A COMPARISON OF SURFACE EMG TEMPORAL AND SPECTRAL PARAMETERS FROM THE VASTUS MEDIALIS OF SUBJECTS WITH AND WITHOUT KNEE JOINT OSTEOARTHRITIS DURING A SUSTAINED, FATIGUING SUBMAXIMAL ISOMETRIC CONTRACTION.

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CERTIFICATE OF AUTHORSHIP

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made in the acknowledgements.

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ABSTRACT

Knee joint osteoarthritis is recognised as a significant subset of osteoarthritis. Little work has examined muscle changes that occur with knee joint osteoarthritis. Much of this work has centred on strength deficits, while little work has examined the effect of joint pathologies, such as osteoarthritis, on the fatigue resistance of the muscles associated with an affected joint.

The purpose of this study was to investigate the relative fatigue-resistance characteristics of the vastus medialis in subjects with and without knee joint osteoarthritis, as well as the ability to predict endurance times in these groups, using high spatial resolution electromyography and a sub-maximal isometric endurance test. Twenty-six subjects with unilateral knee osteoarthritis, and seventeen subjects with no known knee pathology were evaluated. All subjects performed initial tests to evaluate maximum voluntary contraction (MVC), voluntary activation levels, and true maximum force (TMF). Endurance time was assessed during an isometric quadriceps contraction at 50% of the true maximum force. Surface electromyography (sEMG) data was collected from the vastus medialis muscle of the quadriceps group during the endurance test.

MVC tests showed that the affected leg of the group with osteoarthritis was significantly weaker ($p < 0.05$) than the unaffected leg. Voluntary activation data showed that subjects with osteoarthritis presented with significant bilateral deficits ($p < 0.05$). TMF data showed a significantly lower ($p < 0.05$) true potential for force generation in the affected compared to the unaffected leg of the osteoarthritis group. Endurance time data showed no significant difference between groups. Electromyography data showed significant differences ($p < 0.05$) between the affected and unaffected legs in initial values of Median Frequency (MDF), Mean Power Frequency (MPF) and Conduction Velocity (CV), the percentage change in CV and the relative rate of change in the frequency band between 5 and 30Hz (FB1). Finally, significant correlations were seen between endurance time and the relative rate of change of MDF, MPF and CV calculated over the initial thirty seconds of the endurance test. There were no significant correlations from either leg of the group with knee joint osteoarthritis.

It can be concluded from this study that there are differences in strength measures, and in the sEMG signal collected from the vastus medialis muscle of the affected and unaffected legs of subjects with knee osteoarthritis. It appears likely that the differences observed in the sEMG signals were related to a decrease in the representation of type-2 muscle fibres in the vastus medialis of the affected leg. Furthermore, these changes in the behaviour of the signal appear to indicate an improvement in the relative fatigue resistance of the affected leg in relation to the unaffected leg of the group with knee osteoarthritis.

Moderate success was seen with the prediction of endurance time in control subjects in the current work using a short duration (30-second) endurance test. This relationship was not seen in either the affected or unaffected leg of the subjects with knee osteoarthritis. Further investigation utilising different sEMG collection and analysis techniques in this area may improve prediction of endurance time in unaffected and affected subjects.

1. STATEMENT OF THE PROBLEM

1.1 INTRODUCTION

This study investigates the fatigue characteristics in the vastus medialis quadriceps muscle of subjects with osteoarthritis of the knee joint as measured using high spatial resolution (HSR) laplacian surface electromyography (sEMG).

The term *arthritis* can be translated literally into joint inflammation. Arthritis as a pathology covers a multitude of conditions involving inflammation and degeneration of joints. Of these, osteoarthritis is the most common form (Felson et al., 2000; Hurley, 1999; March & Bachmeier, 1997). Osteoarthritis (OA) is one of a group of musculoskeletal conditions that have been identified as a leading cause of morbidity and disability internationally, costing billions of dollars in health-care expenditure and loss of earnings. In the US alone an estimation of the total costs amounts to more than US$250 billion per year (Rice, 2000). As it is a disease that increases in prevalence with age, the burden of osteoarthritis on health resources in countries such as New Zealand will undoubtedly increase due to the aging population.

Osteoarthritis has been described as a complex disease with biochemical and biomechanical components (Felson et al., 2000; Hurley, 1999; Pinals, 1996). The biochemical component of osteoarthritis includes such factors as genetics, dietary intake, and oestrogen levels, while biomechanical components of interest include muscle weakness, muscle fatigue and joint loading. A combination of all or some of these factors can lead to an arthritic joint that can be defined by its symptoms or its pathology (Felson et al., 2000; Hurley, 1999). Symptomatically, patients with osteoarthritis commonly present with pain, swelling, increased joint laxity, decreased strength, or increased fatigability, or a combination of some or all of these symptoms (Hurley, 1999; Pinals, 1996; Sharma et al., 1999). Pathologically, osteoarthritis is characterised by focal and progressive loss of hyaline cartilage (Calvo et al., 2001; Kobayashi, Saito, Horiuchi, Iorio, & Takaoka, 2000). Damage also occurs to the bone beneath the cartilage, manifesting as bony thickening or sclerosis, and the development of bony outgrowths or osteophytes (Hill et al., 2003). Diagnosis of osteoarthritis is typically made using clinical guidelines based around patient signs

and symptoms, supported by x-rays showing evidence of bony changes (Altman et al., 1986). At the stage where a person is symptomatic and presents with changes on x-ray, it is acknowledged that they are significantly advanced along the pathway of osteoarthritis.

As well as the bony components of a joint, changes in soft tissue structures such as the capsule, the synovium, and the muscles bridging the joint are associated with osteoarthritis (Herzog, Longino, & Clark, 2003). While much research has been conducted into the bony changes associated with osteoarthritis, the changes in, and the role of, muscles associated with osteoarthritis in joints such as the knee have only recently gained more attention.

Of the joints affected by osteoarthritis, the knee is the most common (Hurley, Scott, Rees, & Newham, 1997; March & Bachmeier, 1997). As a subset of all osteoarthritis, knee osteoarthritis is recognised as causing the most significant disability, and having the greatest economic impact due to the amount of productive time lost as the result of that disability (Felson et al., 2000; Yelin, Such, Criswell, & Epstein, 1998). During daily life, the knee plays an important role, with many functional, work and sporting tasks requiring movement of the knee joint. Furthermore, adequate function of the knee relies on the effective contraction of the quadriceps muscle group. It is common knowledge that, in association with knee pathology such as osteoarthritis, the quadriceps muscles develop deficits. These deficits affect the ability of the knee to perform optimally.

A general decrease in strength is the most commonly acknowledged muscle deficit of those associated with knee osteoarthritis (Fisher & Pendergast, 1997; Hurley, 1997; Hurley & Newham, 1993; Nordesjo, Nordgren, Wigren, & Kolstad, 1983). A recent study by Pap and co-workers (2004), for example, examined two groups in one study; 68 patients with early osteoarthritis, and 154 patients with severe osteoarthritis. Both groups with osteoarthritis showed strength deficits in the region of 40% when compared to a control group (N = 85).

A less commonly examined change associated with knee osteoarthritis is an alteration in the fatigue resistance or endurance characteristics of the quadriceps. While the ability to perform a single maximal contraction is important, the successful

performance of functional activities requires individuals to sustain muscle activity over longer periods. The importance of fatigue resistance of the quadriceps in the function of the knee has been indicated by studies examining knees without pathology.

Fatigue in general has long been associated with a decrease in a healthy muscle's ability to produce force (Bigland-Ritchie, Johansson, Lippold, & Woods, 1983; Gandevia, Allen, Butler, & Taylor, 1996; Loscher, Cresswell, & Thorstensson, 1996a, 1996b). This relationship between increasing fatigue and a decline in force-generating capacity has been demonstrated specifically in the quadriceps by a number of authors (Kent-Braun, 1999; Mannion & Dolan, 1996).

Fatigue has also been shown to affect joint proprioception in healthy subjects. Skinner, Wyatt, Hodgdon, Conard & Barrack (1986), for example demonstrated decreased position matching of the knee ($p < 0.05$) following a quadriceps fatiguing protocol in eleven healthy subjects. Lattanzio, Petrella, Sproule and Fowler (1997) also observed increases in errors in lower limb position matching tasks of healthy subjects following a fatiguing protocol on a cycle ergometer.

Aside from influencing knee function, these fatigue-induced decreases in strength and proprioception are also thought to contribute to a reduction in the shock absorption ability of the quadriceps (Herzog, Clark, & Wu, 2003; Herzog, Longino et al., 2003; Hurley, 1999). The consequences of fatigue in this situation manifest as an increase in the loading on the joint (Jefferson, Collins, Whittle, Radin, & O'Connor, 1990). Such increases in joint loading are believed to play a role in the development of conditions characterised by joint arthrosis (Hurley, 1999; Slemenda et al., 1997).

Muscle fatigue itself can be measured or appreciated in a number of ways. Typically, in a clinical setting, this involves mechanical tests that require a muscle contraction held to exhaustion, and where the time to exhaustion is measured. Other laboratory methods may also be used, such as Magnetic Resonance Spectroscopy/Imaging (MRI), muscle biopsies, and blood sampling. Over the last three decades, significant work has been conducted utilising surface electromyography (sEMG) techniques to measure objectively and non-invasively the peripheral components of muscle fatigue. Using various sEMG techniques, a number of studies have demonstrated differing

fatigue characteristics in muscles associated with both healthy and unhealthy joints (McHugh, Tyler, Nicholas, Browne, & Gleim, 2001; McNair & Wood, 1993; Roy, De Luca, & Casavant, 1989; Roy, De Luca, Emley, & Buijs, 1995). Other studies have also investigated the ability to predict the point of muscle fatigue in healthy subjects using surface electromyography data (Maisetti, Guevel, Legros, & Hogrel, 2002a; Mannion & Dolan, 1996; Merletti & Roy, 1996). To date, no work appears to have been conducted in these areas involving the quadriceps and knee osteoarthritis.

1.2 PURPOSE STATEMENT

The purpose of this study was to:

1. *Investigate differences in the relative fatigue resistance of the vastus medialis quadriceps muscle of subjects with and without knee joint osteoarthritis. Endurance characteristics were investigated during a sub-maximal (50%) isometric contraction with a novel high-spatial resolution surface electromyography (HSR-EMG) technique. The variables of interest were: conduction velocity; median frequency; mean power frequency; root mean square; and the power within the frequency band 5-30Hz of the power spectrum.*
2. *Investigate the prediction of endurance time in subjects with knee joint osteoarthritis using changes in the sEMG signal collected during a short duration sub-maximal isometric contraction.*

1.3 SIGNIFICANCE OF THE STUDY

Despite the apparent importance of muscle fatigue in relation to quadriceps function, minimal work has examined fatigue resistance changes in the quadriceps associated with knee osteoarthritis. An appreciation of these changes may provide benefits to the knee osteoarthritis population on several levels.

