The previous work reported in this thesis suggest that the most likely theory to explain the reductions in muscle performance is the adaptation to pain theory (Hodges & Tucker, 2011) as it proposes a non-uniform response to pain rather than any single uniform response that one would expect to see on every occasion pain is present Whilst the pain adaptation model (Lund et al., 1991) also suggests a decrease in muscle activity, it specifically predicts a decrease in agonist EMG activity and associated increase in antagonist EMG activity which was not evident in the findings from chapter 5. Given that reductions in activity were identified whilst pain was induced remotely from the target muscle, coupled with concomitant reductions in force, it could be suggested that neuronal inhibition was the likely cause of reductions reported rather than any mechanical or physiological mechanisms within the muscle. However, even though pain was induced on a separate site from the target muscle, it was still on the same limb which means that there may have been some inhibition possible at a local level.

Theories that attempted to identify a specific 'pain pathway' focused upon the physiological aspects of pain sensation (Weddell et al., 1948; Von Frey, 1894) with only limited acknowledgement of the interaction between individual and environment (Marshall, 1894; Melzack, 2001). The Gate Control theory proposed by Melzack and Wall (1965) recognised that whilst a "pain pathway" could exist, it is likely that there was interaction between ascending and descending neural pathways that mediated the pain experience. In contrast, the neuromatrix proposed by Melzack (2001) disregarded the single pathway approach and instead suggested

that the pain experience could not be viewed as distinct from the environment, individual factors and past experience. However, no conclusion for the exact mechanisms of pain related reductions in performance were agreed upon and so further work is required. The vicious cycle theory (Roland, 1986), pain adaptation model (Lund et al., 1991) and adaptation to pain theory (Hodges & Tucker, 2011) all agree that pain is associated with certain changes in muscular performance.

6.2 RESEARCH AIM

To investigate whether peripheral mechanisms are associated with neuromuscular reductions in performance in the presence of pressure pain?

6.3 METHOD

6.3.1 PARTICIPANTS

Nineteen recreationally active male participants (mean ± SD; age = 21.0 ± 3.4 yr, height = 1.8 ± 0.06, mass = 80.6 ± 9.0kg) volunteered for the study after giving written informed consent (see Appendix 1). As this study was exploratory in nature and would be used in conjunction with previous study findings (see chapter 5) this was deemed a large enough sample population to gain meaningful results. The participants were free from any lower limb injury and were asked to refrain from any intense exercise or stimulant use for 24 hrs prior to testing. Ethical approval was granted from the School of Health's Ethics Committee in accordance with the declaration of Helsinki.

6.3.2 PROCEDURE

Equipment, measures and experimental protocol were as described in chapter 4 with all participants visiting the lab on two separate occasions for a familiarisation session and then experimental trial. Each participant undertook three MVCs in three conditions in a counterbalanced and randomised trial. The peak MVC in each trial was selected for measurement.

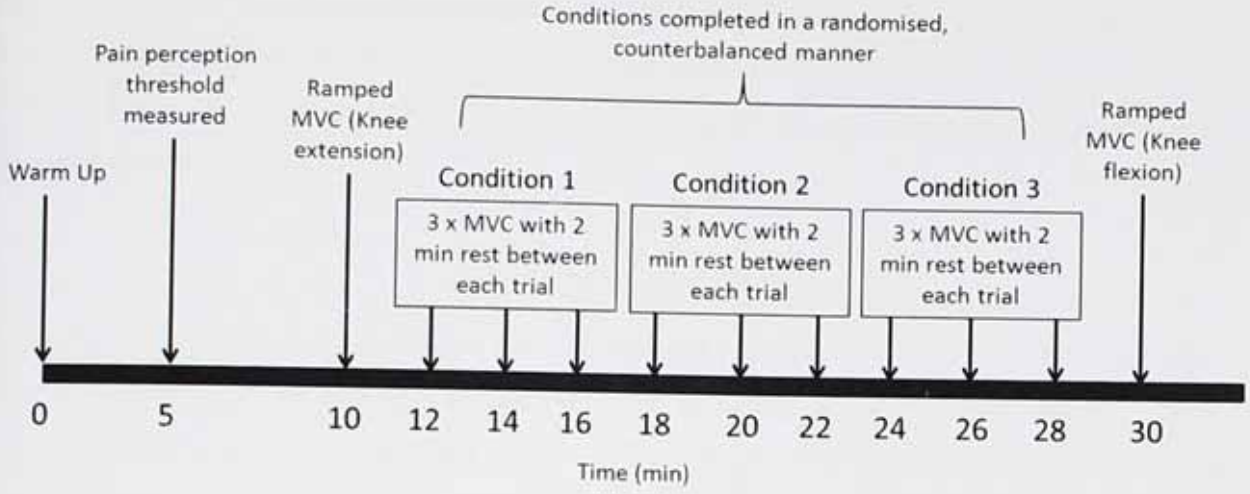


Figure 6.1. Timeline of protocol undertaken by each of the participants during the experimental trials.

6.3.3 EXPERIMENTAL CONDITIONS

Three conditions were performed within the trial in which control, 100% or 200% of the participants previously determined pain perception threshold was induced using the GPD. The GPD was strapped to the lower limb with the cleat positioned over the tibia one third from medial tibial epicondyle to medial malleolus on the leg not being tested in the MVC. The GPD was then inflated at 5 $mmHg.s^{-1}$ to the previously established pain threshold, considered as the lowest experience of pain that a participant would recognise (Loeser & Treede, 2008). The participant was then instructed to perform the MVC trials. Immediately following each MVC, pressure from the cuff was released, while the GPD remained in position between each trial to ensure reliability of the position of the device.

6.3.4 ISOMETRIC JOINT MOMENT

Participants sat upright in the chair of an isokinetic dynamometer (Biodex System 3 Pro) with the knee positioned at a 90° angle (180° full extension) with the lateral femoral epicondyle aligned to the centre of rotation of the dynamometer. The foot and shank were supported by Velcro strapping attached to the lever arm to hold the lower limb in position. The upper body was also strapped into position across the chest, pelvis and femur to ensure reliable subject positioning. Participants were instructed to sit with their hands behind their backs for each trial as pilot testing indicated this position resulted in the most reliable joint moment data. During the MVC trials, joint moment data were exported from the dynamometer to a high-level transducer (model HLT100C, Biopac) before analogue-to-digital conversion at a 1000-Hz sampling rate (model MP150 Data Acquisition, Biopac). The data were then directed to a personal computer running AcqKnowledge software (version 4.0, Biopac) and filtered with a

double-pass 6-Hz Butterworth low-pass filter. Data were normalised as a percentage of the peak isometric joint moment recorded during the ramped MVC. The normalised joint moment (%MVC) from the trials that produced the greatest joint moment were used as a measure of muscular performance.

6.3.5 DATA ANALYSIS

In each condition the peak MVC trial was selected for analysis. All values were normalised to the ramped contractions and reported as percentages; all data are reported as mean ± SD where normally distributed, and as median, IQR and range where non-parametric. Data within each condition were examined for normality using the Kolmogorov-Smirnov test. Where the raw data were not normally distributed appropriate data transformation was used. Where no suitable transformation could be found, non-parametric tests were used. Post-hoc t-tests with Bonferroni correction were used where statistical significance was established. Statistical significance was accepted at $p < 0.05$ (see chapter 4 for full explanation of data analysis).

6.4 RESULTS

Due to the number of hypotheses being examined within this study, the results have been divided up for ease of understanding into; isometric knee extensor moment, EMG of the vastus lateralis, EMG of the semitendinosus and pain perception (VAS). Each section first reports normality of the data being tested (using the Kolmogorov-Smirnov test) and then any significant differences ($p < 0.05$) found as well as showing where those differences lie where appropriate.

6.4.1 ISOMETRIC KNEE EXTENSOR MOMENT (FORCE)

Normality was confirmed in all three conditions; control, ($D(20) = 0.16$, $p > 0.05$), 100%, ($D(20) = 0.17$, $p > 0.05$), and 200%, ($D(20) = 0.11$, $p > 0.05$). An analysis of variance (ANOVA) with repeated measures was therefore used to test the following:

H1: There is a difference in knee extensor moment in an isometric knee extension task when pain is induced contra-laterally via GPD.

H0: There is no difference in knee extensor moment in an isometric knee extension task when pain is induced contra-laterally via GPD.

A significant difference was reported in force produced between the three conditions ($F = 9.273$; $p < 0.05$), with Bonferroni post-hoc t-tests indicating a significant difference existed between all conditions and control ($p < 0.05$). In comparison to control (114.3 ± 16.2% MVC)

mean force was 11.1% lower in the 100% condition (102.5 ± 16.1% MVC) and 15.8% lower in the 200% condition (98.4 ± 17.4% MVC). There was no significant difference detected between 100% and 200% conditions ($p > 0.05$).

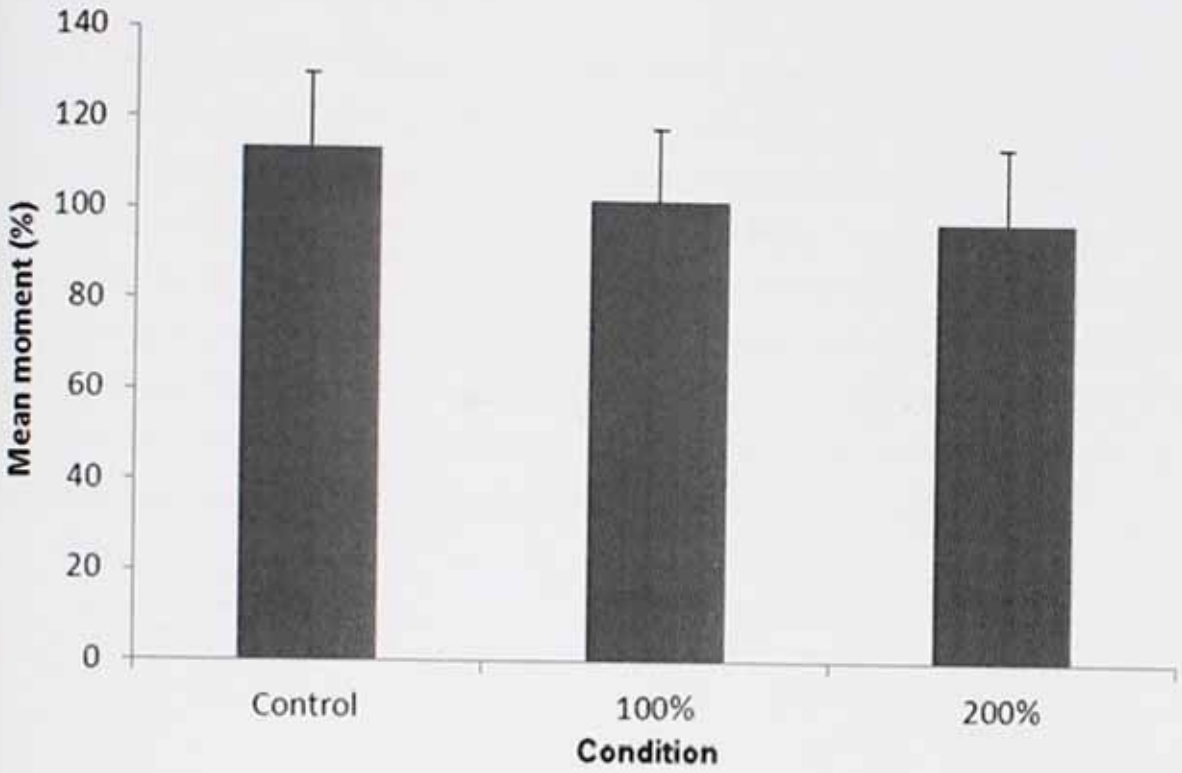


Figure 6.2. Normalised maximum voluntary isometric contractions produced in the control, 100%, and 200% pain perception threshold conditions. Data are presented as mean ± SD (n=19).

Table 6.1. Statistical differences between mean maximal voluntary contractions produced in the three experimental conditions where significance is accepted at $p < 0.05$.

	Control	100%	200%
Control	-	p = 0.004	p = 0.005
100%	p = 0.004	-	NS
200%	p = 0.005	NS	-

NS = No significant difference

6.4.2 EMG VASTUS LATERALIS

The assumptions of normality were violated in the 100% condition, ($D(20) = .24$, $p < 0.05$), 200% condition, ($D(20) = .28$, $p < 0.05$), and control condition, ($D(20) = .24$, $p < 0.05$). No appropriate transforms were found and therefore a Friedman's ANOVA was used to test the following:

H1: There is a difference in VL EMG activity in an isometric knee extension task when pain induced contra-laterally via GPD.

H0: There is no difference in VL EMG activity in an isometric knee extension task when pain is induced contra-laterally via GPD.

There was a significant difference in VL EMG activity detected between the three conditions, ($\chi^2(2) = 23.05$, $p<0.05$). Wilcoxon tests were used to determine where this difference existed. A Bonferroni correction was applied and so all effects are reported at a .0125 level of significance. A significant difference was found between control and 100% ($z = -3.62$, $p = .000$) and between control-200% ($z = -3.78$, $p = .000$), but not between 100%-200% ($z = -2.0$, $p = .841$). In comparison to control (median = 103.1, IQR = 24.6, range = 104.4% MVC) mean EMG activity reduced by 14.8% in the 100% condition (median = 87.8, IQR = 34.3, range = 122.9% MVC) and 17.1% in the 200% condition (median = 85.5, IQR = 28.6, range = 119.0% MVC). There was no significant difference ($p > 0.05$) found between 100% and 200% (see Figure 6.2).

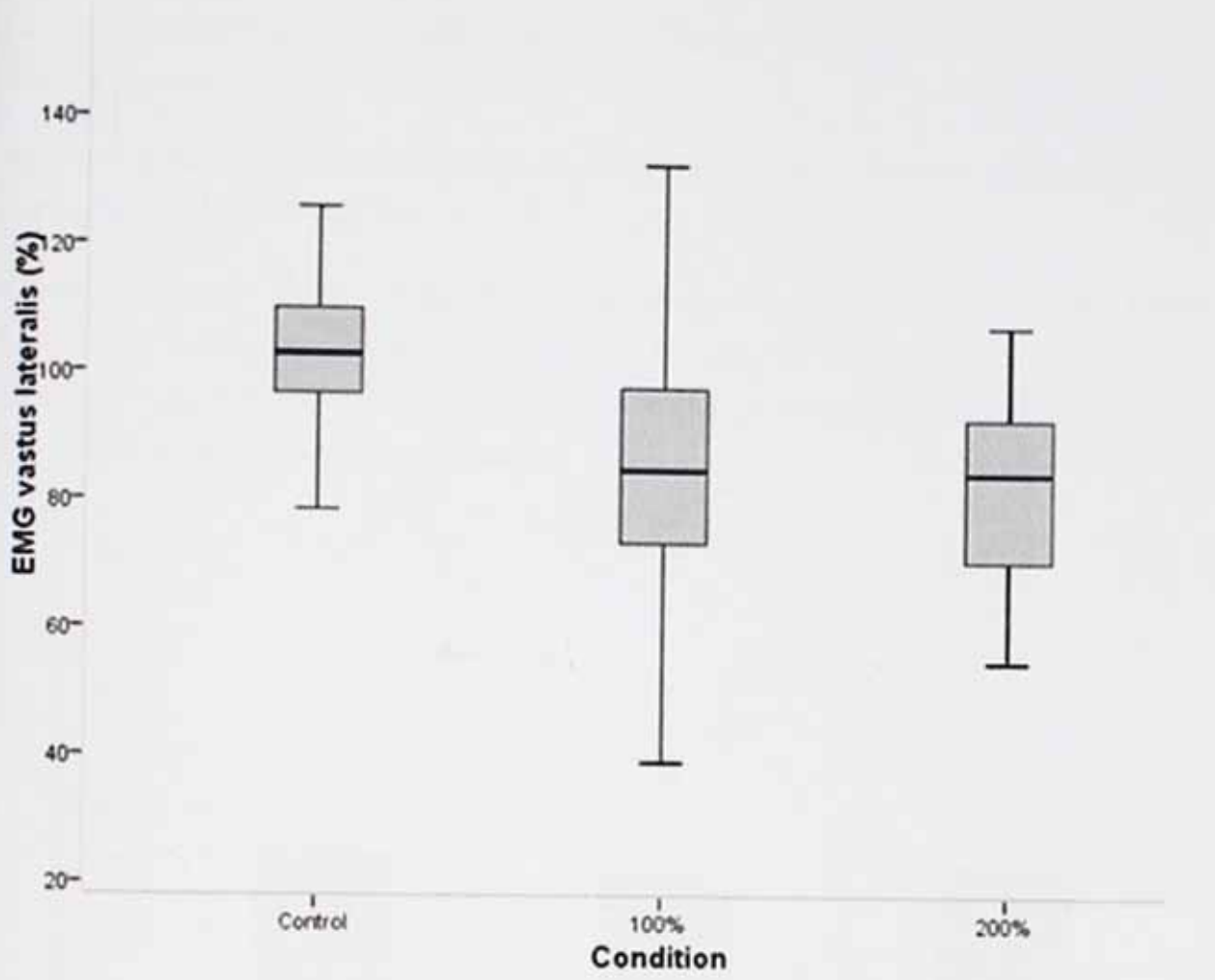


Figure 6.3. Normalised EMG activity recorded in vastus lateralis during the control, 100% and 200% pain perception threshold conditions. EMG recordings corresponding to the peak MVC produced over three trials in each condition were selected. Data are presented as median, IQR & range (n=20).

Table 6.2. Statistical differences between normalised EMG activity of the vastus lateralis recorded in the three experimental conditions where significance is accepted at $p < 0.05$.

	Control	100%	200%
Control	-	p = 0.000	p = 0.000
100%	p = 0.000	-	NS
200%	p = 0.000	NS	-

NS = No significant difference

6.4.3 EMG SEMITENDINOSIS

Data were found to be normally distributed in the control, ($D(19) = 0.15$, $p > 0.05$), 100%, ($D(19) = 0.18$, $p > 0.05$) and 200% condition, ($D(19) = 0.15$, $p > 0.05$). An analysis of variance with repeated measures (ANOVA) was therefore used to test the following:

H1: There is a difference in ST EMG activity in an isometric knee extension task when pain induced contra-laterally via GPD.

H0: There is no difference in ST EMG activity in an isometric knee extension task when pain is induced contra-laterally via GPD.

Repeated measures ANOVA found there to be no significant difference in ST EMG activity between the three conditions ($F = .674$, $p > 0.05$). In comparison to control (13.1 ± 8.3% MVC) mean EMG reduced by 7.4% in the 100% condition (12.2 ± 8.1% MVC) and 3.1% in the 200% condition (12.7 ± 8.9% MVC) (see Figure 6.3).