Current rehabilitation programs, for example, place a significant emphasis on strength training, with minimal attention paid to specifically developing endurance. A better

understanding of the changes in the fatigue resistance characteristics of quadriceps in patients with knee osteoarthritis may allow better management of this condition through specific targeting of any endurance deficits noted. This may in turn reduce the effect of changes in strength, proprioception and knee-joint loading that occur with quadriceps muscle fatigue.

Performance of endurance tests in general requires maximal effort. By nature they are exhausting, and thus they may be affected by factors such as subject motivation and pain. Therefore, investigating the ability to predict muscle fatigue in the thigh muscles of subjects with knee osteoarthritis without subjecting them to an exhaustive test is evidently desirable.

1.4 LIMITATIONS

The methodology used had the following limitations:

1. When used for fatigue analysis, the surface electromyography technique used in the current study requires a constant force isometric contraction. This limits the validity of applying findings from the current study to activities that require dynamic muscle contractions.
2. Quadriceps were tested at 90° of knee flexion only.

1.5 DELIMITATIONS

The following restrictions were placed on the scope of this study:

1. Electromyographic data was collected from one quadriceps muscle (the vastus medialis) only.
2. Only the dominant leg of the control subjects was tested.

2. LITERATURE REVIEW

2.1 INTRODUCTION

This chapter begins with an overview of muscle fatigue. This includes its definition, the contribution of central and peripheral factors to fatigue, and local muscle factors that relate to fatigue resistance. Next, the fatigue resistance of healthy quadriceps is examined in the context of contraction types used, including discussion of the role of central and peripheral factors in each contraction type. Then the literature examining fatigue resistance in muscles affected by immobilisation or disuse is reviewed. This is followed by a review of muscle changes that occur as the result of disuse or immobilisation, and discussion as to how these changes may influence the fatigue resistance characteristics of a muscle. A review of surface electromyography and its use in the measurement of peripheral muscle fatigue follows, including peripheral, central and methodological factors that influence the sEMG signal. This is followed by a summary of sEMG collection techniques, and the use of surface electromyography in the prediction of endurance time in limb muscles. The chapter concludes with a review of the reliability of the measures extracted from sEMG signals in limb muscles.

2.2 MUSCLE FATIGUE

Muscle fatigue is a phenomenon that affects all individuals on a daily basis. It has been defined in a number of ways within the literature. Many definitions have referred to a failure of a muscle to reach or maintain a given force (Degens & Veerkamp, 1994; Edwards, 1981; Enoka & Stuart, 1992; Fitts, 1996). Other authors have also included work or power, so as to include decreases in dynamic performance under the definition of muscle fatigue (Asmussen, 1979; Dugan & Frontera, 2000). It is also commonly recognised that muscle fatigue is a process, rather than a distinct event, that begins almost as soon as a muscle begins contracting (Mannion & Dolan, 1996). For the purposes of the current work, fatigue is thought of as a process resulting in the reduction in the ability of a muscle to produce force and sustain a given force over time.

As well as there being a number of definitions of what characterises muscle fatigue, resistance to fatigue can be appreciated in a number of ways. These include an appreciation of fatigue from an absolute or a relative sense. *Absolute* fatigue resistance can be appreciated in a situation where the same load is applied to two muscles, or to the same muscle before and after immobilisation. Given two muscles with different maximum strength values, the absolute load applied to each will represent different percentages of their respective maximums, and thus different intensities of contraction will be required for the task. It may be expected that the weaker muscle will fatigue more quickly because the applied load represents a greater percentage of its maximum force output. This is consistent with the work of Hagberg (1981) who described an intuitive inverse relationship between contraction intensity and endurance. Expressed simply, the harder a muscle works, the more quickly it will fatigue. In most instances, absolute fatigue resistance is related to muscle strength, and with the strength losses reported for the quadriceps of disused/pathological knees, a decrease in absolute fatigue resistance is expected.

Relative fatigue resistance is a less intuitive concept. It can be appreciated when testing muscles at the same relative contraction intensity, represented by the same percentage of their maximum force output (50% MVC, for example). As the muscles in this situation are working at the same contraction intensity, their fatigue resistance conceivably relies on the performance of the various physiological components that contribute to the endurance characteristics of that muscle.

2.2.1 Central fatigue

Regardless of its appreciation in an absolute or relative sense, the process that leads to a decrease in muscle performance is the manifestation of both central and peripheral mechanisms associated with muscle fatigue. The central components of fatigue occur proximal to the neuromuscular junction, and affect the voluntary activation of a muscle. These components include decreases in cortical drive, changes in motor unit firing rates (Enoka & Stuart, 1992), peripherally mediated decreases in excitability of alpha-motoneurons (Davis & Bailey, 1997; Lepers, Maffiuletti, Rochette, Brugniaux, & Millet, 2002; Millet, Martin, Lattier, & Ballay, 2003), along with possible depletion of neurotransmitters at central synapses (Taylor, Butler, & Gandevia, 2000). Changes in voluntary activation resulting from central fatigue have been observed following both short (Gandevia et al., 1996; Kent-Braun, 1999) and long duration exercise

(Davis & Bailey, 1997). Gandevia and co-workers, for example, examined the effect of central fatigue on force generation after short duration exercise. Studying the biceps of eight healthy subjects they found a decreased ability to activate the tested muscle fully as subjects fatigued. They attributed this finding to a reduction in the excitability of the alpha-motoneurons in the spinal cord either due to sensory input from the fatiguing muscle, or due to descending modulation from the cortex. Millet et al. (2002) also found a decrease in subjects' ability to activate muscles due to fatigue, this time in quadriceps in 12 healthy subjects following a 30-kilometre run; voluntary activation decreased from 98.8% SD±1.8, to 91.3% SD±10.7. In addition to the decrease in activation, the authors also reported that the central aspect was responsible only for a proportion of the total fatigue seen. They attributed the remaining portion of fatigue in their subjects to peripheral factors.

2.2.2 Peripheral fatigue

Mechanisms of peripheral fatigue are those that occur within the muscle itself, distal to the neuromuscular junction (Gandevia, 2001; Millet & Lepers, 2004). It is believed that peripheral factors are largely responsible for the fatigue seen after short-duration activity (Millet & Lepers, 2004). These factors include alterations in concentrations of metabolites and electrolytes in and around muscle cells (Cady, Jones, Lynn, & Newham, 1989; Lunde, Verburg, Vollestad, & Sejersted, 1998), changes in the release, binding and reabsorption of calcium ions (Allen, Lannergren, & Westerblad, 1995; Fitts, 1996; Lannergren & Westerblad, 1991), and changes in the availability of energy sources (Roberts & Smith, 1989). The link between peripheral biochemical changes and the onset of muscle fatigue has been supported by a number of studies using nuclear magnetic resonance (P NMR) spectroscopy (Laurent, Portero, Goubel, & Rossi, 1993; Miller, Boska, Moussavi, Carson, & Weiner, 1988; Vestergaard-Poulsen et al., 1995). Miller et al. (1988), for example, investigated the relationship between five physiological markers (adenosine triphosphate (ATP), phosphocreatine (PCr), inorganic phosphate (Pi), monobasic phosphate ($H_2PO_4^-$), and hydrogen ions (H^+)) and the onset of fatigue in the adductor pollicis muscle of healthy subjects. Increases in concentrations of H^+ and H_2PO4^- were found to have inverse linear relationships ($r = -0.64$-0.77, $r = -0.7$-0.73 respectively) with a decline in force during a maximum voluntary contraction. Subsequent P NMR spectroscopy studies have also shown similar relationships between the development of fatigue and an increase in

concentrations of H^+ and $H_2PO_4^-$ in healthy human calf muscles (Laurent et al., 1993; Vestergaard-Poulsen et al., 1995). A note of caution is added in each of these studies, emphasising that the relationships between these biochemical markers and the development of fatigue were only associative, and that it was not established if they were in fact causative.

2.2.3 Local muscle factors influencing fatigue resistance

Resistance to peripheral muscle fatigue is related to the oxidative capacity of muscles (Burke, Levine, Tsairis, & Zajac, 1973). The oxidative capacity of individual muscles themselves has been related to a number of factors. A key factor is known to be a muscle's fibre composition (Gerdle, Johansson, & Lorentzon, 1988; Hulten, Thorstensson, Sjodin, & Karlsson, 1975; Komi & Tesch, 1979; Linssen et al., 1991; Thorstensson & Karlsson, 1976). While there is a number of known subtypes, muscles contain two main fibre-types: type-1, slow-twitch oxidative fibres, and type-2 fast-twitch glycolytic fibres. As their nomenclature suggests, these fibre-types differ in their contractile, morphological and metabolic properties. Type-1 muscle fibres, for example, develop tension more slowly, and are characterised by a greater density of capillaries (Booth & Thomason, 1991; Degens & Veerkamp, 1994; Hawley & Stepto, 2001; Saltin, 1977), greater numbers of mitochondria and higher concentrations of aerobic enzymes (Degens & Veerkamp, 1994; Gollnick & Saltin, 1982; Hawley & Stepto, 2001). These characteristics give these fibres specific efficiency in aerobic metabolism, and thus they are associated with longer duration, low intensity activity. The multiple subtypes of type-2 muscle fibres, in contrast, develop tension more rapidly, and have more highly developed anaerobic metabolic pathways, making them more suited to shorter duration, higher intensity activity. In relation to the differing characteristics and proportions of these fibres, it is accepted that the better the oxidative capacity of a muscle, the better the delivery of oxygen and fuel sources to the muscle during exercise (Karlsson, Nordesjo, Jorfeldt, & Saltin, 1972; MacDougall, Ward, & Sutton, 1977), and additionally the maintenance of muscle membrane integrity. These are all factors that have been associated with improved fatigue resistance.

Consistent with the properties of different fibre types, the relative proportions of fibre types have been related to a muscle's fatigue characteristics. Initial animal

experiments, for example reported that muscles composed of higher proportions of type-1 muscle fibres are more fatigue-resistant than muscles with more type-2 fibres (Baldwin & Tipton, 1972; Edstrom & Kugelberg, 1968; Kugelberg & Edstrom, 1968). Early human studies involving the quadriceps have reflected these results, reporting that subjects with a higher proportion of fast twitch fibres in their vastus lateralis showed greater rates of general fatigability (Hulten et al., 1975; Komi & Tesch, 1979; Nilsson, Tesch, & Thorstensson, 1977; Thorstensson & Karlsson, 1976). Later studies have supported these early findings. Sadoyama, Masuda, Miyata & Katsuta (1988), for example examined fibre composition of the vastus lateralis in sprint and endurance athletes. They reported that higher proportions of type-2 fibres were associated with the sprinters, and by association, higher rates of fatigue ($p<0.001$). Lorentzon, Johansson, Sjostrom, Fagerlund & Fugl-Meyer (1988) observed a similar relationship across a group of ten athletes containing marathon runners and sprinters, while Hawley and Stepto (2001) reported significantly greater levels of type-1 fibres in the vastus lateralis of elite compared to amateur endurance cyclists.