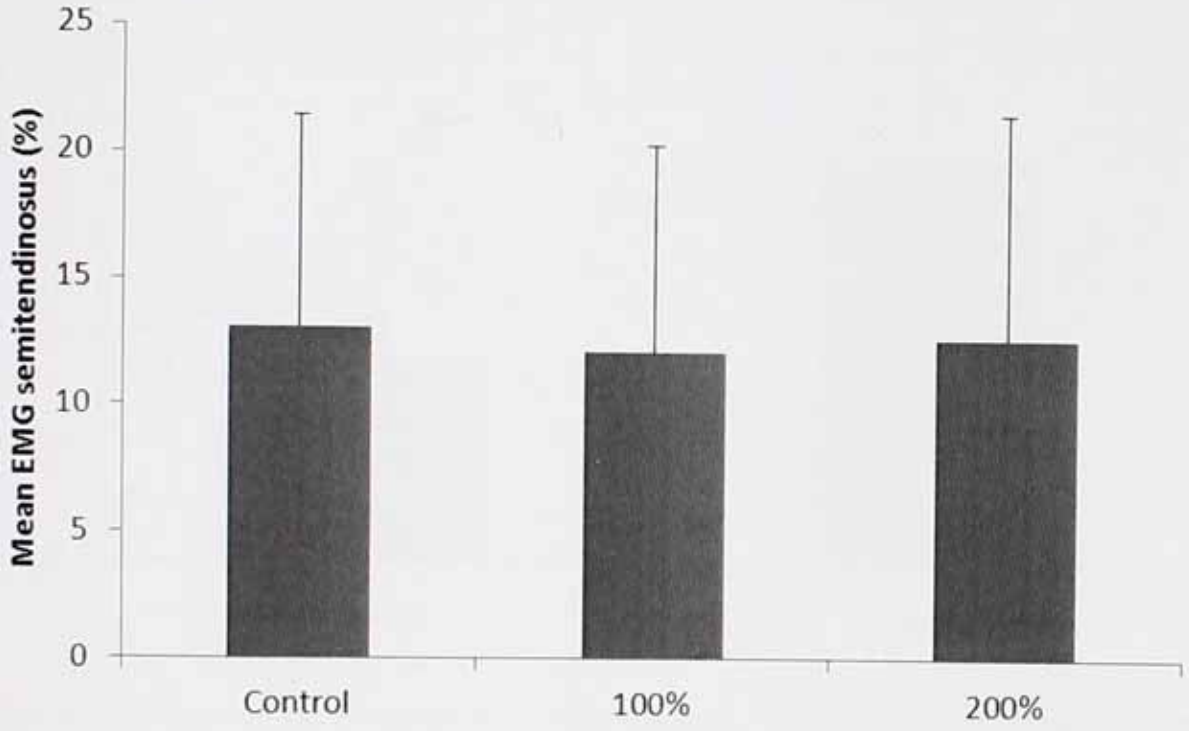


Figure 6.4. Normalised EMG activity recorded in the semitendinosus during the control, 100% and 200% pain perception threshold conditions. EMG recordings corresponding to the peak MVC produced over three trials in each condition were selected. Data are presented as mean ± SD (n=19). No significant differences between conditions were detected ($p > 0.05$).

6.4.4 PAIN PERCEPTION (VAS)

Data were found to be normally distributed in the 100%, ($D(19) = 0.16$, $p > 0.05$), and 200% condition, ($D(19) = 0.11$, $p > 0.05$). An analysis of variance with repeated measures (ANOVA) was therefore used to test the following:

H1: There is a difference in pain perception reported via VAS during an isometric knee extension task when pain is induced contra-laterally via GPD.

H0: There is no difference in pain perception reported via VAS during an isometric knee extension task when pain is induced contra-laterally via GPD.

A significant difference in pain perception reported was found between the two conditions ($F = 38.903$; $p < 0.05$) in line with increased pressure exerted via GPD. In comparison to the 100% condition (18.4% ± 11.8%) there was a 50.8% increase in pain perception in the 200% condition (37.4 ± 19.4%) (see Figure 6.5).

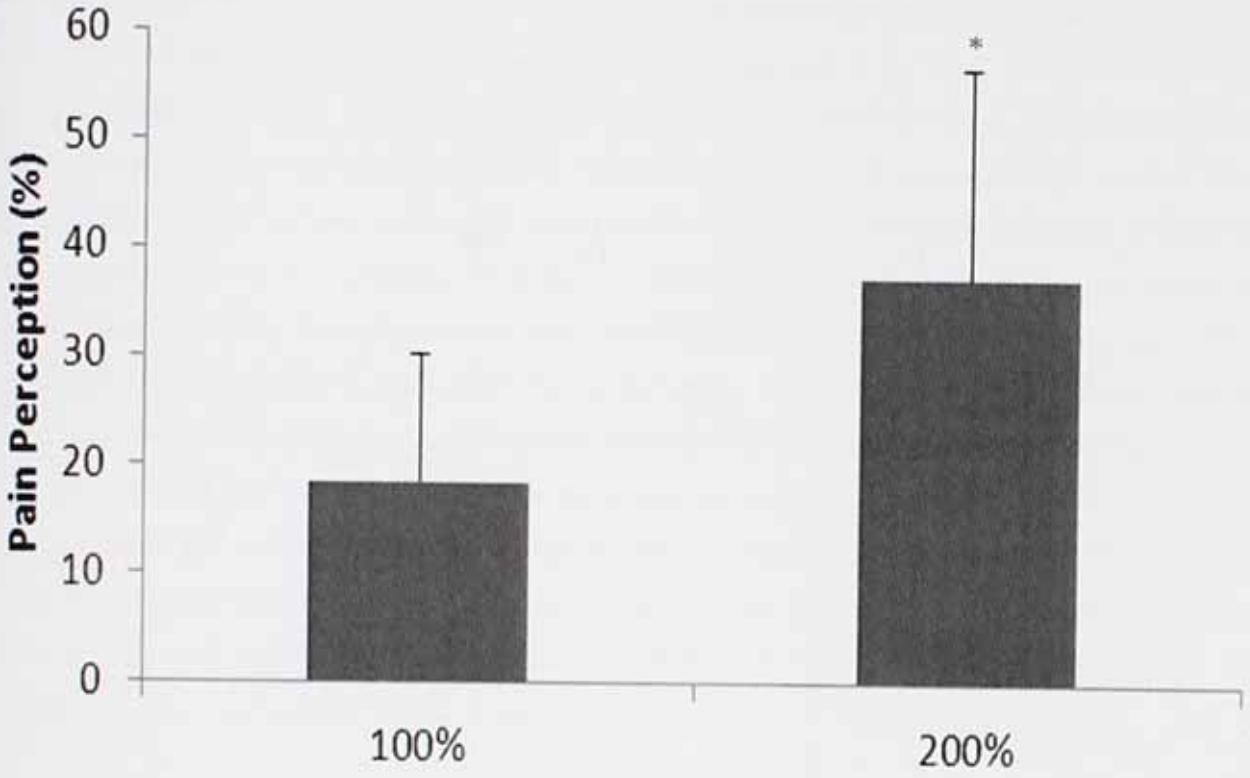


Figure 6.5. Pain perception as reported by participants on a visual analogue scale in the 100% and 200% conditions. Data are presented as mean ± SD (n=19) where * denotes a significant increase compared with 100% condition ($p < 0.05$).

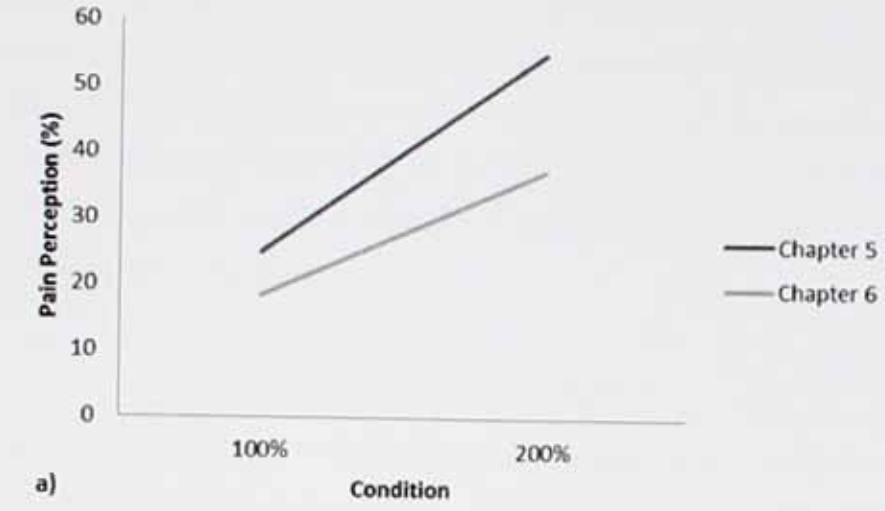
6.5 DISCUSSION

Muscular performance on a maximal isometric knee extension task was found to be inversely proportional to severity of pain induced by GPD contra-laterally, supporting the findings of Chapter 5. This is also in support of Farina et al. (2004) who determined a similar association between pain and muscle performance where hypertonic saline injections were used to induce pain. The present research used a non-invasive method of pain induction, the GPD, and in the current study pain was induced on the opposite limb to the one being measured for muscle performance. This means that the reductions in performance reported in this study could not be caused by mechanical or physiological changes to the muscle being tested as there was no interaction between the GPD and target muscle (Wing et al., 2011b) and thereby suggests

neuronal mechanisms are responsible for the changes (see chapter 4). Further to this, as pain was not only induced remote from the target muscle but was also on the opposite lower limb, it is highly unlikely that the changes in neuromuscular performance reported were as a result of peripheral mechanisms. This therefore suggests that central mechanisms were responsible.

Whilst this study was able to rule out peripheral mechanisms, it was not the intention of the study give a precise location of the neuronal inhibition; this will require further work and more sophisticated techniques. As was discussed in chapter 5, the theory that best supports the findings presented is the adaptation to pain theory (Hodges & Tucker, 2011) as it proposes a non-uniform response to pain dependent upon the conditions of the task and region of muscle activity being measured. The findings reported in chapter 5 showed that as the severity of pain experienced by the individual increased, there was a related decrease in both muscle force produced and EMG activity within the agonist and antagonist. This study was able to replicate these findings, even though the pain stimulus was not on the limb carrying out the isometric knee extension task. This means that even when the participant experienced pain on another region of their body where there could be no fear of the movement task causing further risk of injury or increasing pain, they still reduced the force output. The VAS data confirm that the pressure exerted by the GPD was perceived by the individual to be painful, and incrementally more painful as pressure increased, which means that the location of the GPD on the opposite limb did not appear to change the pain experienced by the participant. This would suggest that the performance losses reported were due to inhibition at a neuronal level, which supports the model proposed by Hodges & Tucker (2011).

The aim of this study was to determine whether peripheral mechanisms could be responsible for changes in muscle performance, which it determined was highly unlikely. As the current study was only intended to explore the possibility of these mechanisms being peripheral, fewer participants were used than previously (N=19), however this still produced significant results. It was decided that only two levels of pain severity would be tested as the intention of this study was to determine whether there was a relationship between pain severity and muscle performance; rather to examine whether similar relationships could be seen as when pain was induced on the same leg. It is interesting to compare the patterns evident between the two studies as they are very closely associated (see Figure 6.6).



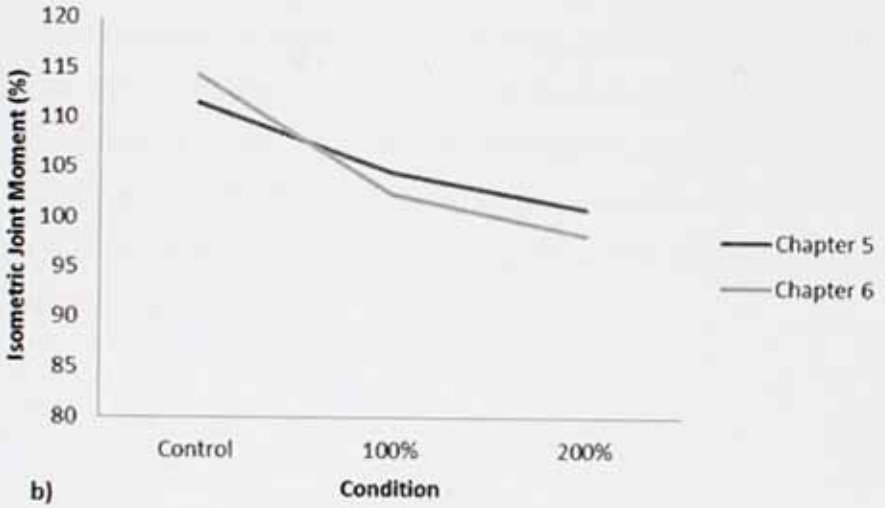


Figure 6.6.a) Normalised perceived pain reported via VAS in the 100% and 200% pain perception threshold conditions by participants in Chapter 5 (N=31) and Chapter 6 (N=19). **b)** Normalised maximum voluntary isometric contractions produced in the control, 100%, and 200% pain perception threshold conditions for Chapter 5 (N=31) and Chapter 6 (N=19).

Figure 6.6.a. shows that severity of pain reported in both the Chapter 5 and 6 studies increased in line with pressure exerted via GPD. There were slightly lower values reported on the VAS for Chapter 6 where pain was induced contra-laterally, perhaps because the mechanics of the task required the participant to push in the direction of where the pain was induced; therefore if it was induced on the opposite leg the participant did not have the fear component associated with pain (Pen & Fisher, 1994) in that there was no risk of them making the pain worse by completing the isometric knee extension task. This may have caused them to perceive the pain as less threatening and therefore not attach such a high value to the stimulus (Addison et al., 1998). Interestingly, whilst the severity of pain reported was lower in the current study, the amount of force produced was also lower across the two experimental conditions in comparison to the chapter 5 results despite the participants producing greater force output in the control

condition initially (see Figure 6.6.b). However, this may have been due to the different participants used within the two samples. There were only small changes between the results, which given the number of participants may account for variance. An examination of the mean differences between the three conditions shows that the pattern of reduction remains similar when pain is induced contra- and ipsi-laterally, suggesting that the location of stimulus did not influence the participants' response to pain.

6.6 CHAPTER SUMMARY

This study was able to demonstrate that peripheral mechanisms are unlikely to be responsible for reductions in performance experienced when pain is induced. In support of previous studies, the findings from this chapter suggest that the mechanism responsible for reducing neuromuscular performance is likely to be neuronal (see chapter 4) and that pain induced remote from the target muscle will reduce performance incrementally as severity of pain increases (see chapter 5). Further to this it was able to suggest that central, rather than peripheral mechanisms are the likely cause of those performance reductions. What remains unclear is at what level central mechanisms impair performance; spinal or supra-spinal, and this requires further investigation.

7 THE INFLUENCE OF EXPECTATION OF PAIN SEVERITY ON NEUROMUSCULAR PERFORMANCE IN A MAXIMAL ISOMETRIC KNEE EXTENSION TASK

7.1 INTRODUCTION

Studies reported in Chapter 4 & 5 determined that reductions in muscular performance associated with the presence of pain are expected to be a result of neuronal inhibition which Chapter 6 proposed was likely to be central rather than peripheral inhibition. However, it is not clear at what level this inhibition occurs; spinal or supra-spinal. It is suggested that pain stems from the detection of noxious stimuli signifying potential risks (Heil & Fine, 1999), however little neurological evidence can be presented to show that the stimuli itself is noxious (Pen & Fisher, 1994), instead it is proposed that every stimulation is simply carried as an impulse along a certain neural pathway and it is only at the point of recognition or perception that these stimuli become recognised as painful (Melzack, 2005). This suggests that the individual may have some form of conscious control over the pain experience, which would be beneficial to sport in which ability to endure pain is necessary to perform well if combined with psychological strategies to overcome or cope with the pain.

Little is currently known about the possible implications of anticipating pain and how this may influence muscle performance. The authors' previous work (chapter 5) has shown that when an individual is exposed to varying levels of pain whilst being asked to perform a maximal knee extension task, their ability to execute the task to maximum is compromised as severity of pain increases (Wing et al., 2011a) but what remains to be determined is whether there is an effect associated with the expectation an individual has that a task is going to be more or less painful and if this will influence muscular output. If there is a change associated with expectation of pain intensity, then it is likely that the mechanism by which this occurs is supra-spinal rather than a reflex.

This study will contain a placebo and nocebo condition used to determine the influence of expectation of pain intensity. Research on the effects of placebos and nocebos suggests an important link between physiology and psychology (Geers et al., 2007) as they are believed to produce an effect in patients that result from intent rather than specific physical or chemical properties (Arnstein, 2003). Numerous psychological factors have been found to influence the placebo response (Pacheco-López et al., 2006) including expectations (Benedetti et al., 2007) and the character of pain (Hauor, 2005). All of these factors can mediate the effect of a message that is being sent to the brain via interactions occurring between ascending and descending

pathways. If an individual has optimistic traits then this may modulate the message accordingly and they would be less prone to reporting pain for example (Geers et al., 2007).

The nocebo effect is the reverse of placebo; the individual has negative expectations of a situation and is therefore likely to act accordingly. At its most extreme, nocebo effects have been attributed to causing death (Voelker, 1996). In these cases it is thought to be the belief that you are going to die that will increase the risk of death. If a substance is believed to increase pain, then it seems feasable to believe that this changes expectations and also anticipatory anxiety (Weich, 2009). It does not have to be a substance that may alter pain perception. In a study by Bayer et al. (1998) participants were lead to believe that a sham stimulator would be passing electrical current through their heads and that they may experience a head ache as a result of this. It was found that in the absence of a painful stimulus, participants still reported pain. Therefore, this study seeks to determine if such a relationship exists between pain expectation and muscle performance.

7.2 RESEARCH AIM

To investigate the influence of expectation of pain severity on neuromuscular performance in a maximal isometric knee extension task.

7.3 METHOD

7.3.1 PARTICIPANTS

Twenty-nine recreationally active male participants (mean ± SD; age = 22.8 ± 5.5 yr, height = 1.8 ± 0.2 m, mass = 84.1 ± 19.6 kg) volunteered for the study after giving written informed consent (see Appendix 1). The participants were free from any lower limb injury and were asked to refrain from any intense exercise or stimulant use for 24 hrs prior to testing. Ethical approval was granted from the School of Health's Ethics Committee in accordance with the declaration of Helsinki.

7.3.2 PROCEDURE

Equipment, measures and experimental protocol were as described in chapter 4 with all participants visiting the lab on two separate occasions for a familiarisation session and then experimental trial. Each participant undertook three MVCs in three conditions in a counterbalanced and randomised trial. The peak MVC in each trial was selected for measurement.

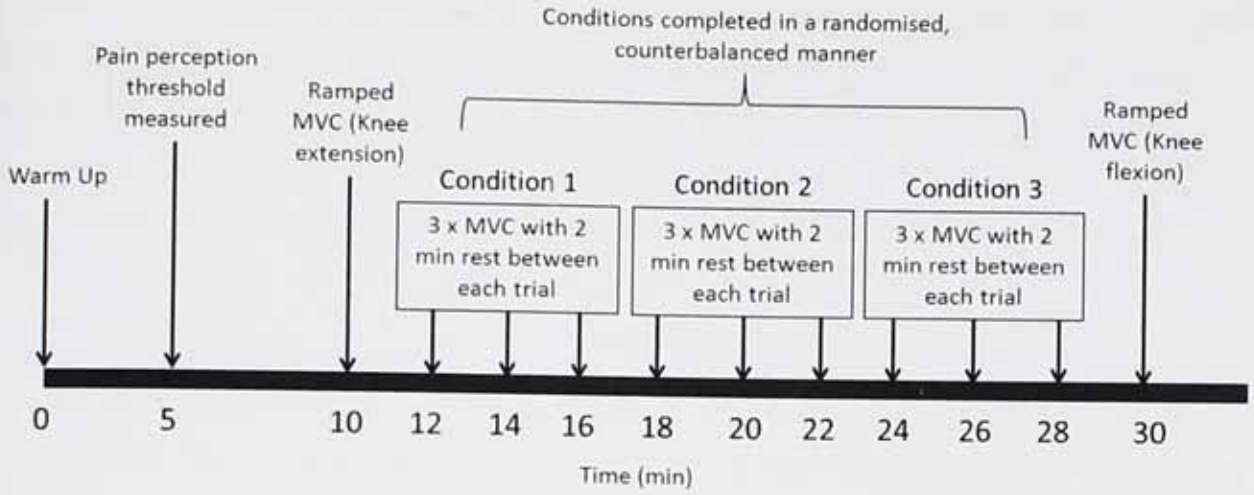


Figure 7.1. Timeline of protocol undertaken by each of the participants during the experimental trials.

7.3.3 EXPERIMENTAL CONDITIONS

Three conditions were performed within the trial in which participants were instructed to expect a certain level of pain: 100%, 200% or 300% of their pain perception threshold. However, in every case the actual percentage of pain threshold induced was 200%, thereby creating a nocebo condition in which participants received a level of pain lower than what they were expecting (told to expect 300%, received 200%), a placebo condition in which the level of pain experienced was greater than what was expected (told to expect 100%, received 200%) and a true condition in which they received the level of pain expected (200%). The GPD was strapped to the lower limb with the cleat positioned over the tibia one third of the distance from medial tibial epicondyle to medial malleolus. Pilot testing revealed that this site was the most sensitive area on the tibia accessible when the leg was positioned on the isokinetic dynamometer. The GPD was then inflated at 5 $mmHg \cdot s^{-1}$ to the previously established pain threshold, considered as the lowest experience of pain that a participant would recognise (Loeser & Treede, 2008). The participant was then instructed to perform the MVC trials. Immediately following each MVC, pressure from the cuff was released, while the GPD remained in position between each trial to ensure reliability of the position of the device. Immediately after each trial the participant was asked to indicate their perception of how much pain they experienced using a visual analogue scale (VAS).

7.3.4 DATA ANALYSIS

In each condition the peak MVC trial was selected for analysis. All values were normalised to the ramped contractions and reported as percentages; all data are reported as mean ± SD where normally distributed, and as median, IQR and range where non-parametric. Data within each

condition were examined for normality using the Kolmogorov-Smirnov test. Where the raw data were not normally distributed appropriate data transformation was used. Where no suitable transformation could be found, non-parametric tests were used. Post-hoc t-tests with Bonferroni correction were used where statistical significance was established. Statistical significance was accepted at $p < 0.05$ (see chapter 4 for full explanation of data analysis).

7.4 RESULTS

Due to the number of hypotheses being examined within this study, the results have been divided up for ease of understanding into; isometric knee extensor moment, EMG of the vastus lateralis, EMG of the semitendinosus and pain perception (VAS). Each section first reports normality of the data being tested (using the Kolmogorov-Smirnov test) and then any significant differences ($p < 0.05$) found as well as showing where those differences lie where appropriate.

7.4.1 JOINT MOMENT (FORCE)

Assumptions of normality were violated in the 200% condition, ($D(31) = 0.17$, $p < 0.05$), and 300% condition, ($D(31) = 0.14$, $p < 0.05$). Normality was confirmed in the 100%, ($D(31) = 0.07$, $p > 0.05$). No suitable data transforms could be found; therefore a Friedman's ANOVA was used to test the following:

H1: There is a difference in knee extensor moment in an isometric knee extension task when expected severity of pain induced via GPD is varied.

H0: There is no difference in knee extensor moment in an isometric knee extension task when expected severity of pain induced via GPD is varied.

No significant difference in force was found between the three conditions, ($\chi^2(2) = .452$, $p>0.05$). In comparison to the 100% condition (median = 104.7, IQR = 37.2, range = 87.2% MVC) median force increased by 6% in the 200% condition (median = 111.0, IQR = 23.8, range = 107.8% MVC), but decreased by 4.7% in the 300% condition (median = 99.8, IQR = 19.7, range = 108.0% MVC). In comparison to the 200% condition mean force was reduced by 10.8% in the 300% condition (see Figure 7.1).

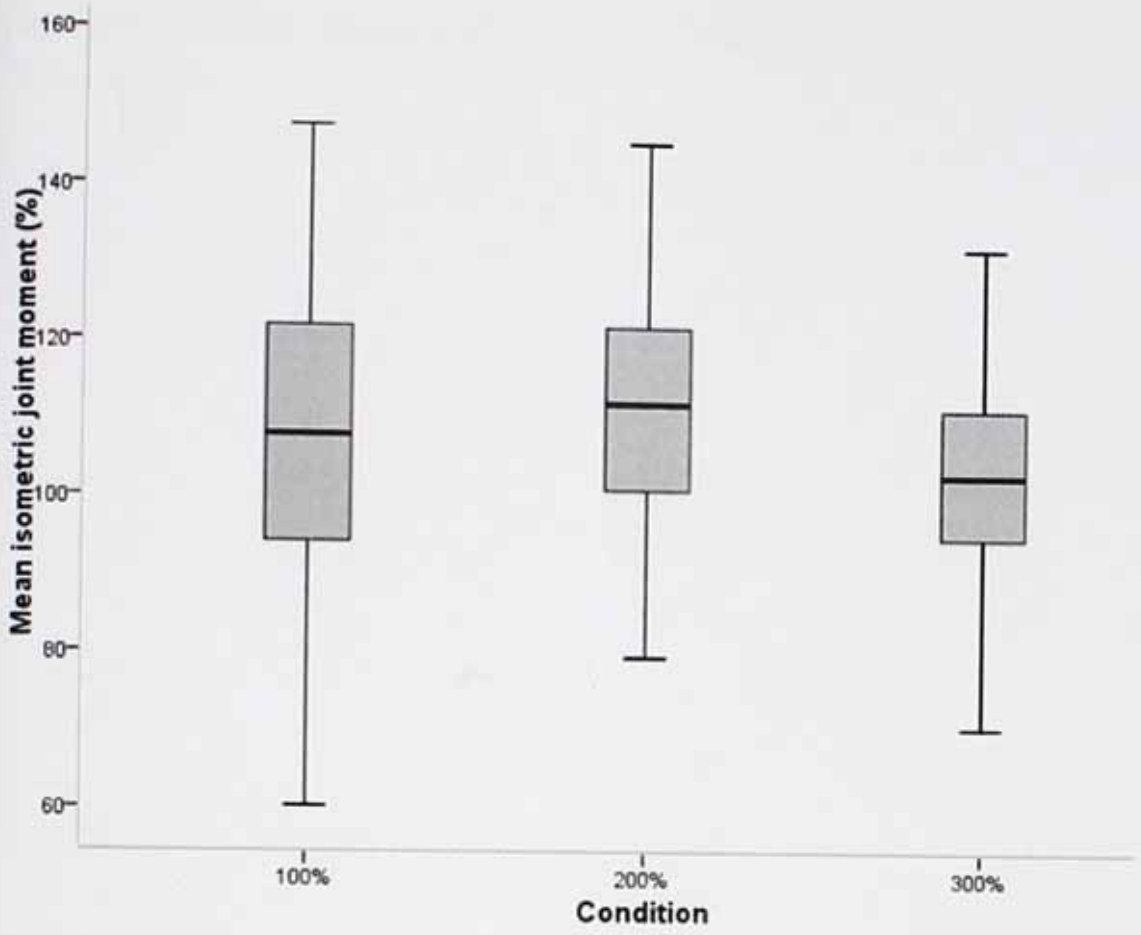


Figure 7.2. Normalised maximum voluntary isometric contractions produced in the 100%, 200% and 300% conditions. Data are presented as median, IQR & range (n=29). No significant difference was detected between the three conditions (p>0.05).

7.4.2 EMG VASTUS LATERALIS

The assumptions of normality were violated in the 300% condition, ($D(31) = .20$, $p < 0.05$). Normal distribution was reported in the 100% condition, ($D(31) = .16$, $p < 0.05$), and 200% condition, ($D(31) = .18$, $p < 0.05$). No appropriate transforms were found and therefore a Friedman's ANOVA was used to test the following:

H1: There is a difference in VL EMG activity in an isometric knee extension task when expectation of pain severity induced via GPD is varied.

H0: There is no difference in VL EMG activity in an isometric knee extension task when expectation of pain severity induced via GPD is varied.

No significant difference in VL EMG activity was reported between the three conditions, ($\chi^2(2) = .452$, $p>0.05$). In comparison to the 100% condition (median = 90.8, IQR = 39.3, range = 166.0% MVC) median EMG activity increased by 1.9% in the 200% condition (median = 92.6, IQR = 30.3, range = 170.6% MVC) and decreased by 3.3% in the 300% condition (median = 87.8, IQR = 30.8,

range = 193.8% MVC). There was a 5.2% reduction in the 300% condition in comparison to the 200% condition.

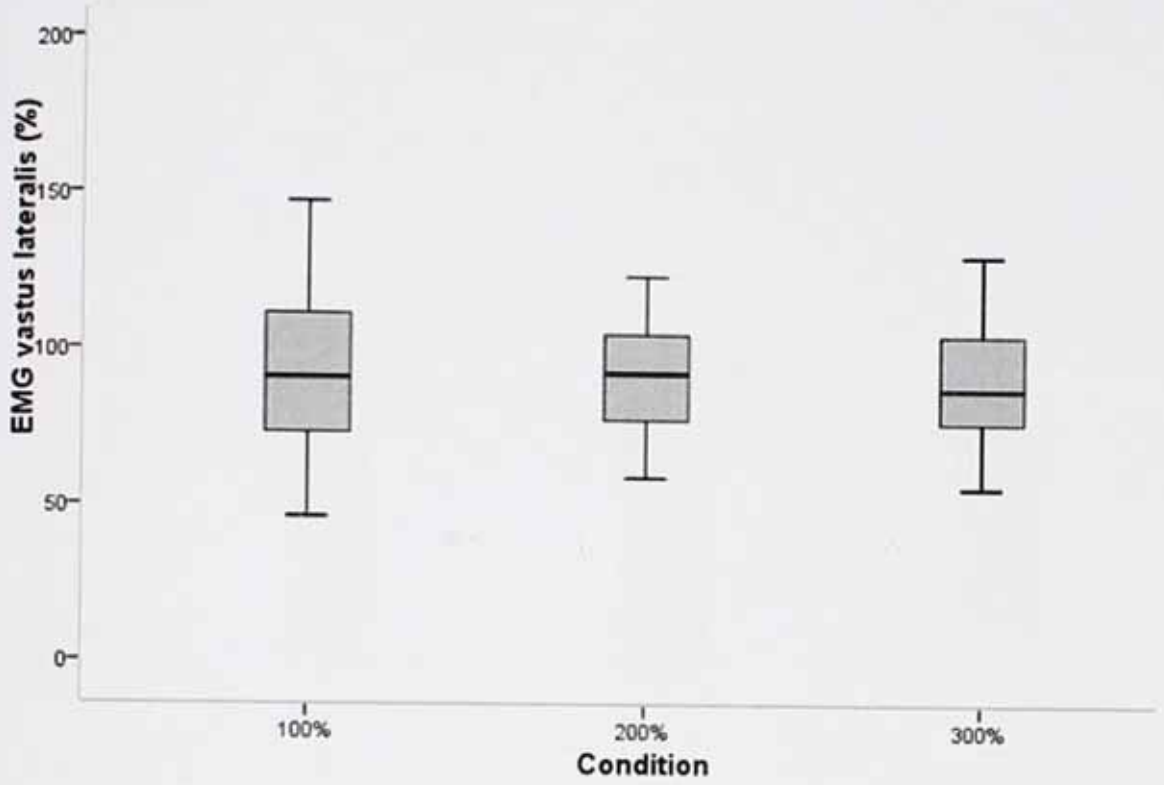


Figure 7.3. Normalised EMG activity recorded in vastus lateralis during the 100%, 200% and 300% pain perception threshold conditions. EMG recordings corresponding to the peak MVC produced over three trials in each condition were selected. Data are presented as median, IQR & range (n=29). There were no significant differences (*p>0.05*) detected in EMG activity in the VL between all three conditions.

7.4.3 EMG SEMITENDINOSUS

The assumptions of normality were violated in the 100% condition, ($D(31) = .21$, $p < 0.05$), 200% condition, ($D(31) = .15$, $p < 0.05$), and 300% condition, ($D(31) = .21$, $p < 0.05$). No appropriate transforms were found and therefore a Friedman's ANOVA was used to test the following:

H1: There is a difference in ST EMG activity in an isometric knee extension task when expectation of pain severity induced via GPD is varied.

H0: There is no difference in ST EMG activity in an isometric knee extension task when expectation of pain severity induced via GPD is varied.

There was no significant difference in ST EMG activity detected between the three conditions, ($\chi^2(2) = .258$, $p>0.05$). In comparison to the 100% condition (median = 7.2, IQR = 4.4, range = 27.6% MVC) median EMG activity increased by 12.5% in the 200% condition (median = 8.1, IQR

= 5.8, range = 24.0% MVC) and 6.9% in the 300% condition (median = 7.7, IQR = 3.8, range = 28.7% MVC). There was a 4.9% reduction in EMG activity in the 300% condition in comparison to the 200% condition (see Figure 7.4).

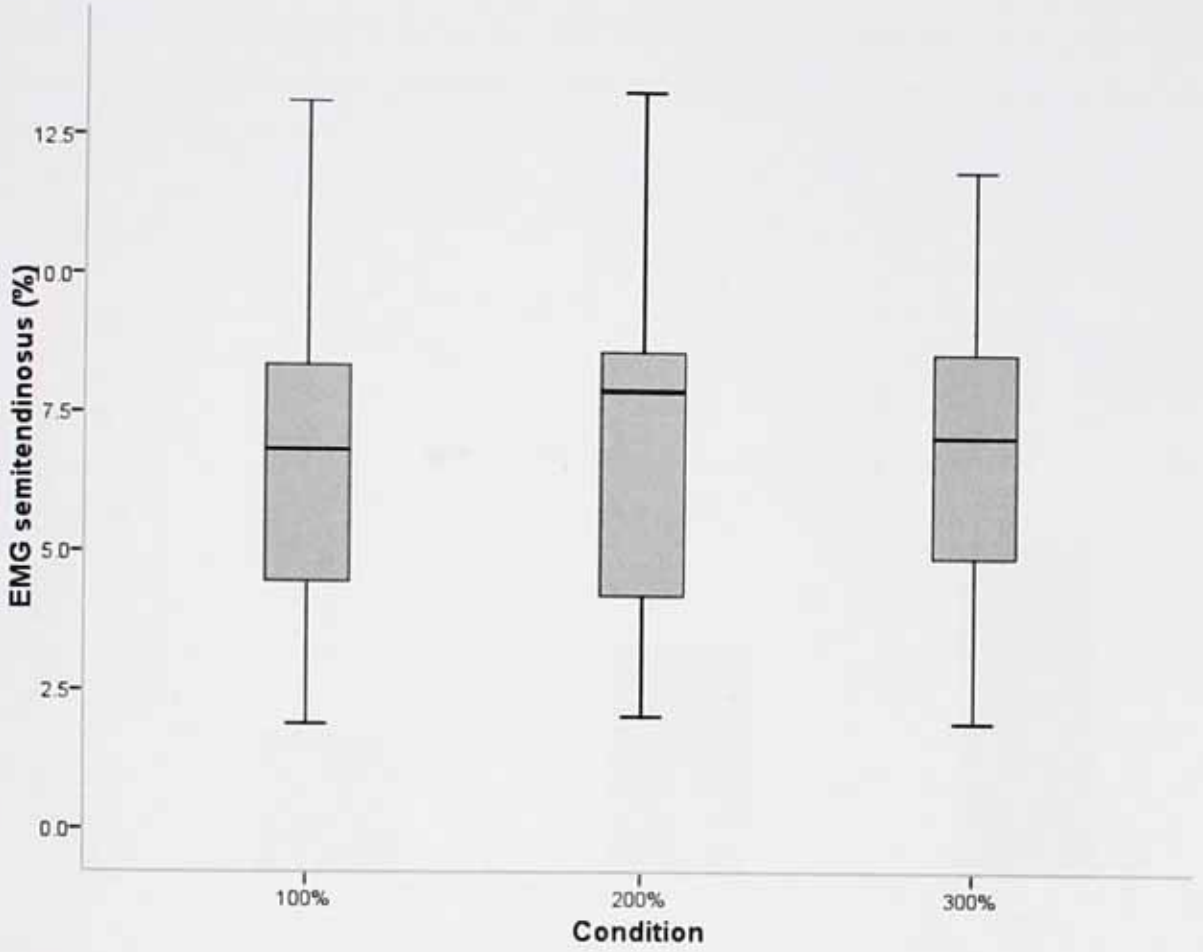


Figure 7.4. Normalised EMG activity recorded in semitendinosus during the 100%, 200% and 300% pain perception threshold conditions. EMG recordings corresponding to the peak MVC produced over three trials in each condition were selected. Data are presented as median, IQR & range (n=29). No significant differences were found in the EMG of the ST between the three conditions ($p > 0.05$).