2.3 FATIGUE RESISTANCE IN HEALTHY QUADRICEPS

In examining muscle fatigue, a number of techniques and measures have been utilised in studies of both trunk and limb muscles. With regard to healthy quadriceps, these techniques and measures have been applied to a variety of mechanical tests involving isometric and isokinetic muscle contraction protocols. These protocols can generally be divided into those that have used sustained or intermittent sub-maximal isometric contractions held to failure, or maximal isometric contractions held for pre-determined times. In addition to the general information gained from such protocols, other studies have also examined the contributions of central and peripheral factors to the fatigue seen during sub-maximal and maximal isometric and dynamic contractions.

2.3.1 Sub-maximal isometric protocols

Much work has looked at the behaviour of healthy quadriceps during a sub-maximal isometric fatiguing contraction. Early investigations into isometric contractions (Hagberg, 1981) have demonstrated that the endurance time of an isometric contraction was closely related to the intensity of muscle contraction, expressed as a

percentage of maximum voluntary contraction (MVC). While Hagberg focused on the elbow flexors, later studies have shown that the same relationship is observed in the quadriceps (Arendt-Nielsen & Mills, 1988; Badier, Guillot, Lagier-Tessonnier, Burnet, & Jammes, 1993; Ebenbichler et al., 1998; Mannion & Dolan, 1996; Nagle, Seals, & Hanson, 1988). A number of studies have specifically reported endurance times for healthy quadriceps at various levels of isometric contraction. Hakkinen and Komi (1983), for example observed endurance times for isometric quadriceps contractions at 50% MVC of 89±27 seconds. Arendt-Neilson and Mills (1988) found that isometric contraction of quadriceps at 60% MVC could be maintained for approximately 52 seconds in five healthy subjects at 45° knee flexion. Grabiner, Koh and Miller (1991) found that nine healthy male subjects could maintain an isometric quadriceps contraction for 130 seconds at 30%, and 52 seconds at 60% MVC. Though they did not indicate how they differentiated between the two muscles, Badier et al. (1993) described average endurance times in the vastus medialis (VMO) and vastus lateralis (VL) of 29 seconds and 32 seconds respectively in six healthy subjects contracting isometrically at 80% MVC, at 60° flexion. Ebenbichler et al. (1998) noted average endurance times of 83.1 seconds (SD±26.2) at 30% MVC, 66.7seconds (SD±18.7) at 50% MVC, and 48.3 seconds (SD±15.3) at 70% MVC.

Studies have also examined quadriceps endurance times during intermittent isometric contractions. Hagberg (1981), and Sjorgaard, Savard and Juel (1988) have reported that endurance times for intermittent contractions were greater than those at equivalent levels of sustained isometric contractions ($p<0.01$) (Hagberg, 1981). This difference in endurance times between the contraction types has been attributed to the different blood-flow characteristics of the contraction types. The intramuscular pressures generated by an isometric contraction have been shown to stop blood flow through a muscle contracting at or above 20% MVC (Edwards, Hill, & McDonnell, 1972). The resulting build up of metabolites, oxygen depletion, and subsequent anaerobic metabolism is believed to cause a state of localised muscle fatigue (Hagberg, 1981; Sjogaard et al., 1988). In contrast to a sustained isometric contraction, the nature of an intermittent isometric contraction is thought to delay the onset of fatigue by allowing blood to flow through the muscle, and delay the metabolic changes that characterise peripheral muscle fatigue.

In both intermittent and sustained isometric contractions, it is the peripheral metabolic changes that are believed to be the major contributing factors to the observed muscle fatigue. This supposition is supported by the work of Bigland-Ritchie, Furbush and Woods (1986). Using a protocol termed *interpolated twitch* (Merton, 1954; Rutherford & Jones, 1988) these authors examined the change in central activation of the quadriceps during a fatiguing protocol of intermittent sub-maximal (50%) isometric contractions. During the fatiguing contraction a supra-maximal electrical stimulus was applied to the muscle, and the resulting increase in force measured. Central fatigue was deemed to be occurring if the size of the superimposed twitch increased, indicating a decrease in central drive to the muscle. The authors reported no evidence of central fatigue, thus concluding that the fatigue observed was due to peripheral mechanisms (B. Bigland-Ritchie, F. Furbush et al., 1986). Using a similar protocol, but this time at an activation level of 30% MVC, Bigland-Ritchie, Caferelli and Vollestad (1986) also reported no evidence of central fatigue in the quadriceps of normal subjects. They also concluded that peripheral mechanisms were responsible for most, if not all of the fatigue observed during the sub-maximal isometric protocol used in their study (B. Bigland-Ritchie, E. Cafarelli et al., 1986).

2.3.2 Maximal isometric protocols

The fatigue characteristics of healthy quadriceps have also been investigated during maximal isometric contractions. A common method of quantifying fatigue has been to measure the decrease in maximal force production during such a contraction. Grange and Houston (1991) for example observed an average decrease in quadriceps force of 43% during a 60 second maximal isometric quads contraction at 90° knee flexion in 12 healthy subjects. Similarly to Grange and Houston, Gerdle, Karlsson, Crenshaw and Friden (1997), along with Karlsson, Ostlund, Larsson and Gerdle (2003) observed force declines of between 40% and 50% in quadriceps, but this time using 40-60 intermittent rather than sustained maximal isometric contractions at 90° flexion. A similar decline in force has also been reported by Mannion and Dolan (1996), who showed a 45% decrease in maximal isometric contractions performed intermittently during a sustained sub-maximal (40%MVC) isometric contraction.

Several studies have also provided evidence to suggest that the angle at which a contraction is held also affects the endurance time for a maximal isometric quadriceps

contraction. In contrast to the 43% decline in force at 90° knee flexion reported by Grange and Houston (1991), McHugh et al. (2001), found only a 4.6% ($p<0.01$) decrease in quadriceps force during a maximal 30-second isometric quadriceps contraction held at 30°. Arendt-Neilson, Gantchev and Sinkjaer (1992) also reported greater sub-maximal (50%MVC) isometric endurance at 45° (52s) compared to 90° (24s) knee flexion ($p < 0.05$).

Both central and peripheral components appear to contribute to the fatigue observed with maximal isometric contractions. The contribution of central factors has been investigated by a number of studies. In contrast to sub-maximal isometric contractions, these studies suggest that the fatigue observed during maximal isometric exercise has a greater central component. An early study by Bigland-Ritchie, Jones, Hosking and Edwards (1978) tested for central fatigue using an interpolated twitch protocol. They examined the decline in central activation of the quadriceps of nine healthy subjects who maintained a maximal voluntary contraction for 60 seconds. Central fatigue was deemed to have developed if the drop in voluntary maximum force was faster than the drop in electrically evoked force. This was observed in six of the nine subjects. In a later study, also using an interpolated twitch protocol, Newham, McCarthy and Turner (1991) described significant decreases in voluntary activation (36.4 +/- 3.1%) during a maximal isometric fatiguing contraction, again attributed to central factors of fatigue. More recent work by Kent-Braun (1999) also examined the central contribution to fatigue during maximal isometric contractions, but this time in the ankle dorsiflexors. Based on a decline in an index related to the twitch interpolation protocol called the *central activation ratio*, Kent-Braun reported that approximately 20% of the decline in force seen during a four minute maximal voluntary contraction was due to central factors. These findings suggest that, as with to sub-maximal isometric contractions, the majority of muscle fatigue that occurs during maximal isometric contractions is mediated by peripheral factors.

2.3.3 Isokinetic protocols

Quadriceps fatigue in healthy subjects has also been examined during isokinetic contractions. Early work by Hagberg (1981) involving the biceps showed no differences in endurance times between isometric and isokinetic contractions performed at an angular velocity of 30°/s. Hagberg proposed that the intra-muscular

pressures during the slow isokinetic contraction were probably very similar to those of the sustained isometric contraction, thus resulting in the same physiological phenomena contributing to the isokinetic induced muscle fatigue, as those described for isometric contractions. Masuda, Masuda, Sadoyama, Inaki and Katsuta (1999) however reported contrasting findings from a protocol of sub-maximal (50% MVC) isokinetic contractions, this time of the quadriceps at a lower angular velocity of 18°/s. They reported that the dynamic test resulted in significantly greater endurance times (range 84-258s, mean 149.7s, SD±50.9s) when compared to a sustained isometric test at the same contraction level (range 54-136s, mean 75s, SD±20.2s). The isokinetic endurance times reported by Masuda et al. (1999) are also clearly longer than those reported in other papers using isometric quadriceps contractions (Arendt-Nielsen & Mills, 1988; Badier et al., 1993; Ebenbichler et al., 1998; Hakkinen & Komi, 1983; Mannion & Dolan, 1996; Nagle et al., 1988). In support of the findings of Masuda et al. (1999), recent work has examined changes in intramuscular pressures during intermittent maximal isokinetic contractions at 90°/s (Crenshaw, Gerdle, Heiden, Karlsson, & Friden, 2000). Using intramuscular pressure catheters, Crenshaw and co-workers reported an increase in the resting intra-muscular pressure between contractions from 6mmHg (+/- 2.1) to 14mmHg (+/- 4.6), as well as a decrease in peak intramuscular pressure during the contractions from approximately 100mmHg to 80mmHg (range 45-245). The significant difference in pressures between relaxation and contraction observed in this study suggest that reperfusion is able to occur, and that muscles undergoing intermittent or dynamic contractions could be expected to fatigue more slowly than during sustained isometric contractions (Crenshaw et al., 2000).

The angular velocity of isokinetic movements has also been shown to affect quadriceps muscle fatigue. Significant differences in force decline have been reported between contractions at 60°/s (44 +/- 18%), and those at 180°/s (65 +/- 14%) and 300°/s (63+/-8%) during 100 maximal isokinetic contractions of the quadriceps in 10 healthy subjects (Perry-Rana et al., 2002). The greater force decline at higher velocities was thought related to the inability of the slow twitch fibres to contribute to the contraction at high angular velocities (Beelen & Sargeant, 1993; Spendiff, Longford, & Winter, 2002). Spendriff et al. (2002) have also reported that maximal fatiguing contractions of the quadriceps at lower angular velocities had a significantly greater effect on the force output of subsequent contractions performed at high and

low velocities (0.52, 1.05, 2.09, 3.14 rad/s). The authors postulated that the fatigue induced during the slow maximal quadriceps contractions influenced both slow and fast twitch fibres, thus affecting the muscle group's ability to perform subsequent contractions at any speed.