7.4.4 PAIN PERCEPTION (VAS)

Data were found to be normally distributed in the 100%, ($D(27) = 0.10$, $p > 0.05$), 200% condition, ($D(27) = 0.14$, $p > 0.05$) and 300% condition, ($D(27) = 0.13$, $p > 0.05$). An analysis of variance with repeated measures (ANOVA) was therefore used to test the following:

H1: There is a difference in pain perception reported via VAS during an isometric knee extension task when expectation of pain severity induced via GPD is varied.

H0: There is no difference in pain perception reported via VAS during an isometric knee extension task when expectation of pain severity induced via GPD is varied.

There was a significant difference in pain perception reported across all three conditions ($F = 27.971$; $p < 0.05$). Post-hoc t-tests revealed a significant increase in pain perception between all conditions compared with the 100%, and 200% conditions ($p < 0.05$). In comparison to the 100% condition (46.1% ± 26.58%) there was a 16.8% increase in pain perception in the 200% condition (55.4 ± 22.9%), and 31.5% increase in the 300% condition (67.3 ± 21.1%). There was a 17.7% increase in pain perception in the 300% condition in comparison to the 200% condition (see Figure 7.4).

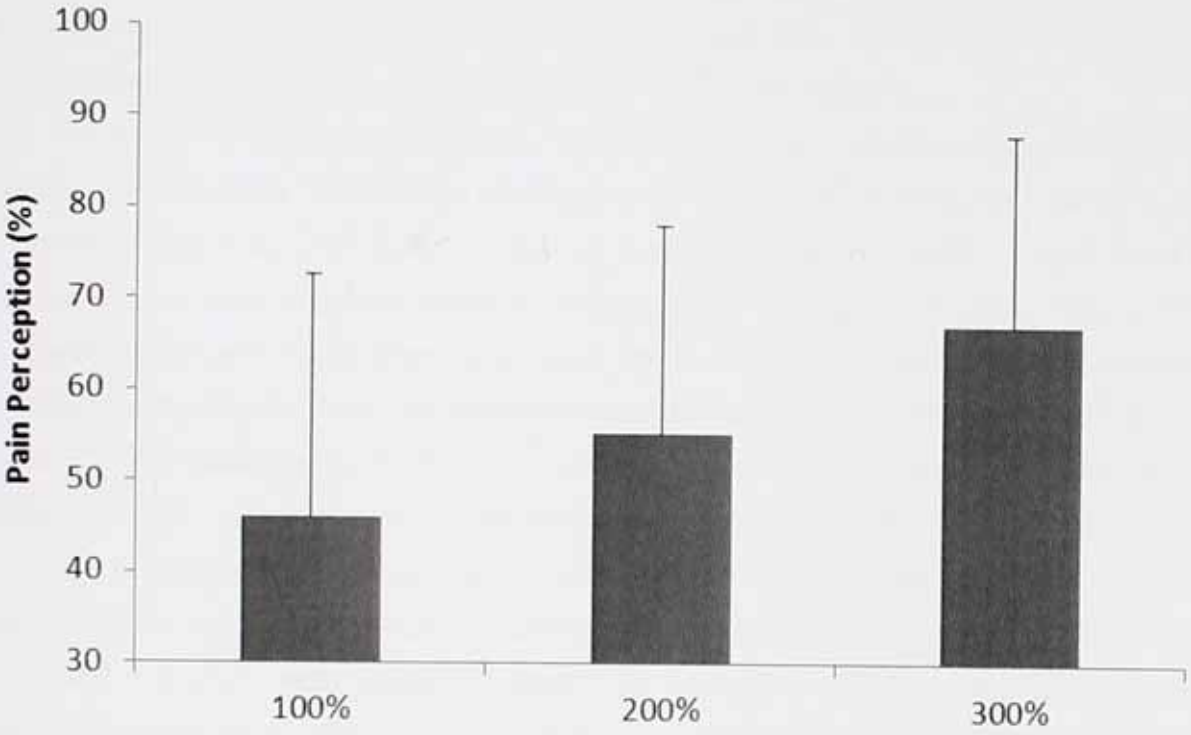


Figure 7.5. Pain perception as reported by participants on a visual analogue scale in the 100%, 200% and 300% conditions. Data are presented as mean ± SD (n=28). The location of significant differences is described in Table 7.1.

Table 7.1. Statistical differences in mean pain perception reported via VAS in each of the three pain conditions where significance is accepted at $p < 0.05$.

	100%	200%	300%
100%	-	p = 0.000	p = 0.000
200%	p = 0.000	-	p = 0.000
300%	p = 0.000	p = 0.000	-

7.5 DISCUSSION

There was a significant increase in the severity of pain reported by participants between the three conditions indicating that participants perceived there to be a difference in pain stimulus intensity, with the expected 100% condition recording the lowest perceived pain and the expected 300% the highest. However, whilst participants reported experiencing pain of differing severity, there was no significant difference in the level of force exerted or the EMG within the agonist or antagonist in all three conditions. This suggests that whilst it is possible to alter an individual's experience of pain, this in itself does not alter muscle performance; instead it is the absolute pain that is more likely to predict muscle performance.

In Chapter 5 the authors reported a decrease in muscle performance as pain severity increased using the same methods but where pain was induced to the true value stated to the participants (Wing et al., 2011a). In that study both moment and EMG activity were found to reduce as pain severity increased which is consistent with findings from Farina et al. (2004) who found pain severity to be inversely proportional to motor unit firing rate; thus indicating that higher levels of pain inhibit the performance of muscles, proportional to the level of pain induced. The mechanisms by which this occurs are unknown, but the pain adaptation model (Lund et al., 1991) and adaptation to pain theory (Hodges & Tucker, 2011) suggests that pain can cause a reduction in muscle activity as a protective mechanism to prevent further damage occurring, which has been supported by numerous studies (Arendt-Nielsen et al., 1996; Stohler et al., 1988). The pain adaptation model argues that there will be a decrease in agonist activity and increase in antagonist activity (Lund et al., 1991), however some studies report an increase in both agonist and antagonist rather than a converse relationship (Ervihla et al., 2004; Wing et al., 2011a) which may be explained by the location of muscle being examined or nature of the tasks (Hodges & Tucker, 2011). The series of studies conducted within this thesis reflect the current state of the literature well due to the varied nature of results when looking specifically at agonist and antagonist recruitment. Chapter 4 and 5 presented the finding that whilst EMG activity of the agonist increased when pain was induced so did EMG of the antagonist which is contrary to the Lund et al. proposal (1991). However the current study did find an increase in antagonist EMG in the presence of pain. This may lend further support to the Hodges & Tucker (2011) theory of a non-uniform response to pain where individual tasks and regions of the body may determine the way in which performance is altered.

The current study found a slight increase in antagonist EMG activity as pain increased from 100% to 200% pain perception threshold, but it was not found to be a significant increase. Further to this there was a reduction in the percentage increase between 100% to 300% conditions rather than a further increase which one may expect to see if the antagonist is being

activated in order to protect the joint as suggested by the pain adaptation model (Lund et al., 1991). However, it could also be suggested that the reason for inconsistency in the findings may be as a result of the limited use of the antagonist in the isometric task used in this study (Wing et al., 2011b). Both the pain adaptation model and adaptation to pain theory concern themselves with the neural circuitry between the muscles and brainstem, suggesting that as a result of a noxious stimulus there is an automatic, reflex-like, alteration in muscle activity (Peck, 2009). Whilst there is currently no theory that explains the relationship between severity of pain and muscle performance, it may be prudent to suggest that increased pain may cause the effect of pain to be strengthened as a greater severity may indicate a higher level of danger and consequently a larger reduction in performance which was found to be the case in Chapter 5 (Wing et al., 2011a).

In the current study, participants expected a change in the severity of pain induced between the three conditions due to the instructions they were given prior to the task which is evidenced by the significant differences found between each of the conditions as reported by VAS. This expectation caused them to alter the level of pain they reported experiencing which would suggest that they believed the pain stimulus was more or less painful that it actually was. In accordance with the study reported in Chapter 5 you would expect this to lead to a proportional decrease in performance if the mechanism by which muscle performance decreases is a conscious process; but it did not. Instead performance across all three conditions reduced from maximum, but did not differ significantly from one another, suggesting that neither placebo, nor nocebo effects altered muscle performance. It could be suggested that as the participants in the current sample group were 'recreationally active' rather than 'athletes', they have less control over muscle performance when pain is induced (Scott & Gijsbers, 1980) and therefore conscious control would not be as high as one might expect from an athletic sample. Had an athlete group been tested then different findings may have been reported and this should be considered for future work.

The pain adaptation model suggests that there is an automatic reaction to a noxious stimulus causing an altered movement (Lund et al., 1991), however there is also suggestion that the circuitry between muscles and brainstem could be modulated by higher brain activity which could therefore override the pain adaptation (Peck, 2009). This could result from a number of factors such as the motivation or competitiveness of the participant (Sternberg et al., 1998) which could potentially allow the individual to continue with normal or near-normal muscle activity despite the fact they are experiencing pain. Alternatively, fear of pain and its consequences would be expected to produce a greater adaptation than normal according to the pain adaptation model (Peck, 2009). This was not evident in the current study where muscle performance remained stable throughout the conditions despite a change in the reported

perception of pain. Therefore alternative explanations for the relationship between pain and muscle performance should be sought. The current findings would suggest that the most likely theory to explain the performance changes is the Hodges & Tucker (20122) adaptation to pain model, but this has not been fully developed yet and should form the basis for further research.

It is not possible from this study to make the assumption that expectation of pain severity (placebo or nocebo) cannot cause changes to muscle perfomance per se. The type of pain induced and the conditions in which this was carried out were inherently safe, thereby reducing any fear components of the pain experience (Finniss, 2005). In mainstream psychology literature, pain-related anxiety has been found to increase perceived intensity of a stimulus, whilst non-pain-related anxiety reduces it (Villemure & Bushnell, 2002) which is suggested to be as a result of motivational priming (Lang, 1995) or increased arousal (Logan et al., 2001), highlighting the importance of the emotional components of pain. In an applied setting where an athlete is experiencing pain, the type of pain experienced and the potential consequences of this (injury, fatigue, loss of performance) are not controlled and therefore would carry with them the associated emotional aspects of pain which may up-regulate an individual's sensitivity towards the stimulus and thereby alter their pain response (Weich, 2009). This may lead to different results to the ones reported in this study and should be examined further.

The type of participant recruited for this study should also be considered when discussing applicability to the athlete population. Participants were defined as recreationally active males which encompass those taking part in competitive sport, and those just exercising recreationally on a regular basis. Previous research has determined a significantly different mental attitude towards pain from normally active individuals and athletes (Nicholls & Polman, 2007; Ord & Gijsbers, 2003) which may mean that different results would be found by using an athlete sample. The fact that athletes also have a better awareness and understanding of proprioceptive input (Ellison et al., 1975) also increases the likelihood that they would be able to distinguish between a true change in level of pain induced and the sham conditions as described in this study. This would be an interesting area for further study as one may expect to see better control of performance from the athlete population.

If a substance is believed to increase pain, then it seems feasable to believe that this changes expectations and also anticipatory anxiety (Weich, 2009) which according to Peck (2009) would cause there to be a greater adaptation to the movement. However this was not the case in the current study where it appears the physical stimulus overrode the psychological aspect of expectation. The mechanisms by which this occurred remain unknown, but in light of the previously established suggestion that the mechnism is likely to be neurolonal rather than physiological (see Chapter 4), that it is not thought to be at peripheral level (see Chapter 6) and the apparent lack of conscious control able to influence performance in the current study, it

could be suggested that the location of any neuronal inhibition is likely to be at spinal level where a sub-conscious response would be responsible for alterations to muscle performance.

7.6 CHAPTER SUMMARY

This study demonstrated that absolute pressure pain rather than expectation of pain severity influenced muscle performance in an isometric knee extension task. Muscle performance (as measured by force exerted and EMG activity) did not change significantly between the three conditions, consistent with actual pressure exerted by the GPD. However perception of pain reported by participants increased significantly in line with the severity of pain they were told to expect. Therefore it is clear that expectation of pain can be manipulated through verbal cues, however this does not lead to a change in muscle performance. Instead, muscle performance more accurately traces changes in absolute pain. It has been previously proposed that the changes in performance associated with pressure pain are as a result of neuronal inhibition, but where this occurred could not be determined (see Chapter 4). This study suggests that reductions in performance are likely to be as a result of neuronal modification at a supra-spinal or spinal level, but not cortical. It is likely that sub-conscious mechanisms are responsible for changes in neural drive rather than any conscious control exerted by the individual.

8 SYNTHESIS OF FINDINGS

8.1 KEY FINDINGS

A series of progressive studies within this research demonstrated that the GPD is a method by which pain can be induced reliably without causing any mechanical or physiological changes to the muscle (chapter 3.2). The highly controllable nature of the GPD also makes it a useful tool in investigating changes associated with different severities of pain (chapter 3.5). Using the GPD to induce pain it was determined that even at very low levels pain reduces maximal muscle performance in the lower limb whether the stimulus was induced ipsi-laterally (chapter 4) or contra-laterally (chapter 6), suggesting that the mechanisms responsible for inhibition of muscle performance are unlikely to be at peripheral level. Inducing pain at perception threshold level produced a reduction in maximal force on an isometric knee extension task by 9% and 6% in chapters 4 and 5 respectively where pain was induced ipsi-laterally, and by 11% in chapter 6 where pain was induced contra-laterally. When the amount of pressure exerted by GPD was doubled from the previously established pain perception threshold of the individual, a further reduction in maximal force was reported, with an 18% decline reported, and subsequently a 21% reduction when pressure exerted was tripled from pain perception threshold (chapter 5).

The studies conducted did not only measure changes in isometric force, but also examined EMG activity within the vastus lateralis and semi-tendinosis in order to help establish possible mechanisms associated with force changes by tracing levels of neural activation. It was reported that where force declined in the presence of pain, there were also concomitant reductions in EMG activity within the target muscles, which coupled with the non-invasive methods used to induce pain, suggests that performance reductions may be associated to neural inhibition. The foundations of this research were based on the finding that pain induced by hypertonic saline injection reduced muscle performance in a range of tasks (Farina et al., 2002; Ervihla et al., 2004, 2005), however the level of pain could not be carefully controlled using these methods. In the current research pressure pain was induced at perception threshold level in studies 4, 5 and 6. This is the lowest stimulus perceived by an individual to be painful and yet it was possible to show a 6-11% decrease in performance associated with this low level of pain (chapters 4, 5 & 6).

A further variable considered important within the current research was how painful participants perceived the conditions to be. VAS scores were taken immediately following every painful trial in order to establish how painful the stimulus was perceived to be by the

participant relative to the pressure exerted via GPD. The GPD was able to accurately measure the amount of pressure exerted on the cleat which caused the pain stimulus, and thereby a baseline threshold level could be established. To date there has been no research evidence available to suggest what the relationship between pressure exerted and perceived pain intensity is which is important to this type of research. It was possible however to demonstrate that the amount of pressure exerted and the level of pain perceived by the individual were very closely associated and therefore the GPD appears to be a reliable method of inducing pain of varying severity (chapters 3, 4 & 5).

A major benefit of doing progressive studies using the same methods is that the results can be compared across studies rather than viewing them in isolation. Of particular interest was data collected in chapter 5 and chapter 7. Chapter 5 examined the difference in performance when three different pain severities were induced, whilst chapter 7 in essence replicated this, but rather than the pain severities changing, they remained constant whilst the participants' expectation of severity was manipulated. The rationale for the chapter 7 study was to determine the level of conscious involvement within the neuromuscular response to pain. If there was little conscious input one would expect the performance scores to be relatively stable and not change between conditions (as the stimulus actually remains constant). If there is conscious involvement then one would expect to see the performance scores decrease as expectation of pain increases (as was the case in chapter 5 when actual pain and expectation of pain severity were aligned). It was found that scores did not change between conditions, despite participants reporting higher or lower pain severity following the placebo and nocebo conditions. Chapter 7 demonstrated that it was possible to manipulate the pain severity a participant perceived through giving them false expectations; however this in turn did not alter motor response. It is unlikely therefore to be a conscious response as expectation did not influence performance output and is therefore not likely to be cortical. However, at what level this inhibition lies is as yet unknown and requires further investigation. More sophisticated techniques utilising MRI or EEG measures may be best placed to help understand brain activity and thereby conscious involvement in the response.

An interesting finding observed between the VAS data presented in chapters 5 and 7 was that the scores reported by participants for the 200% and 300% conditions in both studies were very similar (3% and 1% difference respectively) despite the fact that in the first study the absolute pain matched expectation of pain severity, whereas in the proceeding study absolute pain remained constant whilst expectation was manipulated to make participants believe pain severity would be higher or lower than it was. There is no data currently available in the sports setting that suggests how large a change one would have to report in order for there to be a

functional change in muscle performance, however Jackson et al. (2005) suggest that in order for there to be a clinical difference in terms of VAS reported and functional response in patients, there needs to be a minimum 13% change in VAS scores. Chapter 5 and 7 report very little difference in VAS scores reported in the 200% and 300% conditions and so based on the 13% minimum requirements stated by Jackson et al. (2005) one would not expect to see a change in muscle performance between the two studies; however a difference was found. An increase in absolute pain severity resulted in a decreased force output (chapter 5), but an increase in expected pain severity did not (chapter 7). An examination of the use of VAS scores in this type of research and what constitutes a functional difference in sport would be beneficial for future investigations.

Participants reported very similar perceptions of pain severity in both studies (indicated by the closely aligned VAS scores); however this did not appear to influence performance in the two studies. In contrast, the VAS scores reported for the 100% condition in each study were separated by 22%; participants perceived the severity of pain to be greater in chapter 7 than they did in chapter 5, which according Jackson et al. (2005) would suggest there should be a difference in performance between the two. This was found to be true. A possible explanation as to why there was a disparity between the severity of pain participants identified at 100% when absolute and expected pain severity did not match, but not in the 300% condition, could be due to perception of pain intensity being more sensitive to change at lower levels. It would appear that participants were more able to distinguish between actual and expected pain severity when the pressures exerted were lower than when they were set at higher levels which is in contrast to the suggestion of Kelly (2001a) who reported that there was no difference in minimal clinically significant difference of VAS scores within varying levels of pain severity (mild, moderate and high). It is clear that this finding cannot be fully explained at the present time, but it is felt that this is an important area for further research as the experience of pain is thought to be determined by both psychological and physiological influences (Hirsch & Liebert, 1998) and without a clear understanding of both of these components, effective strategies to overcome the deficits experienced in muscle performance as a result of pain cannot be developed.

This research has supported the findings of previous studies in which hypertonic saline injections were used to induce pain (Ervilha et al., 2004, 2005; Farina et al., 2004) in demonstrating that pain has a detrimental effect on muscle performance. Further to this it was able to establish that severity of pain experienced was proportional to performance deficit and suggests that there is a relationship that moves beyond a simple stimulus response. Whilst mechanisms could not be established using the current methods, it was possible to suggest that

there is a central response to pain and that neuromuscular performance is likely to be limited through neuronal inhibition in the presence of pain. This is also thought to be a subconscious response.

A further finding from this research was that the response to pain does not appear to be uniform. Whilst the methods employed within the studies remained largely similar, there were differences in the neuromuscular activity reported between studies which suggests a non-uniform response is most likely. Lund et al. (1990) proposed the pain adaptation model to explain performance deficits wherein neuromuscular activity is re-ordered to protect the joint. Here the agonist is expected to decrease EMG activity in the presence of pain, whilst the antagonist increases. This was supported by findings reported in chapter 4, however not in the subsequent studies (chapter 5, 6 & 7) where EMG in the antagonist also decreased when pain was induced. This suggests that the adaptation to pain model proposed by Hodges & Tucker (2011) may be a more suitable explanation for the association between pain and neuromuscular performance. Within this theory greater flexibility is afforded to the pain response, suggesting that muscle activation may increase, decrease or remain constant but that recruitment strategies may change to adapt to the painful condition. The current research was not able to demonstrate changes in recruitment strategy as this was beyond the scope of the investigation. Only two muscles were monitored, which was adequate for the purposes of this project but does not allow for recruitment strategy to be examined. However future work should consider what measurements are required to gain the best understanding of muscle activation and thereby develop the theory further to enable its use to predict likely muscle responses in specific tasks. This would then allow opportunity for intervention strategies to be effectively employed to help improve performance.

8.2 STRENGTHS AND LIMITATIONS

This research used a non-invasive method of pain induction that could be carefully controlled and induced remote from the target muscle which is a great strength of this work. This means that the results reported can be said to be due to the presence of pain rather than any physiological or mechanical changes to the muscle properties (Wing et al., 2011a). This is a clear difference to the previous studies reporting pain related changes to muscle performance, where invasive methods were used (Ervilha et al., 2005; Farina et al., 2004, 2005) and yet it supported their findings to show that pain can inhibit motor performance. The fact that the pain induced via GPD is transient in nature and non-invasive makes it an ethically appealing method to use for pain research. The findings from the current studies support its use for future work and demonstrate its versatility and reliability.

An examination of the effects of pain severity could be made due to the methods used, however the precise relationship between pressure exerted and perceived pain requires further examination if it is to be used as a way of quantifying severity. This research only investigated three different severities of pain up to three times the pain perception threshold (chapter 5) and therefore whether the similarity between scores would continue at higher severity remains unknown, as does the sensitivity to which this applies; for example if 150% pain perception threshold pressure was induced would this be distinguishable from 200%? In order to maximise the potential of this method of pain induction further research should be conducted related to this issue.

This was a laboratory based study and carefully controlled, meaning the findings cannot be directly applied to sports performance as the relationship between changes in force detected in laboratory testing and changes in a performance environment are not currently known (Paton & Hopkins, 2006). However they do provide evidence that pain is an important performance factor that warrants further investigation. The methods used in the current study were non-invasive and transient in nature (Ryan & Kovacic, 1966). In high performance sport the pain associated with performance in also transient (assuming there is not occurrence of injury). For example, if one were to stop cycling, running or rowing, the pain would subside almost instantly, therefore the participant is in effect in control of how much pain they tolerate and for how long. This method is therefore applicable to these types of events where pain is induced by performance itself, but less applicable to injury pain or DOMS which is more chronic in nature.

In order for movement to be functional it involves the co-ordination of multiple joints. The current study only investigated isolated muscle groups around the knee in order to be able to minimise the number of potentially confounding variables influencing the force output. This is not unusual in sports research where typically studies exploring determinants of explosive force production assess single joints such as the elbow flexors (Barry et al., 2005), knee extensors (Aagaard et al., 2002a; Andersen & Aagaard, 2006; de Ruiter et al., 2004) or ankle plantarflexors (Gruber et al., 2007; Del Balso &, Cafarelli, 2007) due to the need to control confounding variables. The applications of this task to the real performance environment and the mechanisms resulting in performance reductions need to be fully understood in order to develop intervention strategies to improve performance in the presence of pain. This could be achieved through manipulation of the task to include dynamic elements and subsequently further investigations carried out in a more realistic sports environment.

A further consideration is that of the participants recruited. The studies only investigated male participants which means that result cannot be directly applied to females (Fillingham et al., 1995; Hall & Davies, 1991; Riley et al., 1998). The participants were also defined as 'recreationally active' meaning that they participated in some form of exercise for a minimum of 2 hours per week which was thought to give an adequate sample group for an exploratory study. The type and level of participation was not part of the inclusion criteria for participating. Whilst studies have determined there to be a difference between non-athletes and athletes (Giesbrecht et al., 2005; Hall & Davies, 1991; Janal et al., 1994; Ryan & Kovacic, 1966), level of athlete (Scott & Gijsbers, 1981), and type of sport (Nideffer, 1978; Pen & Fisher, 1994; Williams, 1978) this research was exploratory in nature and it was therefore decided that type of athlete was not important at this stage. However, as pain perception has been found to be different amongst athletes in comparison to normally active controls (Tesarz et al., 2012) this could have important implications on the applicability of the findings to an athletic population. Future work should seek to replicate the methods in order to draw comparisons between athletes, non-athletes and type of sport and thereby gain a more comprehensive understanding of the mechanisms underpinning performance.

8.3 APPLICATION OF THE CURRENT RESEARCH AND FUTURE DIRECTIONS

This research has demonstrated that the presence of even low level pain significantly reduces neuromuscular performance (chapter 4, 5 & 6) which is in support of previous studies conducted using different methods to induce experimental pain (Farina et al., 2002; Ervilha et al., 2005; Ciubotariu et al., 2007). Furthermore it was able to show that as severity of pain increases there is a greater decline in performance (chapter 7). This has important implications to training and recovery strategies adopted in performance sport as it would suggest that by reducing the level of pain experienced by an athlete you would expect to see a performance improvement. High level sport requires an athlete to train at an intensity that often results in muscle soreness and therefore can cause limitations on their ability to perform on subsequent days as they will be experiencing pain. A number of strategies are already in use within elite sport to combat the effects of muscle soreness such as stretching, ultrasound, massage, antioxidant supplementation or use of anti-inflammatory drugs (Cheung et al., 2003). In order to compete at top levels athletes need to be able to train on consecutive days at a high intensity, therefore strategies enabling them to perform optimally on each of these training days and thereby reduce recovery time could be hugely beneficial and further investigation into their use should be undertaken.

The current findings lend support to the rationale behind strategies such as ice baths (Dolan et al., 1997; Rheinecker et al., 1998) and cryotherapy treatment (Eston & Peters, 1999; Howatson & van Someren, 2003) which aim to reduce the effects of delayed onset muscle soreness experienced by athletes following periods of intense training or competition (Sellwood et al., 2007). Whilst this research was not DOMS related, it does support the research already conducted which has used DOMS as the method of pain induction (Dannecker et al., 2008) so similarities can be drawn from it provided caution is exercised with regard to generalisability. Evidence for the effectiveness of these techniques to speed up physiological recovery is equivocal (Sellwood et al., 2007), however there is evidence to suggest that cold therapy following exercise can reduce the perception of muscle soreness up to 48 hours post exercise (Bailey et al., 2007; Eston & Peters, 1999; Howatson & van Someren, 2003) which may then allow athletes to train at a higher intensity than they would without any form of recovery strategy. Further work is needed to determine what strategies are effective in reducing the severity of pain experienced and thus allowing optimal performance.

Studies examining what constitutes a worthwhile gain in performance within various sports have reported that marginal gains are considered beneficial. An improvement of 0.3-0.5% in track athletics (Hopkins, 2005), 0.9-1.5% in field athletics (Hopkins, 2005), ~0.5% in triathlon (Paton & Hopkins, 2005) and 0.5-0.6% in track cycling (Paton & Hopkins, 2006) are thought to have an impact on performance, which further supports the rationale for this project as it demonstrates how small a change can be significant. The current research suggests that the presence of pain reduces muscle performance in an isometric knee extension task by a comparatively large figure (6-11%) which one would expect to influence sporting performance. However one would not expect to see a reduction of 6-11% as there are many other factors responsible for sports performance other than maximal force generation which were not accounted for in this study.

An alternative application of the current work relates to rehabilitation from injury. It is common for patients to experience pain as a result of injury and this can in turn limit range of motion around a joint (Geisser et al., 2000; Hopman et al., 1997; Onder et al., 2006) and thereby inhibit normal daily functioning, or in the case of an athlete recovering from injury, limit ability to perform in their sport (Wilk et al., 2002). It is therefore beneficial to the individual to regain normal range of motion as soon as possible following injury. Recreational and competitive sports people are highly likely to experience injury (Kraus et al., 1984) as a result of their involvement in sport. According to the current research, strategies that could help to deal with the pain associated with movement would help to speed up this process. The findings reported in this research have demonstrated that in the presence of pain there is a reduction of EMG

activity in the agonist which supports the pain adaptation model (Lund et al., 1991) and adaptation to pain theory (Hodges & Tucker, 2011) but there were no uniform responses reported. It is therefore important that further work is carried out to understand the implications of this and what mechanisms are responsible so that they can be overcome and allow the athlete to train and perform optimally.

This research was able to demonstrate that an increase in severity of stimulus will inhibit muscle performance; however where expectation of pain severity increased but stimulus intensity remained the same there was no change in performance. This suggests that changing an individual's perception or expectation of pain may not have any effect on their ability to perform a task; instead an actual reduction in pain is required. This study did however demonstrate the ease with which an individuals' expectation of pain severity could be manipulated, which is consistent with work within a clinical setting (Bucknall et al., 2001). This is an important implication for practice, as those working with injured athletes can have a significant impact on the way in which the athlete perceives their injury and therefore their response to rehabilitation. Further work to establish methods whereby pain can be reduced should be carried out, as well as establishing if perception can alter physical response. Currently extended use of anti-inflammatory drugs following injury is likely to be the most effective strategy allowing the individual to regain full range of motion but this comes with associated risks or re-injury and ethical issues. Therefore psychological techniques should be investigated further.

The involvement of psychology within the pain experience warrants further investigation (Addison et al., 1998; Porro et al., 2002; Wing et al., 2011b). More sophisticated methods in which neuronal activity can be monitored and measured should be employed in future research to further understanding of these processes. The current work suggested that there was little conscious involvement in determining performance within the trials. However this was based on the role of manipulating expectations and then measuring performance, there were no interventions employed to help individuals overcome pain or investigations into the subjective experience of the individual. The affective dimension of pain is thought to be an important determinant of the pain experience (Hirsch & Liebert, 1998) and therefore something that should not be dismissed. For example, the meaning an individual attaches to a particular stimulus has been shown to alter the pain they experience (Arntz & Claassens, 2004) but how this may influence physical performance is unknown and would be an interesting topic for further examination. Psychological coping strategies have been employed within sport to help reduce negative effects of pain (Schomer, 1987), had one of these been implemented within the

current research this may have impacted the results and would be an interesting study in the future.

The studies conducted within this project were all carried out in a laboratory setting which was important as it enabled careful control of the variables under investigation and lead to some interesting findings that would not have been possible by other means. The pain induced within this study was experimental and therefore remains fundamentally different to non-experimental pain as the danger elements are limited (Hudson et al., 2006) and therefore the task requirements may have been deemed inherently safe by participants (Pen & Fisher, 1994). This may have altered the response they gave to the pain stimulus as the pain experience in highly controlled conditions do not carry the same threat as those within the field and this can change the overall experience and therefore response given (Friedman et al., 1985; Hudson et al., 2006). As well as the perception that this was a safe environment for the participants, it is also not possible to infer performance changes from a laboratory experiment such as this where there were only limited measurements taken (force & EMG). This does not account for other variables influencing sports performance such as technique, competition and environmental factors. Whilst a 6-11% decrease in muscle performance was reported in the studies conducted using pain at perception threshold level, this is only in one muscle group and doing a very simple task involving effort only around one joint. If the individual were to do a more complex, multi-joint task which is more realistic to the sports environment there may have been different results. Therefore similar studies in which simulated competition environments and more sport specific tasks are investigated would increase the validity of findings to sport.

8.4 CHAPTER SUMMARY

The studies presented within this thesis have supported the findings of previous work investigating the effect of pain on muscle performance (Farina et al., 2002; Farina et al., 2008; Madeleine et al., 2006). However what makes this project unique is the technique of pain induction utilised that does not interfere with physiological or mechanical properties of the muscle. This means that the performance reductions reported were most likely due to the pain stimulus itself. Furthermore the controllable nature of these methods enabled investigation of the influence of pain severity on muscle performance and demonstrated that as severity increases performance decreases. The scope of the research did not seek to determine exact mechanisms by which performance is inhibited, but exploratory work was conducted which suggested mechanisms are likely to be centrally controlled and sub-conscious. Future work should build on these findings in establishing where inhibition of neuromuscular activation

occurs and whether this is applicable to different settings and in other tasks. It is hoped that this will allow for interventions to be developed within sport to help athletes cope with pain and overcome its limiting effects, but this can only be achieved when the mechanisms are better understood.

REFERENCE LIST

Aagaard, P., Simonsen, E.B., Andersen, J.L., Magnusson, P. & Dyhre-Poulsen, P. (2002). Increased rate of force development and neural drive of human skeletal muscle following resistance training. *J Appl Physiol*, 93,1318–1326.

Addison, T., Kremer, J., & Bell, R. (1998). Understanding the psychology of pain in sport. *Irish Journal of Psychology*, 19(4). 486-503.

Ahles, T. A., Blanchard, E. B., & Leventhal, H. (1983). Cognitive control of pain: Attention to the sensory aspects of the cold pressor stimulus. *Cognitive Therapy and Research*, 7, 159–178.

Alkner, B.A., Tesch, P.A. & Berg, H.E. (2000). Quadriceps EMG/force relationship in knee extension and leg press. *Medicine and science in sports and exercise*, 32 (2), 459-463.

Anderson, D.B. & Pennebaker, J.W. (1980). Pain and pleasure: alternative interpretations for identical stimulation. *Eur J Soc Psychol*, 10, 207–12.

Andersen, L.L. & Aagaard, P. (2006). Influence of maximal muscle strength and intrinsic muscle contractile properties on contractile rate of force development. *European journal of applied physiology*, 96 (1), 46-52.

Anshel, M.H., & Russell, K.G. (1994). Effect of aerobic and strength training on pain tolerance, pain appraisal and mood of unfit males as a function of pain location. *Journal of Sports Sciences*, 12, 535-547.

Arendt-Nielsen, L., Graven-Nielsen, T., Svanner, H. & Svensson, P. (1996). The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain*, 64,231–240.

Arendt-Nielsen, L. & Graven-Nielsen, T. (2008). Muscle Pain: Sensory Implications and Interaction With Motor Control. *Clinical Journal of Pain*, 24 (4), 291-298.

Arendt-Nielsen, L. & Falla, D. (2009). Motor control adjustments in musculoskeletal pain and the implications for pain recurrence. *Pain*, 142 (3), 171-2.

Arnstein, P. (2003). The Placebo Effect. *Seminars in Integrative Medicine*, 1 (3), 125-135.

Arntz, A. & Claassens, L. (2004). The meaning of pain influences its experienced intensity. *Pain*, 109(1-2), 20–5.

Ashton-Miller, J.A., McGlashen, K.M., Herzenberg, J.E., Stohler, C.S. (1990). Cervical muscle myoelectric response to acute experimental sternocleidomastoid pain. *Spine*, 15, 1006–1012.

Backman, E., Bengtsson, A., Bengtsson, M., Lennmarken, C. & Henriksson, K.G. (1988). Skeletal muscle function in primary fibromialgia: effect of regional sympathetic blockade with guanethidine. *Acta Neurol Scand*, 77, 187–191.

Bailey, D.M. (2007). Influence of cold-water immersion on indices of muscle damage following prolonged intermittent shuttle running. *Journal of sports sciences*, 25(11), 1163–70.

Barry, B.K., Warman, G.E. & Carson, R.G. (2005). Age-related differences in rapid muscle activation after rate of force development training of the elbow flexors. *Experimental brain research*, 162, (1), 122-132.

Bartholomew, J. B., Brewer, B., Linder, D. E., Cornelius, A., & Bart, S. M. (1998). A psychometric evaluation of the Sports Inventory for Pain. *The Sport Psychologist*, 12, 29-39.

Basmajian J.V. & DeLuca, C.J. (1985). *Muscles alive, their function revealed by electromyography*. Baltimore: Williams and Wilkins.

Bayer, T.L. (1998). The role of prior pain experience and expectancy in psychologically and physically induced pain. *Pain*, 74(2-3), 327–31.

Beck, T.W., Housh, T.J., Fry, A.C., Cramer, J.T., Weir, J.P., Schilling, B.K., Falvo, M.J. & Moore, C.A. (2008). The influence of myosin heavy chain isoform composition and training status on the patterns of responses for mechanomyographic amplitude versus isometric torque. Journal of strength and conditioning research, 22(3), 818-25.

Behnke, R.S. (2006). *Kinetic Anatomy* (2nd Ed.). Illinois; Human Kinetics.

Benedetti, F., Pollo, A., Lopiano, L., Lanotte, M., Vighetti, S. & Rainero, I. (2003). Conscious expectation and unconscious conditioning in analgesic, motor and hormonal placebo/nocebo responses. *J Neurosci*, 23, 4315–4323.

Benedetti, F., Lanotte, M., Lopiano, L. & Colloca, L. (2007). When words are painful: unraveling the mechanisms of the nocebo effect. *Neuroscience*, 147, 260–71.

Benson H. (1996). *Timeless healing: the power and biology of belief*. New York: Scribner.

Benson, H. (1997). The nocebo effect: History and physiology. *Prev. Med.*, 26, 612-615.

Bilodeau, M., Arsenault, A.B., Gravel, D. & Bourbonnais, D. (1991). EMG power spectra of elbow extensors during ramp and step isometric contractions. *Eur J Appl Physiol*, 63, 24–28.