The contributions of central mechanisms of fatigue have also been investigated in isokinetic contractions of the quadriceps. It is thought in general, from psychology research, that the contribution of central components to muscle fatigue becomes more pronounced as the velocity and complexity of a task increases (Green & Vaid, 1986; Jones, Allen, Griffiths, Marshall, & Richens, 1986). Investigating this phenomenon in the activation of quadriceps, Newham et al. (1991) tested for central fatigue at two isokinetic speeds (20°/s and 150°/s). Twenty-three healthy subjects first underwent a fatiguing protocol involving maximal contractions of their quadriceps at 85°/sec. In contrast to the expected increase in central fatigue at higher velocities, the authors reported significant declines in voluntary activation levels during the slower isokinetic contraction (28.8 +/- 4.1 %) while observing no change in voluntary activation during contractions at 150°/sec. James, Sacco and Jones (1995) presented similar evidence, in which they found minimal activation failure in quadriceps contracting maximally at 90°/second (James et al., 1995). While the isokinetic speeds used in these studies are considered slow by functional standards (Newham et al., 1991), these findings suggest that central fatigue affecting the quadriceps has a velocity-dependent, and possibly task dependent aspect.

2.3.4 Summary

From the literature involving healthy quadriceps it appears that the onset of muscle fatigue is related to both the type and intensity of contraction. It is also evident that both central and peripheral components contribute to the muscle fatigue observed, but their relative contributions to fatigue may vary, also with contraction type and intensity. However, regardless of the nature of muscle activity, it appears that central components explain only a small portion of the fatigue observed, and that the majority of muscle fatigue during both static and dynamic contractions originates from those components acting at a local muscle level.

2.4 FATIGUE RESISTANCE OF AFFECTED QUADRICEPS

While the ability of a muscle to resist fatigue is an important component of function, few studies have examined fatigue resistance characteristics of the quadriceps or other muscles following injury or due to pathology. Studies that have examined this area have done so with various protocols using isometric and dynamic contractions. Unfortunately the methodologies used do not allow comparison with those studies covered in the previous section that reviewed fatigue in healthy quadriceps muscles.

2.4.1 Isometric contractions

Disuse studies that have examined quadriceps endurance have focused mainly on isometric contractions in various forms. An early study by Halkjaer-Kristensen and Ingemann-Hansen (1985), for example examined isometric quadriceps endurance after one month of immobilisation following knee collateral ligament rupture (n=23). The endurance test required subjects to perform 30 maximal isometric contractions, each lasting for five seconds, with three seconds rest between contractions. Relative fatigue resistance was measured as the point at which the decline in force across the contractions reached a plateau, expressed as a percentage of MVC. The authors reported that those subjects who underwent non-surgical management of their knee injury showed no significant change in relative fatigue resistance ($p<0.05$) when compared to the control group or their own unaffected leg (Halkjaer-Kristensen & Ingemann-Hansen, 1985).

Nordesjo et al. (1983) also assessed quadriceps endurance, but this time using a sustained maximal isometric contraction. This study examined the quadriceps of seven women with knee osteoarthritis, using a fatigue test requiring a maximal quadriceps contraction for 120 seconds. A fatigue curve was generated for each subject by plotting force against time. The shapes and slopes of the fatigue curves were then used to characterise the subjects. The authors described significantly lower initial force values for the osteoarthritis subjects, but significantly less drop in force, or a lesser slope, across the endurance test compared to the control subjects. Some subjects even demonstrated an increase in force across the 120-second contraction. Similar findings were reported by McHugh et al. (2001) while investigating quadriceps endurance in 42 subjects before, and 5 weeks after, anterior cruciate ligament reconstruction. While the testing protocol required a sustained maximal isometric contraction for only 30 seconds, the authors reported no decline in force, and even an increase in force production during the contraction by some subjects. As

with the work of Halkjaer-Kristensen (1985) and Nordesjo et al. (1983), these results imply that the quadriceps of those with knee pathology showed either the same or greater relative fatigue resistance during an isometric contraction than those without knee pathology.

2.4.2 Dynamic contractions

Despite the functional importance of dynamic muscle contractions, few studies have examined muscle fatigue in this context in subjects with injury or pathology. Of these studies, Velhuizen, Verstappen, Vroemen, Kuipers and Greep (1993) have examined fatigue by quantifying the change in isokinetic work output of the quadriceps following immobilisation. Eight otherwise healthy subjects had one leg immobilised for four weeks in plaster. Fatigue resistance characteristics were examined by comparing the maximal power output in Watts (W) and one-legged work in Joules (J) on a cycling ergometer pre- and post-immobilisation, as well as the total work performed during a ramped isokinetic knee extension protocol (180°/s, 100° ROM). Subjects showed no significant changes in power output on the cycle ergometer, but demonstrated a significantly greater relative decrease in total work during the isokinetic protocol (9.1kJ to 5.6kJ, $p<0.05$), indicating a reduction in relative fatigue resistance.

No further work appears to have been done in this regard involving the quadriceps. However, using a single case study of a subject immobilised for eight weeks following ankle fracture, Vandenborne et al. (1998) examined isokinetic fatigue changes in the plantar flexors. The subject was required to contract his plantarflexors 50 times maximally at a constant angular velocity of 60°/s through the full available range. Fatigue in this study was quantified as the decrease in work between the first and final third of the contractions. In contrast to the findings of Veldhuizen (1993), the authors reported that the muscles of the involved limb showed less relative decrease in work, and were therefore deemed to have greater relative fatigue resistance than the muscles of the uninvolved limb. While this study did not quantify the differences in fatigue resistance between limbs, a more substantial study using the same protocol across 10 subjects did (Shaffer et al., 2000). Again, in contrast to the findings of Veldhuizen (1993), Shaffer and co-workers reported that the plantarflexors of the involved limb were significantly more fatigue resistant than the

plantarflexors of the uninvolved limb ($p = 0.004$). Declines in work for the involved side averaged 25.4% (+/- 6.2%) compared to 51.8% +/- 4.8% for the uninvolved side.

2.4.3 Condition dependency

While there is evidence to suggest that the fatigue resistance of a muscle at a given percentage of maximum generally improves following injury or immobilisation, evidence also suggests that there may be a form of condition dependency associated with this phenomenon. This condition dependency implies that muscles may respond differently to different types of injury. For example, while Halkjaer-Kristensen et al. (1985) reported no changes in isometric endurance in the quadriceps of subjects who had received conservative management of collateral ligament rupture (n=23), they reported decreases in fatigue resistance in a separate group (n=64) who underwent surgical repair of the same ligament injury. Similar observations were made by Behm & St-Pierre (1997). These authors also reported no changes in fatigue resistance in the plantarflexors of subjects conservatively managed following ankle fracture, while those managed surgically with internal fixation showed decreases in their resistance to fatigue ($p<0.05$). Muscle fibre atrophy research by Nakamura, Kurosawa, Kawahara, Watarai and Miyashita (1986) have also described the effects of different clinical conditions. These authors examined the patterns of fibre atrophy across a range of knee disorders and reported differences in the atrophy of specific fibre types. These differences corresponded to different injuries, with type-1 fibres being affected only in subjects with anterior cruciate ligament (ACL), and ACL meniscus combination injuries, while type-2 muscle fibres showed changes in each injury class (ACL, ACL and meniscus combined, meniscus, collateral ligament) (Nakamura et al., 1986). Although isolated, these findings reported by Nakamura et al. (1986) perhaps suggest there are mechanisms of atrophy specific to certain conditions, which may correspondingly influence the endurance characteristics of muscles differently. No studies could be found that examined such findings in relation to osteoarthritis of the knee joint.

2.4.4 Summary

While relatively sparse, evidence appears to support the concept that there is a general maintenance or improvement of the relative fatigue resistance of muscles as the result of disuse or immobilisation. Furthermore, these findings raise questions regarding

how a muscle and its central control mechanisms change in order to enhance the relative fatigue resistance of a muscle.

2.5 MUSCLE CHANGES WITH DISUSE/IMMOBILISATION

In a broad context, the plasticity of the neuromuscular system has long been recognised. This plasticity can manifest in both positive and negative ways. Examples of the effects of this plasticity that may influence the fatigue resistance of a muscle can be seen in some of the peripheral and muscle control changes that occur as a result of disuse/immobilisation.

2.5.1 Muscle atrophy

Clinicians, patients, and athletes are familiar with muscle atrophy as the result of disuse and/or injury. This atrophy can be appreciated generally in the reduction of muscle cross-sectional area. In relation to the quadriceps, Convertino, Doerr, Mathes, Stein and Buchanan (1989), for example, reported an 8.1% decrease in their cross-sectional area after 30 days of bed-rest. A larger (12%) decrease in quadriceps cross-sectional area was reported by Berg, Dudley, Haggmark, Ohlsen and Tesch (1991) following six weeks of unilateral lower-limb suspension.

As well as the gross changes observed, more specific atrophic changes can be appreciated at the level of the muscle fibres themselves. An early study by Young, Hughes, Round and Edwards (1982) used an ultrasound scanning and needle biopsy technique to examine the effects of atrophy on muscle fibre number and size in the vastus lateralis quadriceps muscle. The authors reported that the decrease in muscle size seen with disuse is due primarily to a decrease in the cross-sectional area of muscle fibres, and not a decrease in the number of muscle fibres themselves (Young et al., 1982). In contrast to the work of Young et al. (1982), Halkaer-Kristensen and Ingemann-Hansen (1985) reported atrophy due partially to a 28% ($p<0.001$) loss in the number of muscle fibres in the vastus lateralis of subjects immobilised for one month following knee collateral ligament rupture. They however qualified their observations by suggesting the significant atrophy may have altered their biopsy results by changing muscle fibre orientation and muscle fibre length.

While there may or may not be losses in the total number of muscle fibres, some authors have reported a transition between muscle fibre types due to disuse. Examining astronauts following five and eleven days of space flight, Edgeton et al. (1995) noted no change in the number of muscle fibres present in the vastus lateralis of subjects. However, the number of type-1 muscle fibres was seen to decrease, offset by an increase in the number of type-2 muscle fibres. Uhlig, Weber, Grob and Muntener (1995) have also reported this phenomenon in subjects with neck muscle atrophy due to significant cervical spine degenerative changes (n=64), observing significant increases in type-2 glycolytic fibres when compared to normal subjects ($p<0.01$). Lastly, a more recent study looking at quadriceps atrophy following hip replacement surgery also demonstrated significant ($p<0.01$) increases in type-2 muscle fibres at the expense of type-1 fibres (Reardon, Galea, Dennett, Choong, & Byrne, 2001).