Birch, L., Graven-Nielsen, T., Christensen, H. & Arendt-Nielsen, L. (2000). Experimental muscle pain modulates muscle activity and work performance differently during high and low precision use of a computer mouse. *Eur J Appl Physiol*, 83, 492–498.

Bloom, W. & Fawcett, D. W. (1968). *A textbook of Histology (*9th ed.). Saunders, Philadelphia.

Bostock, H., Baker, M. & Reid, G. (1991). *Changes in excitability of human motor axons underlying post-ischaemic fasciculations: evidence for two stable states.* J Physiol, 441, 537–557.

Brennum, J., Kjeldsen, M., Jensen, K. & Jensen, T.S. (1989). Measurements of human pressure-pain thresholds on fingers and toes. Pain, 38, 211-217.

Brewer, B. W., & Karoly, P. (1989). Effects of attentional focusing on pain perception. *Motivation and Emotion*, 13, 193–203.

Brewer, B. W., Karoly, P., Linder, D. E., & Landers, D. M. (1990a). Validation of a procedure to induce pain of varied intensities. *Research Quarterly for Exercise and Sport*, 61, 268-271.

Brewer, B. W., Van Raalte, J. L., & Linder, D. E. (1990b). Effects of pain on motor performance. *Journal of Sport and Exercise Psychology*, 12, 353-365.

Brown, C.A., Seymour, B., El-Deredy, W. & Jones, A.K. (2008). Confidence in beliefs about pain predicts expectancy effects on pain perception and anticipatory processing in right anterior insula. *Pain*, 139, 324–332.

Bucknall, T., Manias, E., & Botti, M. (2001). Acute pain management: implications of scientific evidence for nursing practice in the postoperative context. *International Journal of Nursing Practice*, 7, 266-273.

Burden, A. & Bartlett, R. (1999). Normalisation of EMG amplitude: an evaluation and comparison of old and new methods. *Medical engineering & physics,* 21 (4), 247-257.

Butler, J.E., McKenzie, D.K. & Gandevia, S.C. (1999). Discharge properties and recruitment of human diaphragmatic motor units during voluntary inspiratory tasks. *J Physiol,* 518 (3), 907–20.

Byl, N.N. & Sinnott, P.L. (1991). Variations in balance and body sway in middle-aged adults: subjects with healthy backs compared with subjects with low back dysfunction. *Spine,* 16, 325–30.

Charman, R. (2004). *Pain and nociception: mechanisms and modulation in sensory context.* In Boyling, J. & Palastanga, N. *Grieve's modern manual therapy: The vertebral column (2nd Ed.).* New York: Churchill Livingstone.

Cheung, K., Hume, P.A. & Maxwell, L. (2003). Delayed onset muscle soreness: Treatment strategies and performance factors. *Sports Medicine,* 33 (2), 145-164.

Ciubotariu, A., Arendt-Nielsen, L. & Graven-Nielsen, T. (2007). Localized muscle pain causes prolonged recovery after fatiguing isometric contractions. *Experimental brain research.* 181 (1), 147–58.

Cowan, S.M, Bennell, K.L., Hodges, P.W., Crossley, K.M. & McConnell, J. (2001). Delayed onset of electromyographic activity of vastus medialis obliquus relative to vastus lateralis in subjects with patellofemoral pain syndrome. *Archives of Physical Medicine and Rehabilitation,* 82, 183-189

Craig, K. D. & Coren, S. (1975). Signal detection of social modelling influences on pain expressions. *Journal of Psychosomatic Research,* 19, 105-112.

Craig, K. D. & Weiss, S. M. (1971). Vicarious influences on pain threshold determinants. *Journal of Personality and Social Psychology,* 18, 53-59.

Cram, J.R. & Steger, J.C. (1983). EMG scanning in the diagnosis of chronic pain. *Biofeedback Self Regulat,* 8, 229–41.

Crombez, G., Eccleston, C., Baeyens, F., Eelen, P. (1998a). Attentional disruption is enhanced by the threat of pain. *Behaviour Research and Therapy,* 36, 195-204.

Crombez, G., Eccleston, C., Baeyens, F., Eelen, P. (1998b). When somatic information threatens, catastrophic thinking enhances attentional inference. *Pain,* 75, 187-198.

Crombez, G., Eccleston, C., Baeyens, F., van Houdenhove, B., & van den Broecke, A. (1999). Attention to chronic pain is dependent upon pain-related fear. *Journal of Psychosomatic Research,* 47, 403-410.

Dannecker, E. A., Price, D. D., O'Connor, P. D., & Robinson, M. E. (2008). Appraisals of pain from controlled stimuli: Relevance to quantitative sensory testing. *British Journal of Health Psychology,* 13, 537-550.

De Luca, C.J. (1985). Control properties of motor units. *Journal of Experimental Biology,* 115, 125-136.

Del Balso, C., Cafarelli, E. (2007). Adaptations in the activation of human skeletal muscle induced by short-term isometric resistance training. *J Appl Physiol,* 103, 402–411.

Del Santo, F., Gelli, F., Spidalieri, R. & Rossi, A. (2007). Corticospinal drive during painful voluntary contractions at constant force output. *Brain Res,* 1128, 91–98.

Derbyshire, S. W., Jones, A. K., Gyulai, F., Clark, S., Townsend, D., & Firestone, L. L. (1997). Pain processing during three levels of noxious stimulation produces differential patterns of central activity. *Pain,* 73, 431-445.

Derbyshire, S. W., Jones, A. K., & Vogt, B. A. (1998). Pain and Stroop interference tasks activate separate processing modules in anterior cingulated cortex. *Experimental Brain Research,* 118, 52-60.

Descartes R. (1664). Treatise of man. In Hall, T.S., *Translator.* Cambridge, MA: Harvard University Press.

Desmedt, J.E. & Godaux, E. (1977). Ballistic contractions in man: characteristic recruitment patters of single motor units of the tibialis anterior muscle. *J Physiol,* 264, 673-693.

de la Fuente-Fernández, R., Schulzer, M. & Stoessl, A.J., (2004). Placebo mechanisms and reward circuitry: clues from Parkinson's disease. *Biol. Psychiatry,* 56, 67–71.

De Luca, C.J. (1997). The use of surface electromyography in biomechanics. *J Appl Biomech,* 13, 135–163.

de Ruiter, C.J., Kooistra, R.D., Paalman, M.I. & de Haan, A. (2004). Initial phase of maximal voluntary and electrically stimulated knee extension torque development at different knee angles. *J applied physiology,* 97 (5), 1693-1701.

de Ruiter, C.J., Van Leeuwen, D., Heijblom, A., Bobbert, M.F. & de Haan, A. (2006). Fast unilateral isometric knee extension torque development and bilateral jump height. *Medicine and science in sports and exercise,* 38 (10), 1843- 1852.

de Ruiter, C.J., Vermeulen, G., Toussaint, H.M. & de Haan, A. (2007). Isometric kneeextensor torque development and jump height in volleyball players. *Medicine and science in sports and exercise,* 39 (8), 1336-1346.

Devor, M. & Wall, P. D. (1978). The physiology of sensation after peripheral nerve injury, regeneration and neuroma formation. In Waxman, S.G. *The Physiology and Pathobiology of Axons.* New York: Raven Press.

Dickensen, A.H. (2002). Gate control theory of pain stands test of time. *British Journal of Anaesthesia,* 88 (6), 756-757.

Dolan, M.G., Thorton, R.M., Fish, D.R. & Mendel, F.C. (1997). Effects of Cold Water Immersion on Edema Formation After Blunt Injury to the Hind Limb of Rats. *Journal of Athletic Training.* 32, 233-237.

Eaker, E., Pinsky, J. & Castelli ,W.P. (1992). Myocardial infarction and coronary death among women: psychosocial predictors from a 20 year follow-up of women in the Framingham Study. *Am J Epidemiol.,* 135, 854–64.

Eccles, J. C. (1964). Presynaptic inhibition in the spinal cord. In Eccles, J.C. & Schade, J.P. Progress in Brain Research (Vol. 12). Amsterdam: Elsevier.

Eccleston, C., & Crombez, G. (1999). Pain demands attention: a cognitive-affective model of the interruptive function of pain. *Psychological Bulletin,* 125, 356-366.

Egan, S. (1987). Acute pain tolerance among athletes. *Canadian Journal of Sport Sciences*, 12,175-178.

Ellison, K. & Freischlag, J. (1975). Pain tolerance, arousal, and personality relationships of athletes and non-athletes. *Research Quarterly*, 46, 250-255.

Enoka, R. M. (1995). Morphological features and activation patterns of motor units, *Journal of Clinical Neurophysiology*, 12 (6), 538–559.

Ervilha, U.F. (2004a). The effect of muscle pain on elbow flexion and coactivation tasks. *Experimental brain research*, 156(2), 174–182.

Ervilha, U.F. (2005). Effect of load level and muscle pain intensity on the motor control of elbow-flexion movements. *European journal of applied physiology*, 92(1-2), 168–175.

Ervilha, U. F., Farina, D., Arendt-Nielsen, L., Graven-Nielsen, T. (2005). Experimental muscle pain changes motor control strategies in dynamic contractions. *Experimental Brain Research*, 164 (2), 215–224.

Eston, R. & Peters, D. (1999). Effects of cold water immersion on the symptoms of exercise induced muscle damage. *Journal of Sports Sciences*, 17 (3), 231-238.

Falla, D., Arendt-Nielsen, L. & Farina, D. (2009). The pain-induced change in relative activation of upper trapezius muscle regions is independent of the site of noxious stimulation. Clinical Neurophysiology, 120, 150-157.

Falla, D., Farina, D., Dahl, M.K. & Graven-Nielsen, T. (2007). Muscle pain induces task-dependent changes in cervical agonist/antagonist activity. *J Appl Physiol*, 102, 601– 609.

Falla, D. & Farina, D. (2008). Neuromuscular adaptation in experimental and clinical neck pain. *Journal of Electromyography and Kinesiology*, 18 (2), 255-261.

Farina, D. & Merletti, R. (2000). Comparison of algorithms for estimation of EMG variables during voluntary isometric contractions. *J Electromyogr Kinesiol*, 10, 337–350.

Farina, D., Fosci, M. & Merletti, R. (2002). Motor unit recruitment strategies investigated by surface EMG variables, 235–247.

Farina, D., Merletti, R., Indino, B., Nazzaro, M. & Pozzo, M. (2002). Cross-talk between knee extensor muscles. Experimental and model results. *Muscle Nerve*, 26, 681–695.

Farina, D., Merletti, R. & Enoka, R.M. (2004). The extraction of neural strategies from the surface EMG. *J Appl Physiol*, 96, 1486–1495.

Farina, D. (2004). Effect of experimental muscle pain on motor unit firing rate and conduction velocity. *Journal of neurophysiology*, 91(3), 1250–9.

Farina, D., Arendt-Nielsen, L. & Graven-Nielsen, T. (2005). Experimental muscle pain decreases voluntary EMG activity but does not affect the muscle potential evoked by transcutaneous electrical stimulation. *Clin Neurophysiol*, 116(7), 1558–65.

Farina, D., Arendt-Nielsen L, Merletti R, Graven-Nielsen T (2004) Effect of experimental muscle pain on motor unit Wring rate and conduction velocity. *Journal of Neurophysiology*, 91, 1250–1259.

Farina, D. (2008). The pain-induced decrease in low-threshold motor unit discharge rate is not associated with the amount of increase in spike-triggered average torque. *Clinical neurophysiology*, 119(1), 43–51.

Field, A. (2009). *Discovering statistics using SPSS* (3rd Ed.). SAGE Publications; London.

Fields, H.L. (2000). Pain modulation: expectation, opioid analgesia and virtual pain. *Prog Brain Res*, 122, 245–53.

Fields, H.L., & Price, D.D. Placebo analgesia. In: Wall, P.D. & Melzack, R. *Textbook of pain*. Churchill Livingstone.

Fillingim, R.B. & Maixner, W. (1995). Gender differences in the responses to noxious stimuli. *Pain Forum*, 4, 209-221.

Finniss, D.G. & Benedetti, F. (2005). Mechanisms of the placebo response and their impact on clinical trials and clinical practice. *Pain*, 114, 3–6.

Fitts, R.H. & Holloszy, J.O. (1976). Lactate and contractile force in frog muscle during development of fatigue and recovery. *Am J Physiol*, 231,430–433.

Frey, I.H. (1991). Social risk and the meaning of sport. *Sociology of Sport Journal*. 8, 136-145.

Gandevia, S. (2001). Spinal and supraspinal factors in human muscle fatigue. *Physiological Reviews*, 81(4), 1725-89.

Ganong, W. F. (1971). *Review of Medical Physiology* (5th ed.). Lange Medical Publications, Los Altos, California.

Gardiner, P.F. (2001). *Neuromuscular aspects of physical activity*. Human Kinetics; Champaign, IL.

Garnett R, Stephens JA (1981) Changes in the recruitment threshold of motor units produced by cutaneous stimulation in man. *The Journal of Physiology*, 311, 463-473.

Gauron, E. F., & Bowers, W. A. (1986). Pain control techniques in college-age athletes. *Psychological Reports*, 59, 1163-1169.

Geers, A.L. (2007). Further evidence for individual differences in placebo responding: an interactionist perspective. *Journal of psychosomatic research*, 62(5), 563–70.

Geisser, M., Hang, A. & Theisen, M. (2000). Activity avoidance and function in persons with chronic back pain. J Occup Rehabil, 10, 215–227.

Gerdle, B., Eriksson, N.E. & Brundin, L. (1990). The behaviour of the mean power frequency of the surface electromyogram in biceps brachii with increasing force and during fatigue. With special regard to the electrode distance. *Electromyogr Clin Neurophysiol*, 30, 483–489.

Gerdle, B., Henriksson-Larsen, K., Lorentzon, R. & Wretling, M.L. (1991). Dependence of the mean power frequency of the electromyogram on muscle force and fibre type. *Acta Physiol Scand*, 142, 457–465.

Gerdle, B., Karlsson, S., Crenshaw, A.G., Elert, J. & Friden, J. (2001). The influences of muscle fibre proportions and areas upon EMG during maximal dynamic knee extensions. *Eur J Appl Physiol*, 81, 2–10.

Giesbrecht, S., Jason, R., & Battie, M. C. (2005). A comparison of pressure pain detecton thresholds in people with chronic low back pain and volunteers without pain. Physical Therapy, 85 (10), 1085-1092.

Graven-Nielsen, T, Svensson, P. & Arendt-Nielsen, L, (1997). Effects of experimental muscle pain on muscle activity and co-ordination during static and dynamic motor function. *Electroencephalography and clinical neurophysiology*, 105(2), 156–64.

Graven-Nielsen, T., Lund, H., Arendt-Nielsen, L., Danneskiold, B. & Bliddal, H. (2002). Inhibition of maximal voluntary con- traction force by experimental muscle pain: a centrally mediated mechanism. *Muscle Nerve*, 26, 708–712.

Gribble, P.L. & Ostry D.J. (1998). Independent coactivation of shoulder and elbow muscles. *Exp Brain Res*, 123, 355–360.

Gruber, M., Gruber, S.B., Taube, W., Schubert, M., Beck, S.C. & Gollhofer, A. (2007), Differential effects of ballistic versus sensorimotor training on rate of force development and neural activation in humans. *Journal of strength and conditioning research*, 21 (1), 274-282.

Gulch, R.W. (1994) Force-velocity relations in human skeletal muscle. International Journal of Sports Medicine, 15 (Suppl. 1), S2-10.

Hahn, R.A. (1997). The nocebo phenomenon: concept, evidence and implications for public health. Prev. Med., 26, 607-611.

Hall, E. G., & Davies, S. (1991). Gender differences in perceived intensity and affect of pain between athletes and nonathletes. Perceptual and Motor Skills, 73, 779 - 786.

Hammond, M.C., Fitts S.S., Kraft G.H., Nutter P.B., Trotte M.J. & Robinson L.M. (1988). Co-contraction in the hemiparetic forearm: quantitative EMG evaluation. *Arch Phys Med Rehabil*, 69, 348–351.

Haour, F. (2005). Mechanisms of the placebo effect and of conditioning. Neuroimmunomodulation, 12, 195-200.

Hatayama, T. & Shimizu, K. (1993). An examination by forearm EMG on pain reaction time to radiant heat. *Perceptual and Motor Skills*, 76, 1139-1146.

Heckman, C. & Binder, M. (1990). Neural mechanisms underlying the orderly recruitment of motoneurons. In Binder, M. & Mendell, L. The segmental motor system, Neural mechanisms underlying the orderly recruitment of motoneurons. Oxford UP, New York, 182–204

Heil, E. (1993). *Psychology of sport injury*. Champaign, IL.: Human Kinetics.

Heil, J., & Fine, P. (1999). Pain in sport: A biopsychological perspective. In Pargman, D. Psychological Bases of Sport Injuries. Morgantown, WV: Fitness Information Technology.

Henneman, E., Somjen, G. & Carpenter, D.O. (1965). Functional Significance of Cell Size in Spinal Motoneurons. *Journal of neurophysiology*, 28, 560-580.

Hill, A.V. (1938). The heat of shortening and the dynamic constants of muscle. *Proceedings of the Royal Society of London*, B125, 136-195.

Hill A.V. (1953) The mechanics of active muscle. Proc Roy Soc Lond (Biol), 141, 104-117.

Hirsch, M.S. & Liebert, R.M. (1998). The physical and psychological experience of pain: the effects of labeling and cold pressor temperature on three pain measures in college women. Pain, 77, 41–48.

Hodges, P.W. & Richardson C.A. (1996). Inefficient muscular stabilisation of the lumbar spine associated with low back pain: a motor control evaluation of transversus abdominis. *Spine*, 21, 2640–50.

Hodges, P.W., Ervilha, U.F., Graven-Nielsen, T. (2008). Changes in Motor Unit Firing Rate in Synergist Muscles Cannot Explain the Maintenance of Force During Constant Force Painful Contractions. *Journal of Pain*, 9, 1169-1174.

Hodges, P. W., Moseley, G. L., Gabrielsson, A., & Gandevia, S. C. (2003). Experimental muscle pain changes feedforward postural responses of the trunk muscles. *Experimental Brain Research*, 151, 262-271.

Hodges, P.W. (2011). Pain and motor control: From the laboratory to rehabilitation. *Journal of electromyography and kinesiology: official journal of the International Society of Electrophysiological Kinesiology*, 21(2), 220–8.

Hodges, P.W. & Tucker, K. (2011). Moving differently in pain: a new theory to explain the adaptation to pain. *Pain*, 152(3 Suppl), S90–8.

Hopkins, W.G. (2005). Variability and Smallest Worthwhile Enhancements. *Sportscience*, 9, 17-20.

Hopman-Rock, M., Kraaimaat, F.W. & Bijlsma, J.W.J (1997). Quality of life in elderly subjects with pain in the hip or knee. Qual Life Res, 6, 67–76.

Horowits, R. & Podolsky, R. J. (1987a). Thick filament movement and isometric tension in activated skeletal muscle. Biophysical Journal, 54 (1), 165-171.

Horowits, R. & Podolsky, R. J. (1987b). The positional stability of thick filaments in activated skeletal muscle depends on sarcomere length: evidence for the role of titin filaments. J. Cell Biol, 105, 2217-2223.

Howatson, G., & van Someren, K. A. (2003). Ice massage: Effects on exercise-induced muscle damage. *Journal of Sports Medicine and Physical Fitness*, 43, 500 – 505.

Hudson, M., McCormick, L., Zalucki, K., Moseley, N. & Lorimer, G. (2006). Expectation of pain replicates the effect of pain in a hand laterality recognition task: bias in information processing toward the painful side? *European Journal of Pain*, 10 (3), 219-224.

Huxley, A.F. (1957). Muscle structure and theories of contraction. *Progress in biophysics and biophysical chemistry*, 7, 255-318.

Iso-Ahola, S. E., & Hatfield, B. (1986). *Psychology of sports: A social psychological approach.* Dubuque, IA: Brown.

Jackson, T. et al., 2005. Gender, interpersonal transactions, and the perception of pain: an experimental analysis. *The journal of pain: official journal of the American Pain Society*, 6(4), 228–36.

Janal, M. N., Glusman, M., Kuhl, J. P., & Clark, W. C. (1994). Are runners stoical? An examination of pain sensitivity in habitual runners and normal active controls. *Pain*, 58 (1), 109-116.

Jarmenko, M. E. (1978). Cognitive strategies in pain tolerance. *Journal of Behavior Therapy and Experimental Psychiatry*, 9, 239.

Jaremko, M.E., Silbert, L. & Mann, T. (1981). The differential ability of athletes and non-athletes to cope with two types of pain: A radical behavioural model. *The Psychological Record*, 31. 265-275.

Jing, Z. L., Robert, W. Brown, & Guang H. Y. (2002). A Dynamical Model of Muscle Activation, Fatigue, and Recovery. *Biophysical Journal Volume*, 82, 2344–2359.

Johnston, D. & Mannell, RC. (1980). The effects of experimentally created team norms on pain perception. In Klavora, P. & Wipper, K.A.W. *Psycho- logical and sociological factors in sport.* Toronto: University of Toronto Publications Division.

Jones, A., Spindler, H., Jorgensen, M.M. & Zachariae R. (2002). The effect of situation-evoked anxiety and gender on pain report using the cold pressor test. *Scand J Psychol*, 43, 307-313.

Jones, D.H.,& Kilgour, R.D. (2007). Test-Retest Reliability of Pressure Pain Threshold Measurements of the Upper Limb and Torso in Young Healthy Women. The Journal of Pain, 8 (8), 650-656.

Kaplanis P.A., Pattichis C.S., Hadjileontiadis L.J., Panas S.M. (2000). Bispectral analysis of surface EMG. Proceedings of the 10th Mediterranean Electrotechnical Conference, 2, 770-773.

Kelly, A. (2001). The minimum clinically significant difference in visual analogue scale pain score does not differ with severity of pain, 205–207.

Kniffki, K.D., Schomburg, E.D. & Steffens, H. (1979). Synaptic responses of lumbar alpha-motoneurones to chemical algesic stimulation of skeletal muscle in spinal cats. *Brain Res,* 160 (3), 549–52.

Kolt, G.S. & Mackler, L. (2003). *Physical Therapies in Sport and Exercise.* Elsevier Health Sciences.

Kooistra, R.D., de Ruiter, C.J. & de Haan, A. (2007). Conventionally assessed voluntary activation does not represent relative voluntary torque production. *European journal of applied physiology,* 100 (3), 309-320.

Kraus, J.F., & Conroy, C. (1984). Mortality and morbidity from injuries in sports and recreation. In Weinberg, R.S. & Gould, D. Foundations of Sport and Exercise Psychology (3rd ed.). Champaign, IL: Human Kinetics.

Kress, J. L. (1999). A naturalistic investigation of former Olympic cyclists' cognitive strategies for coping with exertion pain during performance. *Dissertation Abstracts* International (B), 59(9), 4763.

Lang, P. L. (1995). The emotion probe: Studies of motivation and attention. *American Psychologist,* 50, 372–385.

Leventhal. H. (1993). The pain system: A multilevel model for the study of motivation and emotion. *Motivation and Emotion*, 17, 139-146.

Leventhal, H. & Everhart, D. (1979). *Emotion, pain and physical illness.* In Izard, C.E. *Emotion and psychopathology.* New York: Plenum Press.

Liu, J.Z., Brown, R.W. & Yue, G.H. (2002). A Dynamical Model of Muscle Activation, Fatigue, and Recovery. Biophysical Journal, 82, 2344–2359.

Loeser, J. D., & Treede, R. D. (2008). The Kyoto protocol of IASP Basic Pain Terminology. *Pain*, 137, 473-477.

Logan, H., Lutgendorf, S., Rainville, P., Sheffield, D., Iverson, K., & Lubaroff, D. (2001). Effects of stress and relaxation on capsaicininduced pain. Journal of Pain, 2, 160–170.

Lorenz, J., & Bromm, B. (1997). Event related potential correlates of interference between cognitive performance and tonic experimental pain. *Psychophysiology*, 34, 436-445.

Lorenz, J., Beck, H., & Bromm, B. (1997b). Differential changes of laser evoked potentials, late auditory evoked potentials and P300 under morphine in chronic pain patients. *Electroencephalography and Clinical Neurophysiology*, 104, 514-521.

Low, C. (2005). A Comparison of Pressure Pain Detection Thresholds. 85(10), 1085–1093.

Lund, J. P., Donga, R., Widmer, C. G., & Stohler, C.S. (1991). The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. *Canadian Journal of Physiology and Pharmacology*, 69(5), 683–694.

Luscher, H.R., Ruenzel, P. & Henneman, E. How the size of motoneurones determines their susceptibility to discharge. *Nature*, 282, 859–61.

LeUnes, A. (2008). *Sport Psychology*. Psychology Press, New York.

MacDonald, D., Moseley, G.L. & Hodges, P.W. (2009). Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain, 142(3), 183–8.

MacIntosh, B.R., Gardiner, P.F. & McComas, A.J. (2006). Skeletal muscle: form and function (2nd ed.), Human Kinetics, Champaign, IL.

Madeleine, P. (2006). Experimental muscle pain changes the spatial distribution of upper trapezius muscle activity during sustained contraction. Clinical neurophysiology, 117(11), 2436–45.

Magnusson, M., Aleksiev, A., Wilder, D., Pope, M., Spratt, K. & Goel, V. (1996). Sudden load as an aetiologic factor in low back pain. Eur J Phys Med Rehabil, 6, 74–81.

Major, W. F. (1996). Serious Running: An interpretive analysis. International Institute for Sport and Human Performance, University of Oregon at Eugene: Microform Publications.

Marshall, H.R. (1894). Pain, pleasure and aesthetics. London: Macmillan.

Martin, S. (No date). Top End Sports: Qoutes about pain and effort [Online] Available at www.topendsports.com/psychology/quotes-pain.htm (24 June 2011).

Martini, F. H. (2004). Fundamentals of Anatomy and Physiology (6th Ed.). Pearson Education Inc. USA.

Masters, K.S. & Lambert, NJ. (1989). The relationship between cognitive coping strategy, reasons for running, injury and performance of marathon runners. Journal of Sports Exercise and Psychology. 11, 161-170.

Matre DA, Sinkjaer T, Svensson P, Arendt-Nielsen L. (1998). Experimental muscle pain increases the human stretch reflex. Pain, 75(2–3), 331–9.

McArdle, W.D., Katch, F.I. & Katch, V.L. (1996). Exercise physiology : energy, nutrition, and human performance (4th ed.). Baltimore: Williams & Wilkins.

McMaster, W.C. & Troup, 1. (1993). A survey of interfering shoulder pain in US competitive swimmers. American Journal of Sports Medicine. 21, 67-70.

Meichenbaum, D.H. (1977). Cognitive behaviour modification: An integrative approach. Plenum, New York.

Meijer, K., Bosch, P., Bobbert, M.F., van Soest, A.J. & Huijing, P.A. (1998). The isometric knee extension moment-angle relationship: experimental data and predictions based on cadaver data. J Appl Biomech, 14, 62–79.

Merletti, R., Farina, D. & Granata, A. (1999). Non-invasive assessment of motor unit properties with linear electrode arrays. Electroencephalogr Clin Neurophysiol, 50, 293–300.

Mellor. R. & Hodges. P.W. (2005). Motor unit syncronization is reduced in anterior knee pain. Journal of Pain 6: 550-558.

Melzack, R., & Wall, P. D. (1965). Pain mechanisms: A new theory. *Science*, 50, 971-979.

Melzack. R. & Togerson. W.S. (1971). On the language of pain. *Anaesthesiology*, 34.51-59.

Melzack, R. (1973). *The puzzle of pain*. Harmondsworth, London: Penguin Books.

Melzack, R. W. (1988). *The challenge of pain*. Harmondsworth, London.

Melzack. R. (1993). Pain: Past, present and future. *Canadian Journal of Experimental Psychology*, 47,615-629.

Melzack, R. (2001). Pain and the neuromatrix in the brain. *J Dental Education*, 65, 1378–1382.

Mendell, L.M. (2005). The size principle: a rule describing the recruitment of motoneurons. *Journal of Neurophysiology*, 93, 3024-3026.

Meyers, M.C., Bourgeois, A.E., Stewart, S. & LeUnes, A. (1992). Predicting pain response in athletes: Development and assessment of the sports inventory for pain. Journal of Sport and Exercise Psychology, 14, 249-261.

Mok, N., Brauer, S. & Hodges, P. (2004). Hip strategy for balance control in quiet standing is reduced in people with low back pain. Spine, 29, E107–12.

Müller, J. (1842). *Handbuch der Physiologie des Menschen für Vorlesungen*. In Baly, W. *Elements of Physiology*. London, Taylor & Walton.

Nathan, P. W. (1976). The gate-control theory of pain. A critical review. *Brain*, 99, 123-158.

Neddermeyer, T. J., Fluehr, K., & Loetsch, J. (2008). Principle components analysis of pain thresholds to thermal, electrical, and mechanical stimuli suggests a predominant common source of variance. *Pain*, 138, 286-291.

Nicholls, A.R. & Polman, R.C. (2007). Coping in sport: A systematic review. *Journal of Sport Sciences*, 25, 11-31.

Nideffer. R.M. (1978). *Attention control training*. New York: Wyden Books.

Nideffer, R.M. (1992). *Psyched to win*. Leisure Press, Champaign, IL.

Nigg B.M., & Herzog W. (1999). *Biomechanics of the Musculo-Skeletal system*. Wiley.

Nixon, H.L. (1992). A social network analysis of influences on athletes to playing with pain. *Journal of Sport and Social Issues.* 16, 127-135.

Nixon. H.L. (1993). Accepting the risks of pain and injury in sport: Mediated cultural influences on playing hurt. *Sociology of Sport Journal.* 10, 183-196.

Noakes, T. D. (2004). Central regulation of skeletal muscle recruitment explains the reduced maximal cardiac output during exercise in hypoxia. *Am J Physiol Integr Comp Physiology,* 287, 996–9.

Noakes, T. D., St Clair Gibson, A., & Lambert, E. V. (2005). From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *Br J SportsMed* 39, 120–124.

Noordenbos, W. (1959) *Pain.* Amsterdam: Elsevier.

Nouwen, A., Cloutier, C., Kappas, A., Warbrick, T., & Sheffield, D. (2006). The effects of focusing and distraction on cold-pressor induced pain. *Journal of Pain,* 7, 62–71.

Onder, G. et al. (2006). Association between daily pain and physical function among old-old adults living in the community: results from the ilSIRENTE study. *Pain,* 121(1-2), pp.53–9.

Orbach, R. & Gale, E.N. (1989). Pressure pain thresholds in normal muscles: reliability, measurement effects, and topographic differences. Pain, 37, 257–263.

Ord, P. & Gijsbers, K. (2003). Pain thresholds and tolerances of competitive rowers and their use of spontaneous, self-generated pain coping strategies. *Perceptual Motor Skills,* 97, 1219-1222.

Pacheco-López, G. (2006). Expectations and associations that heal: Immunomodulatory placebo effects and its neurobiology. *Brain, behavior, and immunity,* 20(5), 430–46.

Paton, C.D. & Hopkins, W.G. (2005). Competitive Performance of Elite Olympic-Distance Triathletes: Reliability and Smallest Worthwhile Enhancement.

Paton, C.D. & Hopkins, W.G., (2006). Variation in performance of elite cyclists from race to race. European Journal of Sport Science, 6(1), pp.25–31.

Pavlov, I. P. (1927). *Conditioned Reflexes: An Investigation of the Physiological Activity of the Cerebral Cortex.* Translated and Edited by G. V. Anrep. London: Oxford University Press.

Pearce, J.M.S. (2005). The Law of Specific Nerve Energies and Sensory Spots. *Eur Neurol,* 54, 115–117.

Peck, C.C., Murray, G.M., & Gerzina, T.M. (2008). How does pain affect jaw muscle activity? The Integrated Pain Adaptation Model. Aust Dent J, 53(3), 201-7.

Pen. L,J. & Fisher. C.A. (1994). Athletes and pain tolerance. *Sports Medicine,* 18, 319-329.

Pen. L,J., Fisher., A.C., Sforzo, O.A. & McManis. B.O. (1995). Cognitive strategies and pain tolerance. *Journal of Sport Rehabilitation.* 4.181-194.

Peri, E. R. (2011). Pain mechanisms: A commentary on concepts and issues. *Progress in Neurobiology,* 94 (1), 20–38.

Petrie, A. (1960). Some psychological aspects of pain and the relief of suffering. *Annals of the New York Academy of Sciences,* 86, 13.

Petrofsky, J.S. (2001). The blood pressure and heart rate response to isometric exercise—the effect of spinal cord injury . Eur J Appl Physiol, 85, 521–526.

Petrofsky, J.S. & Laymon, M. (2005). Muscle temperature and EMG amplitude and frequency during isometric exercise. Aviat Space Environ Med, 76, 1024–30.

Porro, C.A., Baraldi, P., Pagnoni, G., Serafini, M., Facchin, P., Maieron, M. & Nichelli, P. (2002). Does anticipation of pain affect cortical nociceptive systems? *J Neurosci,* 22 (8), 3206–3214.

Price, D.D. (2000). Psychological and neural mechanisms of the affective dimension of pain. *Science,* 288, 1769–1772.

Prokop, L. (2000). The significance of pain in sport. *Kinesiology,* 32(1), 77-84.

Rassier, D.E., MacIntosh, B.R. & Herzog, W. (1999). Length dependence of active force production in skeletal muscle. *Journal of applied physiology* 86, (5), 1445-1457.

Rheinecker, S.B. (1998). Using ice therapy to alleviate pain and edema. *J Am Acad Physician Assist,* 11(6), 73-81.

Rhudy, J. L., & Meagher, M. W. (2000). Fear and anxiety: Divergent effects on human pain thresholds. *Pain,* 84, 65–75.

Riek, S. & Bawa, P. (1992). Recruitment of motor units in human forearm extensors. *J Neurophysiol,* 68(1), 100–8.

Riley, J.L., Robinson, M.E., Wise, E.A., Myers, C.D. & Fillingim, R.B. (1998). Sex differences in the perception of noxious experimental stimuli: A meta-analysis. *Pain,* 74, 181-187.

Robertson, D.G.E., Hamill, J., Caldwell, G.E. & Kamen, G. (2004). Research Methods in Biomechanics. Champaign, IL: Human Kinetics Publishers.

Roland, M. (1986). A critical review of the evidence for a pain–spasm–pain cycle in spinal disorders. *Clin Biomech,* 1, 102–9.

Rose, J. E. & Mountcastle, V. B. (1959). *Touch and kinesthesis.* In Field, J., Magoun, H.W. & Hall, V. E. *Handbook of physiology.* Washington, DC: American Physiological Society; 1959:387–429.

Ross, H., Cleveland, S. & Haase, J. (1975). Contribution of single motoneurons to Renshaw cell activity. *Neurosci Lett,* 1, 105–108.

Ryan, E. D., & Kovacic, C. R. (1966). Pain tolerance and athletic participation. *Perceptual and Motor Skills,* 22, 383-390.

Ryan, E.D. & Foster, R. (1967). Athletic participation and perceptual augmentation and reduction. *Journal of personality and social psychology,* 6(4), 472–6.

Salomoni S, Graven-Nielsen T Experimental muscle pain increases normalized variability of multidirectional forces during isometric contractions. European Journal of Applied Physiology: 1-11.

Saunders, Philadelphia.Bonifazi, M., Ciccarone, G., della Volpe, R., Spidalieri, R. & Rossi, A. (2004). Influences of chemically-induced muscle pain on power output of ballistic upper limb movements. *Clinical Neurophysiology,* 115, 1779-1785.

Schulte, E., Ciubotariu, a, Arendt-Nielsen, L., Disselhorst-Klug, C., Rau, G., & Graven-Nielsen, T. (2004). Experimental muscle pain increases trapezius muscle activity during sustained isometric contractions of arm muscles. *Clinical neurophysiology*, 115(8), 1767–78.

Schultz, W. (1998). Predictive reward signal of dopamine neurons. *J. Neuro- physiol.* 80, 1–27.

Scott, V., & Gijsbers, K. (1981). Pain perception in competitive swimmers. *British Medical Journal*, 283, 91-93.

Sellwood, K.L. (2007). Ice-water immersion and delayed-onset muscle soreness: a randomised controlled trial. *British journal of sports medicine*, 41(6), 392–7.

Semmler, J.G. (1994). Compound group I excitatory input is differentially distributed to motoneurones of the human tibialis anterior. *Neuroscience Letters*, 178, 206-210.

Sessle, B.J. (1999). Neural mechanisms and pathways in craniofacial pain. *Can J Neurol Sci*, 26 (Suppl 3), S7–S11.

Siegel, S. (2002). *Explanatory mechanisms for placebo effects: Pavlovian conditioning.* In Guess, H.A., Kleinman, A., Kusek, J.W. & Engel, L.W. *The science of the placebo: toward an interdisciplinary research agenda.* London: BMJ Books, 133–57.