As discussed earlier, the fibre composition of a muscle has been related to the fatigue resistance of that muscle. Changes in a muscle's fibre composition due to atrophy may therefore influence the fatigue resistance of that muscle. However, while it is accepted that muscle fibres decrease in size following disuse/immobilisation, which fibres are most affected in limb muscles is not clear from the literature. A number of early studies have suggested that it is the type-1 oxidative muscle fibres that are more affected by atrophy through disuse (Edgerton, Barnard, Peter, Maier, & Simpson, 1975; Haggmark, Jansson, & Eriksson, 1981; Sargeant, Davies, Edwards, Maunder, & Young, 1977). An animal study by Edgerton and co-workers (1975) in which the hind limb of a primate (*Bush baby* - Galago senegalensis) was immobilised, reported pronounced atrophy of type-1 oxidative fibres. A number of studies involving humans appear to support this observation. While not reporting significance figures, Sargeant & Davies (1977) found greater atrophy of type-1 (46%) than type-2 (37%) fibre types in vastus lateralis after immobilisation for an average of 131 days following knee injury (n=7). Haggmark et al. (1981) also reported selective atrophy of the type-1 fibres of 26.5% following immobilisation for 5 weeks following knee surgery. This was significantly different to the type-2 fibres examined ($p<0.0025$), which showed no change in average size over the same period. Halkaer-Kristensen and Ingemann-Hansen (1985) also reported a selective loss of type-1 muscle fibre area (21%, $p<0.001$) in the vastus lateralis of subjects immobilised for one month following knee collateral ligament rupture. More recent work by Portero, Vanhoutte and Goubel

(1996) reported a preferential atrophy of muscles (tibialis anterior 8% vs. gastrocnemius 14%) that have an antigravity or postural function following four weeks of bed-rest (n = 12). The greater atrophy of these muscles that are associated with higher proportions of type-1 muscle fibres lends further support to the premise that muscle atrophy affects type-1 muscle fibres more so than type-2 fibres (Portero et al., 1996).

In contrast to the above findings, preferential atrophy of type-2 glycolytic muscle fibres has also been reported in the literature. Nakamura et al. (1986), for example, noted that type-2 fibre atrophy occurred in separate knee disorders involving the anterior cruciate ligament (ACL), the collateral ligaments, and the menisci or a combination of each, while type-1 fibre atrophy only occurred in ACL and ACL-meniscus combination injuries. Preferential atrophy of type-2 muscle fibres has also been noted as more common (73.1% vs 6.3%) in the vastus lateralis of 26 subjects with knee osteoarthritis when compared to age-matched control subjects (Nakamura & Suzuki, 1992). Examining muscle atrophy resulting from space flight, Edgeton et al (1995) also reported a consistent, but statistically non-significant trend for greater atrophy of the type-2 muscle fibres in the vastus lateralis. Similar, but again non-significant, findings of selective type-2 fibre atrophy were also reported by Rantanen, Hurme and Kalimo (1999) in a study examining the calf muscles of immobilised hind limbs of rats. Further evidence of selective type-2 atrophy is also reported by Reardon et al. (2001). Using needle biopsy the authors sampled the vastus lateralis of 12 subjects before and after total hip joint replacement surgery. Though they did not report significance levels, Reardon and co-workers described reductions in type-2a and 2b muscle fibre size of 41% and 24% respectively in relation to the type-1 fibres of the same muscle.

Other atrophy studies have indicated that all muscle fibres may be affected equally by immobilisation and disuse. In work using computed tomography (CT) scanning and muscle biopsies, Gerber and co-workers (1985) reported no differences in the atrophy of muscle fibre types in the quadriceps of 41 subjects with chronic anterior cruciate ligament insufficiency. Further examination in a group of otherwise healthy individuals (n=8) by Veldhuizen et al. (1993) also yielded no differences in the atrophy of quadriceps fibre types ($p > 0.05$) according to muscle biopsy results following four weeks of lower limb immobilisation. Based on EMG frequency

analysis findings rather than biopsy or CT data, Tho, Nemeth, Lamontagne and Erikson (1997) also concluded there were no differences in the atrophy of type-1 and type-2 fibres in the quadriceps at 4 to 24 months following a rupture of the anterior cruciate ligament in 15 subjects ($p>0.05$) . More recent work by Yoshihara, Shirai, Nakayama and Uesaka (2001) involving biopsy of the lumbar multifidus muscles has also reported significant ($p < 0.01$) atrophy in both type-1 and type-2 muscle fibres in subjects with lumbar disc herniations. While percentage changes in each muscle fibre type were slightly different, the authors reported no significant differences in the changes between fibre types.

Finally, with regard to muscle fibre atrophy, a number of studies investigating the effects of de-training have suggested that the training state of a muscle may influence which muscle fibres are affected most by atrophy as the result of immobilisation or disuse. With regard to type-1 muscle fibres, Larsson and Ansved (1985) for example reported a 10% decrease in the relative area of type-1 muscle fibres in elite rowers following cessation of training. Analogous to Larsson's and Ansved's work, type-2 muscle fibres have been reported to decrease in size in strength-trained athletes after eight weeks of detraining by Hakkinen, Komi and Tesch (1981). This decrease was quantified by a change in the cross-sectional area ratio of type-2 to type-1 muscle fibres from 1.11 to 1.04. A further case-study by Hakkinen and Alen (1986) also reported a reduction in the ratio of type-2 muscles fibres to type-1 muscle fibres from 1.32 to 1.04, this time in an Olympic power lifter after 7 months of detraining. No studies that examined training state with respect to atrophy from immobilisation or disuse could be located.

2.5.2 Changes in oxidative capacity

In addition to changes in fibre-type proportions in muscle, a number of other changes occur with disuse or immobilisation that may influence the oxidative capacity of a muscle. The mitochondria, for example are the main source of energy (Adenosine triphosphate - ATP) for all cells in the body. Their density reflects the energy demands and capabilities of a cell (Marieb, 2001a). It is well established that endurance training causes a significant increase in the numbers of mitochondria in all muscle fibres (Fitts, 1996). Literature examining the effect of disuse/immobilisation on muscle mitochondria is, however, sparse. In an early study involving rat muscle, Krieger, Tate, McMillin-Wood and Booth (1980) have reported a reduction in the

number of mitochondria in some parts of the muscle and not others after two weeks of hind-limb immobilisation. In further work involving humans and space flight, Fitts, Romatowski, De La Cruz, Widrick and Desplanches (2000) did report a loss of mitochondria through disuse, but to a lesser extent than the contractile proteins in the muscles investigated. The authors surmised that this resulted in an increase in the relative density of mitochondria in the disused muscle. In light of their role in fatigue resistance, increases in the density of mitochondria may thus contribute to any improvement in relative fatigue resistance reported following disuse/immobilisation.

The concentrations of oxidative enzymes within the mitochondria are also associated with the fatigue resistance of a muscle (Degens & Veerkamp, 1994). While few in number, studies have shown significant decreases in the activity of these oxidative enzymes as the result of immobilisation. Early findings in this area have shown significant decreases in levels of the aerobic metabolism marker succinate hydrogenase (SDH) after five weeks of knee immobilisation following anterior cruciate ligament repair (Haggmark et al., 1981). More extensive work has supported these findings. Examining 84 healthy young soccer players after one month of knee immobilisation following collateral ligament rupture, Halkjaer-Kristensen and Ingemann-Hansen (1985) also reported significant decreases ($p < 0.001$) in the aerobic metabolism marker succinate dehydrogenase (SDH), and significant increases ($p < 0.001$) in the marker of anaerobic metabolism, α-glycerophosphate dehydrogenase. More recent work by Kitahara et al. (2003) on the forearm has also described a decrease in oxidative enzyme activity following three weeks of cast immobilisation. In the immobilised muscle group the authors measured a 45% decrease in the rate of phosphocreatine recovery, an index shown to be significantly correlated with mitochondrial oxidative enzyme activity (McCully, Fielding, Evans, Leigh, & Posner, 1993), when compared to the unaffected arm (Kitahara et al., 2003). In contrast to the apparent increases in relative mitochondrial density, the reduction in concentrations of oxidative enzymes reported by these studies would appear to contribute to a reduction rather than an improvement in the relative fatigue resistance of a disused/immobilised muscle.

Muscle capillarisation has also been related to the fatigue resistance of a muscle. This factor represents the ease with which blood can be supplied to a working muscle. In relation to this factor, Behm and St-Pierre (1998) have suggested that any

improvements in relative fatigue resistance seen with disuse may be related to improvements in the perfusion of blood within a muscle. The validity of this hypothesis appears to rely on studies that show less atrophy or loss of capillaries in relation to the loss of muscle mass from disuse/immobilisation. In support of Behm and St Pierre, Hather, Adams, Tesch and Dudley (1992), for example, have reported no decrease in the number of capillaries, and a significant increase ($p < 0.05$) in the density of capillaries (14-15%) in the quadriceps and hamstring muscles after six weeks of lower-limb suspension (n=8). Other studies have been more equivocal. In a study similar to that of Hather et al. (1992) involving six weeks of lower-limb suspension, Berg, Dudley, Hather and Tesch (1993) also reported no change in the number of capillaries present per muscle fibre, but did not measure capillary density. Further work by Ferretti et al. (1997) involved more extreme disuse with 42 days of bed-rest for seven healthy subjects. Despite showing a 22% decrease ($p < 0.05$) in capillary length, similarly to Hather et al. (1992), they reported no change in the number of capillaries in the thigh muscles. As with the work of Berg et al (1993), Ferretti and co-workers did not report capillary density figures. While it is evident from these studies that changes in muscle capillarisation occur with disuse/immobilisation, the influence on any changes in perfusion are not clear. Furthermore, the influence of perfusion changes on improvements in relative fatigue resistance following disuse is difficult to verify.

2.5.3 Contraction coupling mechanism

The maintenance of a muscle contraction is influenced to some degree by the efficiency of the contraction-coupling mechanism. The contraction coupling mechanism includes the structural and chemical components of a muscle that are responsible for the physical shortening of a muscle during muscle activation (Marieb, 2001b). The sarcoplasmic reticula (SR) of muscle cells are an important component of the contraction-coupling mechanism. Changes in SR activity following disuse have been reported in several animal studies (Kim, Witzmann, & Fitts, 1982; Schulte, Navarro, & Kandarian, 1993). In an early *in vitro* study, Kim et al. (1982) reported significant increases in the activity of SR in rat muscle after six weeks of immobilisation. Similarly, Schulte et al. (1993) also described significant increases in SR activity in rat hind-limb muscle after 28 days of immobilisation. Consequently, Behm and St-Pierre (1998) have contended that this relatively greater activity of the

SR results in greater movement of calcium in and out of the contractile mechanism of a muscle. These authors further stipulate that the greater SR activity improves the efficiency of the contraction-coupling mechanism, and may prolong muscle activation.

Furthermore, in relation to the contraction-coupling mechanism, an early animal study by Kentish (1986) reported that elevated levels of inorganic phosphate (Pi) may inhibit cross-bridge cycling during muscle contractions of rat heart muscle. Phillips, Wiseman, Woledge and Kushmerick (1993) reported also that elevated muscle Pi levels inhibited the contraction coupling-mechanism in rat soleus muscle. Subsequent studies using magnetic resonance spectroscopy have shown significantly elevated levels of inorganic phosphate (Pi) in disused calf muscle (Vandenborne et al., 1998), as well as patients with muscular damage through disease (Argov & Bank, 1991) or injury (McCully et al., 1988). Behm and St-Pierre (1998) have thus speculated that such an inhibition of cross-bridge cycling may lead to an improvement in the efficiency of the contraction coupling mechanism, and improved resistance to fatigue (Behm & St-Pierre, 1998)

2.5.4 Central Control Changes

Muscles are controlled by the central nervous system. Disuse and immobilisation through injury appear to induce changes in the role of the central nervous system in the control of a muscle. These changes include alterations in the voluntary activation of a muscle, and the rate at which a muscle is stimulated by the central nervous system.

Voluntary Activation

The impairment of the central-nervous-system's ability to activate a muscle following injury results in a loss of force that can be generated by that muscle. In relation to injury or pathology, this impairment has been termed *arthrogenic muscle inhibition* (Young, 1993), and results in a voluntary activation deficit in a muscle. Although the levels of activation deficit reported vary widely, a number of authors have provided evidence that patients with knee osteoarthritis have significant decreases in their ability to activate their quadriceps. Hurley and Newham (1993), for example, investigated muscle inhibition in ten subjects with moderate knee osteoarthritis using an interpolated twitch protocol. They reported a mean decrease in muscle activation

levels of 19% (SEM +/-7%). In a larger, more recent study Pap, Machner and Awiszuz (2004) examined 68 subjects with early osteoarthritis. They reported a mean voluntary activation deficit of 29.8%. This study also investigated voluntary activation levels in 154 subjects with severe osteoarthritis. Interestingly, voluntary activation deficits in these subjects were shown to be less than those found in subjects with early osteoarthritis (22.8%). Voluntary activation deficits in both patient groups were significantly different ($p < 0.01$) to the control group (10.7%).

In light of evidence showing voluntary activation deficits in the quadriceps of subjects with knee osteoarthritis, closer examination is warranted of the work describing maintenance or improvements in the relative fatigue of muscles following disuse, immobilisation or injury. In the work of Nordesjo et al. (1983) described earlier, for example, the authors reported that five of the 12 female subjects with knee arthritis in their study could not complete the fatiguing test due to pain. All subjects were also said to have moderate to severe knee osteoarthritis. Because of the pain, or the significant structural joint damage, or possible concurrent swelling (Hopkins, Ingersoll, Krause, Edwards, & Cordova, 2001) that are likely to be associated with this level of knee arthritis, it is unlikely that the subjects in this study were able to activate their quadriceps fully during the maximum voluntary contraction required for the fatigue test. The same issue of reduced voluntary activation levels could also be said to have affected the work of Halkjaer-Kristensen and Ingemann-Hansen (1983) involving collateral-ligament-deficient knees. An inability to activate fully the quadriceps during a maximal contraction may have implications on the endurance times reported for an isometric endurance test such as that used by Halkjaer-Kristensen and Ingemann-Hansen (1985). In subjects with diminished voluntary activation, a maximum voluntary contraction would generate less than its true maximum, and hence the *maximum* voluntary contraction is in effect *sub-maximal*. This appears relevant in light of the work of Hagberg (1981) discussed earlier, where a muscle working at a lower percentage of its maximum is expected to fatigue more slowly. Thus, it would appear that a muscle working at less than its true maximum due to an activation deficit, such as may be the case in the arthritis studies of Nordesjo et al. (1983) and Halkjaer-Kristensen and Ingemann-Hansen (1985), will perform better in an isometric endurance test compared to an unaffected muscle working at its true maximum, thus giving an impression of improved relative fatigue resistance.

Electrical Stimulation

In attempts to overcome the issue of activation levels during fatigue testing, experimental methods involving various forms of electrical stimulation have been used to circumvent the central mechanisms responsible for a decrease in voluntary activation. In this manner, Snyder-Mackler, Ladin, Schepsis and Young (1991) investigated quadriceps endurance following anterior cruciate ligament (ACL) reconstruction in 18 otherwise healthy subjects. The authors utilised electrical stimulation to generate a sub-maximal fatiguing contraction of the quadriceps. Despite these efforts to control for the effect of decreased voluntary activation levels, the authors still reported increases in fatigue resistance of the quadriceps following ACL reconstruction ($p<0.001$) (Snyder-Mackler et al., 1991). Other studies on different muscle groups have also reported similar results. Duchateau and Hainaut (1987) found increases in fatigability following immobilisation of hand muscles and testing with a fatiguing protocol using full muscle electrical stimulation to adjust for activation deficits. A more recent study by Behm & St-Pierre (1997) examined fatigue of the ankle plantarflexors following non-surgical management of ankle fractures (Behm & St-Pierre, 1997). The authors used a twitch interpolation method (Merton, 1954; Rutherford & Jones, 1988) to estimate the true maximum force that could be generated by each subject's quadriceps. To standardise the contraction intensity used during the fatiguing task, and thus reduce the effect of diminished central activation, the subsequent sub-maximal (50%MVC) contractions were calculated using a true maximum force value calculated from the interpolated twitch procedure. Despite this procedure, this study also reported no differences or improvements in the relative fatigue resistance of plantarflexors in the control group when compared to the experimental group. Possible explanations for these findings were offered by Snyder-Mackler et al. (1991) in relation to their own study. These authors proposed that the increase in endurance times may have been the result of selective atrophy of type-2 muscle fibres, resulting in a muscle with a greater representation of type-1 muscle fibres. The authors also felt that a loss of type-2 fibre area was also consistent with the lower force levels generated by the quadriceps in the reconstruction group (60.3%, $p<0.001$) compared to the control leg in their study. Finally, the authors also hypothesised that they may have activated a greater number of type-1 muscle fibres in the reconstruction group compared to the control group during the muscle stimulation used for the fatiguing contraction. Type-1 muscle fibres have been shown to reside more deeply in a muscle belly (Johnson, Polgar, Weightman, & Appleton, 1973).

These deeper fibres may have thus been activated to a greater extent in the reconstruction group than in the control group due to the greater current intensities used by the authors to adequately stimulate the quadriceps of the reconstruction group. A greater type-1 contribution to the fatiguing electrically- evoked contraction may have been a contributed to the increase in endurance times recorded. This hypothesis appears to be supported by work demonstrating lesser fatigue rates during isometric contractions associated with quadriceps muscles that have a higher proportion of type-1 muscle fibres in their structure (Hulten et al., 1975).

Firing Rates

The rate at which a muscle is stimulated by the central nervous system, known as the firing rate, is a method the central nervous system has at its disposal to vary force production. Work by Duchateau and Hainaut (1990) has reported a decrease in the maximum firing rate of muscles following six to eight weeks of immobilisation. These authors examined the adductor pollicis and first dorsal interossei of 5 subjects, and reported a decrease in maximal firing rates of between 39% and 43%. These maximal firing rates following immobilisation were significantly lower than the contralateral muscle of each subject ($p < 0.001$). Behm & St-Pierre (1998) have hypothesised that these decreases in firing rates may contribute to maintaining or improving the relative endurance characteristics of a disused muscle. In this case they surmised that the reduction in motor unit firing would reduce the metabolic stress on the muscle membrane, reducing membrane impairment and ultimately prolonging muscle contraction.

2.5.5 Summary

It is clear that there is a number of changes that occur in muscle as the result of disuse/immobilisation. These changes include muscle atrophy, changes in the oxidative capacity and contraction coupling mechanism, as well as alterations in the central control of a muscle. Indications from the literature are that these changes contribute to the maintenance of or even an improvement in the relative fatigue resistance of a muscle following disuse/immobilisation. Notably, it also appears that the mechanisms responsible for the alterations in fatigue resistance are predominantly peripheral.

2.6 MEASUREMENT OF FATIGUE USING SURFACE ELECTROMYOGRAPHY

A number of clinic-based mechanical endurance tests are available to investigate muscle fatigue. Other laboratory-oriented tests such as muscle biopsy, MRI and surface electromyography (sEMG) are also available. While clinical tests offer simplicity and practicality, laboratory methods offer the advantage of being able to examine specific peripheral components of muscle fatigue, distinct from central components such as the motivation levels of a subject. Among the laboratory methods available, surface electromyography is a non-invasive method that has been used extensively to examine the peripheral components of muscle fatigue.

Surface electromyography (sEMG) involves the measurement of the electrical potentials generated by a contracting muscle. Muscle may be divided functionally into motor units, that is, groups of homogeneous fibres that are supplied by a common motor nerve. These motor units can be of varied size and fibre type (De Luca, 1979). The electrical potential that propagates along individual motor units is termed a motor unit action potential (MUAP), and can also be referred to as the M-wave. Invariably, multiple motor units (MU) are active during a muscle contraction. The number of motor units activated during a contraction has been shown to be dependent on the force required for that contraction (De Luca, 1979, 1984; Guha & Anand, 1979). It is the MUAPs of these active MUs that can be detected by electrodes on the surface of the skin. The conventional surface electromyographic (sEMG) signal represents the temporal and spatial sum of the MUAPs that pass beneath one or multiple electrodes (Hagg, 1992; Kamen & Caldwell, 1996; Soderberg & Knutson, 2000) and can be analysed to provide physiological indices that relate to muscle fatigue.

Two fundamental domains may be used to represent the sEMG signal: temporal and frequency. Typical measures of the temporal domain relate to the amplitude of the signal and the muscle fibre conduction velocity. Amplitude measures include the *Average Rectified Value* (ARV) and *Root Mean Square* (RMS) (Merletti, Knaflitz, & De Luca, 1990). These measures give an indication of the level of activity in a muscle, and have been related to motor unit recruitment, changes in motor unit firing rate, synchronisation of motor units and the duration of contributing action potentials

(Dimitrova & Dimitrov, 2003). Muscle fibre conduction velocity can be measured directly using a number of methods (Broman, Bilotto, & De Luca, 1985b). A method commonly used for direct CV estimation is the cross-correlation technique. The cross-correlation technique requires at least two differential signals from three electrodes. A cross-correlation function is used to compare the waveforms of the signals, a correlation coefficient greater than 0.7 implying that the signals are sufficiently similar for CV estimation (Merletti, Rainoldi, & Farina, 2001). With the correlation value satisfactory, the time delay between the two signals is calculated and used to estimate the conduction velocity (Merletti et al., 2001).