Sinclair, D.C. (1955). Cutaneous sensation and the doctrine of specific nerve energies. *Brain*, 78, 584–614.

Sohn, M.K. (2004). Effects of experimental muscle pain on mechanical properties of single motor units in human masseter. *Clinical Neurophysiology*, 115(1), 76–84.

Spink, K. S. (1988). Facilitating endurance performance: The effect of cognitive strategies and analgesic suggestions. *The Sport Psychologist*, 2, 97-104.

Staats, P. S., Staats, A., & Hekmat, H. (2001). The additive impact of anxiety and a placebo on pain. *Pain Medicine*, 2, 267–279.

Sternbach, R. A. (1978). *The psychology of pain*. Oxford: Oxford University Press.

Sternberg, W.F., Bailin, D., Grant, M. & Gracely, R.H. (1998). Competition alters the perception of noxious stimuli in male and female athletes. *Pain*, 76(1-2), 231–8.

Stewart-Williams, S. (2004). The placebo puzzle: putting together the pieces. *Health Psychol*, 23, 198–206.

Stewart-Williams, S. & Podd, J. (2004). The placebo effect: dissolving the expectancy versus conditioning debate. *Psychol Bull*, 130, 324–40.

Stohler, C.S., Ashton-Miller, J.A., Carlson, D.S. (1988). The effects of pain from the mandibular joint and muscles on masticatory motor behaviour in man. *Arch Oral Biol*, 33,175-182.

Straub, W. F., Martin, S. B., Williams, D. Z., & Ramsey, A. L. (2003). Pain apperception among athletes playing contact and non contact sports. *The Sport Journal*, 6(2).

Stuart, D.G. & Enoka, R.M. (1983). *Motoneurons, motor units, and the size principle*. In Rosenberg, R.N. *The clinical neurosciences*. New York: Churchill Livingstone; 471–517.

Suzuki, N. & Endo, S. (1983). A quantitative study of trunk muscle strength and fatigability in the low-back-pain syndrome. *Spine*, 8, 69–74.

Svensson, P., Arendt-Nielsen, L. & Houe, L. (1995). Sensory–motor interactions of human experimental unilateral jaw muscle pain: a quantitative analysis. *Pain*, 64, 241–9.

Szeto, G.P.Y., Straker, L.M. & O'Sullivan, P.B. (2005). A comparison of symp- tomatic and asymptomatic office workers performing monotonous keyboard work. *Man Ther*, 10, 270–280.

Taylor, J. & Taylor, S. (1998). Pain education and management in the rehabilitation from sports injury. *The Sport Psychologist*, 12, 68-88.

ter Haar Romeny, B.M., van der Gon, J.J.D. & Gielen, C.C.A.M. (1982). Changes in recruitment order of motor units in the human biceps muscle. *Experimental Neurology*, 78, 360-368.

Tesarz, J., Schuster, A.K., Hartmann, M., Gerhardt, A. & Eich, W. (2012). Pain perception in athletes compared to normally active controls: A systematic review with meta analysis. *Pain*, 153, 1253-1262.

Thomas, J.S., Schmidt, E.M. & Hambrecht, F.T. (1978). Facility of motor unit control during tasks defined directly in terms of unit behaviors. *Exp Neurol*, 59(3), 384–97.

Todd, B. (1987). The placebo effect. Real or imaginary? Geriatric Nursing, 8, 154–156.

Torebjork, H. E., Hallin, R. G., Hongell, A. & Hagbarth, K. E. (1970). Single unit potentials with complex waveform seen in microelectrode recordings from the human median nerve. Brain Res. 24, 443-450.

Torebjork, H. E. & Hallin, R. G. (1973). Perceptual changes accompanying controlled preferential blocking of A and C fibre responses in intact human nerves. *Experimental Brain Research*, 16, 321-332.

Torebjork, H. E. & Hallin, R. G. (1974). *Identification of afferent C units in intact human skin nerves*. Brain Research, Amsterdam, 67, 387-403.

Torebjork, H. E. & Ochoa, J. L. (1980). Specific sensations evoked by activity in single identified sensory units in man. Acta physiol. scand. 110, 445-447.

Tucker, R., Marle, T., Lambert, E. V., & Noakes, T. D. (2006). The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. *Journal of Physiology*, 574, 905–915.

Turker, K.S. (1993). Electromyography: some methodological problems and issues, *Physical Therapy*, 73 (10), 698-710.

Turner, J.A., Deyo, R.A, Loeser, J.D., Von Korff, M. & Fordyce, W.E. (1994). The importance of placebo effects in pain treatment and research. *Journal of the American Medical Association*, 271, 1609-1614.

Tursky, B., & O'Connell, D. (1972). Reliability and interjudgment predictability of subjective judgments of electrocutaneous stimulation. *Psychophysiology*, 9, 290-295.

UK Sport (2011). *Annual Report and accounts 2010/11*. UK Sport: London.

Van Damme, S., Crombez, G., Eccleston, C. & Goubert, L. (2004). Impaired disengagement from threatening cues of impending pain in a crossmodal cueing paradigm. *Eur J Pain*, 8, 227–36.

Van Damme, S., Crombez, G., & Eccleston, C. (2008). Coping with pain: A motivational perspective. *Pain*, 139, 1-4.

van Dieën JH, Selen LP, Cholewicki J. (2003). Trunk muscle activation in low-back pain patients, an analysis of the literature. *J Electromyogr Kinesiol*, 13(4), 333–51.

Van Raalte, J. L., & Brewer, B. W. (1996). *Exploring sport and exercise psychology*. Washington, DC: American Psychological Association.

Vase, L., Robinson, M.E., Verne, G.N. & Price, D.D. (2005). Increased placebo analgesia over time in irritable bowel syndrome (IBS) patients is associated with desire and expectation but not endogenous opioid mechanisms. *Pain*, 115, 338–47.

Veiersted, K., Westgaard, R. & Andersen, P. (1990). Pattern of muscle activity during stereotyped work and its relation to muscle pain. *International Archives of Occupational and Environmental Health*, 62, 31-41.

Villemure, C., & Bushnell, M. C. (2002). Cognitive modulation of pain: How do attention and emotion influence pain processing? *Pain*, 95, 195–199.

Voelker, R., (1996). Nocebos Contribute to Host of Ills. *The Journal of the American Medical Association*, 275(5), 345.

Wager, T.D. (2005). Expectations and anxiety as mediators of placebo effects in pain. *Pain*, 115, 225–6.

Wager, T.D., Rittling, J.K., Smith, E.E., Sokolit, A., Casey, K.L., Davidson, R.J., Kosslyn, S.M., Rose, R.M., Cohen, J.D. (2004). Placebo-induced changes in fMRI in the anticipation and experience of pain. *Science*, 303, 1162–1167.

Walker, I. (1971). Pain and distraction in athletes and non athletes. *Perceptual and Motor Skills*, 33, 1187-1190.

Wall, P.D. (1967). The laminar organization of dorsal horn and effects of descending impulses. *Journal of Physiology*, 188, 403-423.

Wall, P. D. (1978). The Gate Control Theory of Pain Mechanisms. *Brain*, 101 (1), 1-18.

Wall, P.D. (1995). Do nerve impulses penetrate terminal arborisations? A pre-presynaptic control mechanism. *Trends in Neuroscience*, 18, 99-103.

Wang, J.Y., Zhang, H.T., Chang, J.Y., Woodward, D.J., Baccalá, L.A. & Luo, F. (2008). Anticipation of pain enhances the nociceptive transmission and functional connectivity within pain network in rats. *Mol Pain*, 4, 1–34.

Watson, A., El-Deredy, W., Vogt, B. & Jones, A.K.P. (2006). Categories of placebo response in the absence of site-specific expectation of analgesia. *Pain*, 126, 115–22.

Weddell, G., Sinclair, D.C. & Feindel, W.H. (1948). An anatomical basis for alterations in quality of pain sensibility. *J Neurophysiol*, 11, 99–109.

Weich, K., & Tracey, I. (2009). The influence of negative emotions on pain: behavioral effects and neural mechanisms. *NeuroImage*, 47 (3), 987-94.

Whitaker, A. (2010). Instantanatomy.net. Accessed 26/02/10.

Wilk, K.E., Meister, K. & Andrews, J.R. (2002). Current concepts in the rehabilitation of the overhead throwing athlete. Am J Sports Med, 30, 136-151.

Williams, L.R.T. (1978). Transcendental meditation and mirror tracing. *Perceptual and Motor Skills*. 46. 371-378.

Wing, A.E., Jones, P.G.W., Campbell, J.A. & Kay, A.D. (2011a). *The severity of experimentally induced pain influences muscular performance during maximal voluntary isometric knee extensor contractions*. Presented at the European Congress of Sport Sciences. Liverpool, UK.

Wing, A.E., Jones, P.G.W., Campbell, J.A. & Kay, A.D. (2011b). *Expectation of pain intensity does not influence neuromuscular performance but does influence pain perception during a maximal isometric knee extension task*. Presented at the British Association of Sport and Exercise Sciences Annual Conference. Essex, UK.

Yerkes, R.M. & Dodson, J.D. (1908). The relation of strength of stimulus to rapidity of habit formation. *Journal of Comparative and Neurological Psychology*, 18, 459-482.

Young, A. (1993). Current issues in arthrogenous inhibition. *Annals of the rheumatic diseases*, 52(11), 829–34.

Zedka, M., Prochazka, A., Knight, B., Gillard, D. & Gauthier, M. (1999). Voluntary and reflex control of human back muscles during induced pain. *J Physiol*, 520 (2), 591–604.

Zinsser, N., Bunker, L., & Williams, J. M. (2001). *Cognitive techniques for building confidence and enhancing performance.* In Williams J. M. (Ed.), *Applied sport psychology: Personal growth to peak performance* (4th ed., pp. 284–311). Mountain View, CA: Mayfield.

APPENDICES

Appendix 1:
Informed Consent Form

Consent Form
For Participating in the Study of:
Pain and Exercise: Is there a relationship?

(Details of project can be found in attached letter and information sheet)

Please tick the boxes

	Yes	***No***
I have read the study information sheet and understand what is involved.	☐	☐
I understand that the information I disclose will remain confidential and that my data will be destroyed or returned to me after being collated.	☐	☐
I understand that I can withdraw my participation at any time.	☐	☐
I am willing to participate in the Pilot Studies*	☐	☐
Study 1 *	☐	☐
Study 2 *	☐	☐
Study 3 *	☐	☐
I confirm that I do not have and never have had any bone diseases or injuries to my lower leg that may affect my participation within this study	☐	☐
I confirm that I do not have and never have had any respiratory diseases or circulatory diseases that may affect my participation within this study	☐	☐

If you have been affected by any of the conditions above please give details in the space provided below (including details of which leg was affected with regard to leg injuries/bone disease)

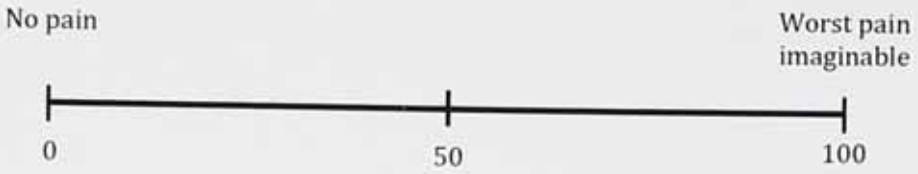
_____

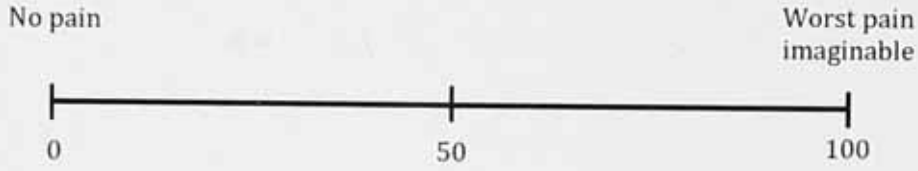
_____

Signed: ..... Date: .....

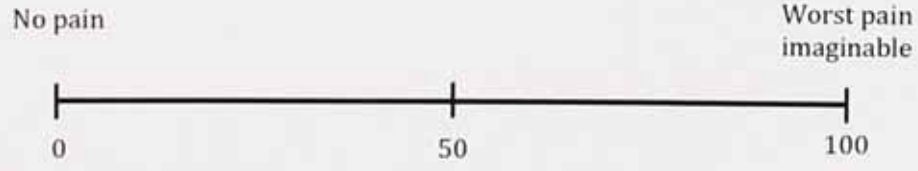
* If you agree to take part in any of the above studies you are still able to withdraw at any time point.

Appendix 2:
Visual Analogue Scale


No pain
Worst pain imaginable
0
50
100


No pain
Worst pain imaginable
0
50
100


No pain
Worst pain imaginable
0
50
100


No pain
Worst pain imaginable
0
50
100

Appendix 3:
Participant Recruitment Poster

Are you interested in being a participant in a study involving pain and exercise?

Are you aged 18 or above with no previous injuries to your lower legs?

I am a Postgraduate research student at the University of Northampton and am completing a series of studies looking into the link between pain and exercise. I am looking for healthy male volunteers to take part in my studies.

If you are aged 18+ and would be interested in taking part then please contact me for further information.

Annika Wing

University of Northampton
Park Campus
Boughton Green Road
Northampton
NN2 7AL

Email: annika.wing2@northampton.ac.uk
Tel: 01604 892934

Appendix 4:
Participant Recruitment Email

My name is Annika Wing and I am a research student at the University of Northampton in the School of Health. I am looking for healthy male volunteers to take part in my study investigating the effects of pain when exercising.

Are you interested in being a participant in a study involving pain and exercise? Are you aged 18 or above with no previous injuries to your lower legs? If so then please contact me for more information about what would be involved.

I can be contacted at:

University of Northampton
Park Campus
Boughton Green Road
Northampton
NN2 7AL

Email: annika.wing2@northampton.ac.uk
Tel: 01604 892934

Thank you for taking the time to read this

Regards,

Annika Wing

Appendix 5:
Participant Information Sheet

PARTICIPANT INFORMATION SHEET

About The Researcher:

I am currently undertaking a PhD in the School of Health at the University of Northampton. As part of my research I am carrying out a number of studies relevant to my thesis in the study of pain tolerance within sport and exercise. I am being supervised in this project by Dr. Peter Jones of Moulton College and Professor Jackie Campbell from the University of Northampton.

Study Title:

Pain and Exercise: Is there a relationship?

Aim of Study:

This study will look at:

- How people feel pain and how this relates to sport
- How we can measure pain accurately for the purposes of sports research

What the study involves:

In this initial study I hope to undertake tests on a group of individuals to help validate the use of the equipment that is needed for this research project. If you were to volunteer to be a participant you would be asked to come in to the University of Northampton to take part in a trial which will last no more than an hour. This trial will involve testing your pain perception threshold (the point at which a stimulus is recognised as pain rather than simply a sensation) through pressure being applied to your tibia (shin bone).

Taking part in the study will involve doing three isometric knee contractions (this means it is a static contraction rather than your joint actually moving) whilst the amount of electrical activity in your leg is measured to show what percentage of your muscle fibres are being used. When pain is being tested a "Gross Pressure Device" will be used which is essentially a shin pad with a block of wood attached that gets pressed onto your shin bone using a blood pressure cuff. This does not cause any long term damage and as the participant you would have a release valve so that the pressure could be released by you at whatever point you wanted. As soon as the pressure is released the pain stops, there is no lasting effect.

There are a number of studies planned in this project. The studies will take place at the University in the biomechanics lab. Future studies would involve participants (perhaps not you) being required to attend more than once for different trials. In these cases it would be necessary for you to come at the same time of day on the same day of the week each time as it has been shown that time of day can affect your ability to tolerate pain.

The information required:

You will be asked to supply information regarding your age, gender and ethnicity for research purposes. However all information will remain anonymous and will not be shared with any parties not directly involved in the research project.

As this study is looking to determine tests of pain it is necessary to induce pain. However the type of pain being induced is very temporary in nature and will not result in any long term harm. As the research is trying to establish pain perception threshold rather than tolerance, this means that the level of pain will be minimal and will be dependent upon the participant. The participant will control when they start to feel uncomfortable and the test will then stop immediately by the participant. As the participant you will also be given a quick release valve so that you yourself can stop the trial at any point by releasing the pressure.

Can anyone take part?

As this study involves pain, there are certain restrictions on who can take part. The Gross Pressure Device that will be used puts pressure on to the shin bone. Therefore anyone that has had any injuries to their lower leg is unable to take part. This may be something like a broken bone or any large cuts. The Gross Pressure Device also uses air pressure to push the wooden tablet on to the bone. This is done by inflating a blood pressure cuff which surrounds the leg, thereby restricting the blood flow around the lower leg. Whilst the restriction is likely to be minimal and very temporary in nature, it is still important that anyone taking part in the study does not have any health issues that may make this problematic. For this reason anyone that has had, or does have, any circulatory problems or respiratory problems should not take part in this study. This may include lung disease, heart disease or problems with blood pressure.

What will happen to the information?

All information gathered will be stored in a locked cabinet and only researchers involved in the project will have access to it. Participants will be referred to by number so that names remain anonymous; this means that no one will be able to see your results. There will be a list of names against numbers stored separately from the rest of the data to identify participants; however this will only be accessed by the researcher and will be kept in a locked cabinet. Any data stored electronically will be password protected and encrypted so that no one can obtain access to it other than the researcher. On completion of the research project any personal information will be destroyed with the exception of data required for published articles which will be kept for a further three years post publication and then destroyed.

Not sure about participating?

If you are not willing to participate that is fine. There is no obligation to complete the trial even if you do agree to participate, so it is not a problem if you change your mind. You will be free to withdraw at any point, no matter how far into the process you are. Whether you participate or not will not affect your studies in any other way.

Your valued input:

There is no gain for you as an individual participant in this study. Your participation would be on a purely voluntary basis, however it would be helping towards a better understanding of pain within sport and all publications emerging from this project would be made available to you.

Contact the Researcher:

I hope that the above information is useful to you and gives you an understanding of what I am studying. If you are interested in the study and would like some more detailed information about the study or what is involved in participating then please do not hesitate to contact me. I will be happy to answer any questions you may have.

I can be contacted at:

University of Northampton
Park Campus
Boughton Green Road
Northampton
NN2 7AL

Email: annika.wing2@northampton.ac.uk
Tel: 01604 892934

Thank You

Thank you for your interest and support. If you would like to participate in the research please complete and return the consent form in the stamped addressed envelope provided.