In the frequency domain, the sEMG signal is analysed using a power spectrum representing the level of signal power present at certain frequencies. A power spectrum, such as the one in figure 2.1, is calculated using a mathematical function called a *Fourier Transformation*, and provides a snapshot of the frequency content of a signal over a given time or epoch (Kamen & Calwell, 1996). A typical epoch used in sEMG frequency analysis is 0.5 seconds. The typical frequency range of a sEMG signal is known to be between 5 and 450Hz (De Luca, 1997). A number of parameters can be calculated from an EMG power spectrum. These include central tendency measures such as median frequency (MDF) and mean power frequency (MPF). The MDF divides the power spectrum into two portions containing equal amounts of power, while the MPF is the average frequency of the power spectrum (Kamen & Calwell, 1996). Measures such as these allow changes in the frequency content of the sEMG signal to be appreciated over time. Of these central tendency measures, MPF has been reported to be the most sensitive to any changes in the sEMG power spectrum (Hary, Belman, Propst, & Lewis, 1982). However, in a theoretical study by Stulen and De Luca (1981), MDF was reported to be less sensitive to noise, and was deemed the preferred parameter of the two.

Ratio measures have also been used within the literature to describe the power spectrum. These examine the power within certain parts of the spectrum in relation to the power across the whole spectrum. While earlier studies examined the relative power in broad “high” and “low” frequency bands (Badier et al., 1993), more recent studies have used the relative power in narrower frequency bands from 5 to 30, and 30 to 60 Hertz: frequency band 1 (FB1) and frequency band 2 (FB2) respectively (Dolan, Mannion, & Adams, 1995; Maisetti, Guevel, Legros, & Hogrel, 2002b).

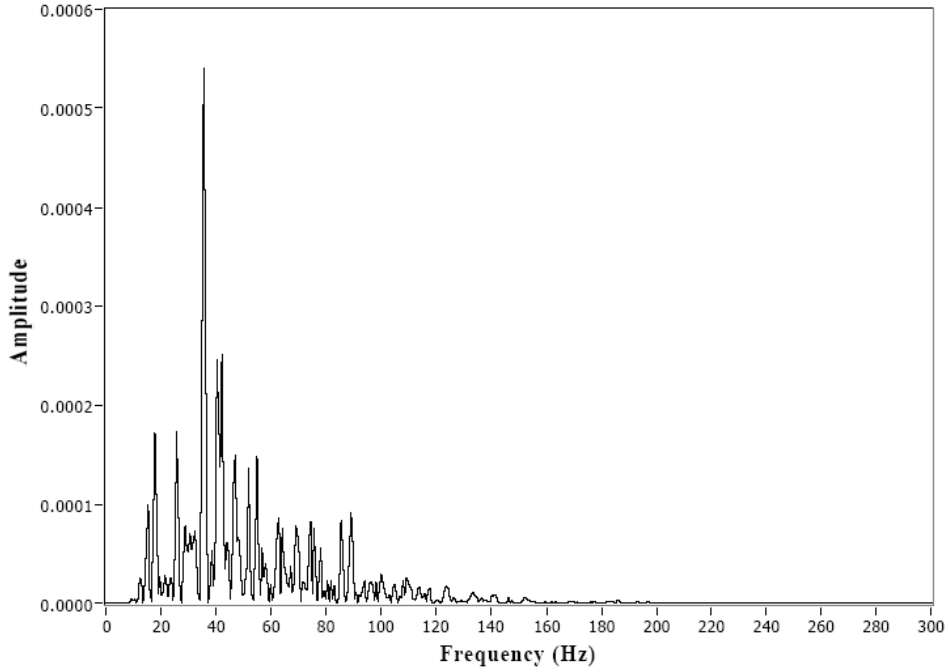

Figure 2.1. sEMG Power Spectrum.

Power spectrum generated from a Fourier transformation of a surface-electromyographic (sEMG) signal.

2.6.1 THE INFLUENCE OF FATIGUE ON THE EMG SIGNAL

Since the historical work of Piper (1912) it has been known that both the amplitude and frequency content of the sEMG signal change during the course of a sustained, fatiguing muscle contraction. These changes in the signal manifest as an increase in sEMG amplitude parameters, and a decrease in sEMG frequency and conduction velocity parameters (De Luca, 1997). While the amount of change in these parameters reported in the literature varies, the direction of change appears consistent in healthy subjects (C. J. De Luca, 1984; Maisetti et al., 2002a; Merletti & Roy, 1996). The changes in these parameters have been related to peripheral factors, along with changes in the central control of a muscle.

2.6.1.1 Peripheral Factors

Peripheral factors appear to generate the majority of the changes seen in the sEMG signal with muscle fatigue. These peripheral factors include conduction velocity, membrane excitability and muscle fibre-composition. In relation to the dominance of peripheral factors in sEMG signal changes, studies by Kranz, Williams, Cassell, Caddy and Silberstein (1983), and Merletti et al. (1990) for example have shown that sEMG changes during fatigue induced by a voluntary contraction are the same as those seen during an electrically evoked contraction. These authors concluded that significant contributions by central factors to the change in the sEMG signal would have seen significant differences in the behaviour of the sEMG signal between the voluntary and electrically invoked contractions. That there was no significant difference suggests that the changes evoked in the myoelectric signal by fatigue are due, mainly, to changes in the peripheral muscle environment (Kranz et al., 1983; Merletti et al., 1990).

In line with studies that have shown a correlation between peripheral physiological changes and the onset of fatigue, changes in the sEMG power spectrum have also been shown to be related to the process of peripheral muscle fatigue, and its underlying physiological changes, by a number of studies utilising magnetic resonance imaging (MRI) with sEMG. Vestergaard-Poulsen and co-workers (1992), for example, reported a high linear correlation between muscle pH levels and decreases in MDF ($r = 0.82$). Significant correlations were also reported between H^+

($r = 0.34$) and $H_2PO_4^-$ ($r = 0.45$) concentrations and decreases in MPF in calf muscles of healthy subjects in a study by Laurent et al. (1993). It is these links between the sEMG power spectrum and muscle physiology that make sEMG an appealing medium through which to examine peripheral muscle fatigue.

Conduction velocity

The role of muscle fibre conduction velocity (CV) has been of particular interest to many researchers (Arendt-Nielsen & Mills, 1985, 1988; Brody, Pollock, Roy, De Luca, & Celli, 1991; Broman, Bilotto, & De Luca, 1985a; Merletti & Roy, 1996). This interest has stemmed from the link between changes in CV and metabolic changes in a muscle such as decreases in intramuscular pH and an accumulation of metabolites (Brody et al., 1991; Sadoyama et al., 1988). Early mathematical models by Lindstrom, Kadefors and Petersen (1977) and Stulen & De Luca (1978) predicted that a reduction in conduction velocity would result in an increase in the time it took for MUAPs to travel along the muscle fibre, and cause an increase in the duration of the MUAP. The authors speculated that this slowing and width increase elicited by a fatiguing contraction would cause a shift in the sEMG power spectrum towards lower frequency levels. Empirical studies have supported these hypotheses, with studies by Sadoyama and Miyamo (1981) and Arendt-Nielsen & Mills (1985) reporting relationships between CV and the changes in the sEMG power spectrum. While the effect of CV changes on the sEMG power spectrum is recognised, the magnitude of this effect during sustained fatiguing contractions is debated. A number of studies have shown that while spectral parameters such as MDF decrease with CV, these decreases are not totally explained by the changes in the MUAP waveform (duration) caused by the decrease in CV.

Bigland-Ritchie, Donovan and Roussos (1981), for example, examined CV and MDF changes in thumb muscles during fatiguing and non-fatiguing contractions. Based on the model of Lindstrom et al. (1977), they proposed that an absolute relationship between CV and power spectrum changes would mean that regardless of how they reduced CV, there should be a corresponding reduction in MDF. These researchers examined this by cooling muscles during fatiguing and non-fatiguing contractions. Their results showed that while CV and MDF were reduced with muscle cooling, the reduction in CV was significantly greater than the reduction in MDF. They concluded

from this that there must be reasons other than a decrease in CV for the changes in the power spectrum seen with muscle fatigue. Low subject numbers (n = 3), and the use of an indirect method of measuring CV add caution to the interpretation of these results. However, more substantial work by Broman et al. (1985) drew similar conclusions. Examining CV in eight healthy subjects, Broman and co-workers reported that spectral parameters decreased almost twice as much as conduction velocity during a sub-maximal (80%) isometric fatiguing contraction of tibialis anterior . In further support of these findings, Brody et al. (1991) in an *in vitro* study involving isolated hamster diaphragm muscle also found that while the CV decreased during a sustained contraction, its change was only 65% of that seen in the MDF. They thus also concluded that the changes in CV were not wholly responsible for the changes in waveform shape and characteristics that affect the sEMG power spectrum during a sustained fatiguing voluntary muscle contraction.

Membrane excitability

In addition to a reduction in conduction velocity (CV), other peripheral mechanisms have been suggested as causative in the MUAP waveform changes that affect the sEMG frequency spectrum as a result of fatigue. Changes in muscle membrane excitability, for example, had been shown to affect the shape of the MUAP independent of a change in CV in rat soleus muscle (Juel, 1986). In addition, a study involving hamster diaphragm muscle by Brody et al. (1991) related changes in MUAP shape to alterations in MDF. The authors reported that artificially decreasing membrane excitability through a decrease in muscle pH *in situ* resulted in an increase in the width of the MUAP during an electrically evoked fatiguing contraction. This widening of the MUAP was thought to explain the greater decline in MDF in comparison to the decline seen in CV ($p<0.01$) reported in their study. Recent work by Dimitrova and Dimitrov (2003) supports the association between MDF change and M-wave width made by Brody et al. (1991). Dimitrova and Dimitrov (2003) showed through mathematical modelling that changes in MUAP width caused changes in MDF independent of changes in CV. They also reported that changes in other MUAP characteristics, such as overall duration, duration of different phases of the wave, and amplitude, also caused decreases in spectral variables in addition to those seen due to changes in MFCV alone.

Fibre composition and surface electromyography

Another peripheral factor known to influence characteristics of the sEMG signal is the fibre composition of a muscle. This relationship is based around the conduction velocity and metabolic differences associated with the various fibre types that make up a muscle. As mentioned earlier, these fibre types also differ metabolically, with these differences manifesting as different fatigue characteristics, as well as different conduction velocities (Kupa, Roy, Kandarian, & De Luca, 1995). This combination of conduction velocity and metabolic differences has been related to differences in the behaviour of sEMG variables collected from muscles with different muscle fibre compositions. For example, the shorter duration of the MUAP associated with type-2 muscle fibres contributes high frequency components to the sEMG signal (Kupa et al., 1995). Thus a muscle with a greater proportion of type-2 muscle fibres can be expected to have higher general frequency values than a muscle with a lesser proportion of type-2 fibres.

Different rates of change of sEMG variables have also been related to muscle fibre types. *In vivo* work by Kupa et al (1995), for example, reported that muscles with higher proportions of type-2 muscle fibres had not only greater general frequency values, but also saw greater decreases in frequency during a fatiguing contraction. The reasons for the greater magnitude of change in sEMG parameters were thought to relate to the metabolic and membrane characteristics of the different muscle fibre-types. Two factors that have been related to a change in conduction velocity are extra-cellular potassium accumulation and decrease in muscle pH. In relation to potassium accumulation, type-2 muscle fibres have been shown by Juel (1998) to be less capable than type-1 fibres of preventing and buffering against its accumulation. With respect to muscle pH, it is recognised that type-2 fibres produce and accumulate more lactic acid than type-1 fibres during a fatiguing contraction (Tesch, Komi, Jacobs, Karlsson, & Viitasalo, 1983), resulting in a lowering of pH levels and a slowing of CV (Brody et al., 1991). The decreases in CV and membrane excitability seen under both of these conditions are believed to contribute to the greater magnitude of sEMG parameter change reported in muscles with a higher proportion of type-2 fibres.

Differences in the sEMG signal collected from the quadriceps of different individuals have been observed in a number of studies. Some of these differences have been related to different muscle fibre composition between those individuals. Early work

by Komi and Tesch (1979) observed the behaviour of the mean power frequency (MPF) of subjects with low and high proportions of fast twitch fibres in their vastus laterali during 100 maximal isokinetic contractions. They reported that the absolute and relative declines in MPF were greatest in subjects with a higher proportion of type 2, or fast twitch muscle fibres. MPF was seen to decline 13+/- 4 Hz (12 +/- 4%) in subjects with a lower proportion of fast twitch fibres compared to 26+/- 3 Hz (25+/- 3%) in subjects with a high proportion of fast twitch fibres ($P<0.001$).

Linssen and co-workers (1991) reported contrasting findings during isometric contractions. These authors conducted a study in which subjects were required to contract thirty times per minute at 80% MVC until the target force could no longer be reached. The study used six patients with 95-100%, and two with 80% type-1 fibre proportions due to a congenital myopathy, who were compared to 12 subjects with a normal distribution of muscle fibres (45% type-1). While the authors noted a trend towards longer endurance times and lesser MDF declines in both of the type-1-dominant groups compared to the controls, this trend did not attain statistical significance.

Gerdle et al. (1997) have also demonstrated a relationship between MDF decline and muscle fibre composition during isometric contractions of the quadriceps. The authors reported moderate but significant correlations between MDF decline and the measures of type-2 fibre proportion, and average fibre cross-sectional area in vastus lateralis during sub-maximal fatiguing isometric contractions (30% MVC: $r = 0.57$, $r = 0.53$; 70% MVC: $r = 0.52$, $r = 0.52$). These moderate correlations between MDF and fibre type were consistent with moderate linear relationship between *endurance times* and type-1 fibre percentage ($r=0.7$) reported by Hulten et al. (1975), in vastus lateralis for a sub-maximal (50%MVC) isometric quadriceps contraction.

Surface electromyography has also been used to investigate the fatigue characteristics of the different quadriceps within individuals. Mannion and Dolan (1996), for example, have reported different magnitudes of fatigue in vasti lateralis and rectus femoris of 10 healthy knees, as measured using EMG parameters. During isometric quadriceps contractions held to failure at 20, 30, 40, 50 and 60% MVC, they reported a 28%-32% decrease in the MDF from rectus femoris, and 15-24% decrease in MDF from the vastus lateralis. The differences between the two muscle groups were

statistically significant ($p<0.01$). These findings appear consistent with morphological studies reporting increased proportions of type-2 fibres in rectus femoris (70.5% surface; 58% deep, Johnson et al., 1973) when compared to the vastus lateralis (67.3% in superficial fibres; 53.1% in deep fibres, Johnson et al., 1973; 64.8% Gerdle, 1997), and vastus medialis (56.3% surface; 38.5% deep, Johnson et al., 1973; 35.2% Gerdle, 1997) muscles. These different compositions suggest both slightly different functional roles of each muscle, and also different fatigue characteristics of the individual muscles within the quadriceps group.

Other EMG studies have investigated the fatigue characteristics within the quadriceps group, but expressed them in terms of the slopes or rates of change of the EMG parameters. Grabiner et al. (1991), for example examined differences in fatigue rates between vastus lateralis and vastus medialis. During isometric contractions at 30% MVC they calculated normalised regression slopes of –0.12 (SD 0.11) and –0.20 (0.07) for vastus medialis and vastus lateralis respectively, and slopes of –0.14 (0.11) and –0.21 (0.15) respectively during a contraction at 60% MVC. Although the differences were non-significant ($p > 0.05$), the authors noted a trend for more rapid decline in MDF in vastus lateralis compared to vastus medialis (Grabiner et al., 1991). Additionally, using an isometric contraction held at 50%MVC until exhaustion in 18 healthy subjects, Ebenbichler et al. (1998) showed graphically that there was no difference between absolute regression slopes of MDF for vastus lateralis and vastus medialis (-0.2Hz/s, $p > 0.05$). However, they did indicate greater regression slopes (-0.29Hz/s) for rectus femoris during the same test. In contrast to the inconclusive results of Grabiner et al (1991), and the results of Ebenbichler et al (1998) a more recent study by Maisetti et al (2002) did show greater fatigue rates in VM compared to VL (slopes for MPF of –0.143 (0.090) for VL, -0.191 (0.102) for VM, and for MDF VL of –0.146 (0.091) and VM –0.176 (0.094) at 50% MVC in 14 healthy subjects (Maisetti et al., 2002b).

2.6.1.2 Central Mechanisms

In addition to the role of peripheral components, changes in the central mechanisms of firing rate modulation, recruitment and synchronisation are believed to explain a portion of the frequency shifts seen in the sEMG spectrum with the onset of fatigue.

Firing rate

The firing rate (FR) of a motor unit is the number of times it is stimulated per second by the central nervous system. It is expressed in Hertz (Hz), or can be inversely expressed as inter-pulse intervals (IPI): the time interval between pulses of a motor unit. An increase of a motor unit's firing rate is a method the central nervous system has at its disposal to increase or maintain force. Its relative importance in force production is known to vary from muscle to muscle, and with the force levels involved (De Luca, 1984). Early studies have reported that most muscles rely on both recruitment and firing rate strategies to generate force below 75% of their MVC, while force generation and maintenance above this level relies on increases in MU firing rates (De Luca, 1984). In relation to specific muscles, Kulkuka and Clamann (1981) have indicated that muscles with a predominance of type-1 fibres rely more on firing rate modulation for force changes, when compared to muscles with a predominance of type-2 fibres. With respect to most muscles, including the quadriceps, firing rates are the main mechanism of force increase above approximately 75% of maximum voluntary contraction (De Luca, 1984).

Early theoretical papers by Lindstrom et al. (1977) and Stulen & De Luca (1978) have predicted, using mathematical modelling, that the firing rates of motor units would affect the lower end of the sEMG power spectrum. This effect was predicted to be most significant between 0 and 40Hz, and several empirical studies have provided some evidence to support this hypothesis. Van Boxtel and Schomaker (1983), for example, observed distinct peaks in the power spectrum between 10 and 40Hz during varied force contractions of facial muscles. They attributed these peaks to the dominant firing rates of the muscles being examined. These peaks were generally seen at lower contraction levels, and were smoothed out as contraction intensity increased. This smoothing was thought to be due to the recruitment of more and larger MUs with a variety of firing rates, such that there was no longer a dominant firing rate. Further work by van Boxtel (1984) reported that firing-rate peaks observed in the power spectrum were inversely related to the strength of muscle contraction, and therefore the effect on sEMG power spectrum parameters decreased with increasing level of muscle activation.

Firing rates and fatigue

While it is acknowledged that a change in motor unit firing rates has an influence on the frequency content of the power spectrum, it is not clear whether or not firing rates actually change during a constant force fatiguing contraction. De Luca (1979) presents a model that predicts a gradual decrease in firing rates of MUs during constant force fatiguing contractions. Bigland-Ritchie, Dawson, Johansson and Lippold (1986) provide support for this model with an empirical study, showing a decrease in MU firing rates during a 50% fatiguing contraction of the quadriceps, although the magnitude of the decrease was not quoted**.** Work by Garland, Enoka, Serrano and Robinson (1994) also reported a gradual decrease in firing rates in 32 of 45 motor units monitored from the start of a sub-maximal fatiguing contraction in an upper limb muscle (biceps brachii).

A number of authors have also reported no changes in motor unit firing rates with fatigue. Bigland-Ritchie, Cafarelli and Vollestad (1986), for example reported no changes in MU firing rates in the quadriceps during a contraction at 30% of MVC. Later work by Christova and Kossev (1998) examined firing rates in seven healthy biceps brachii muscles during a fatiguing contraction at 50% MVC, and found no change. These findings were also replicated in a subsequent study by the same authors (Christova & Kossev, 2001). Additionally, using a 50% sub-maximal contraction, Thomas and de Valle (2001) examined firing rates in the triceps muscle of normal and spinal cord injured subjects. They, too, found that firing rates during the fatiguing contraction did not change in either group with fatigue.

In contrast, other studies have reported increases in firing rates associated with fatiguing contractions. Dorfman, Howard and McGill (1990), for example, examined motor unit firing rates in the biceps brachii (n = 10) during a sub-maximal (30% MVC) fatiguing isometric contraction. They reported an initial reduction in firing rates, followed by a more pronounced, gradual increase in firing rates as the contraction progressed to failure ($p<0.05$). A more recent study by Jensen, Pilegaard and Sjogaard (2000) also looked at a sub-maximal (20%MVC) fatiguing contraction in an upper limb muscle, supraspinatus (n=8). Jenson and co-workers also reported an increase in firing rates within the supraspinatus. While this increase did not attain statistical significance, Jenson and colleagues surmised that any increase in firing rates was caused by an increase in FR variability. They believed that this variability

was likely due to the recruitment of additional, larger motor units, rather than an increase in the firing rates of already active motor units. It is possible that recruitment also accounted for the FR increases observed by Dorfman and co-workers (1990).

Recruitment

Recruitment is a phenomenon that is known to influence the power spectrum of the sEMG signal through an increase in average muscle fibre conduction velocity. Recruitment typically follows the 'size principle' (Henneman & Olson, 1965) in which larger motor units are progressively recruited as the force of a contraction increases (Broman, De Luca, & Mambrito, 1985; Gazzoni, Farina, & Merletti, 2001; Moritani & Muro, 1987). These larger MUs have higher recruitment thresholds, are capable of generating higher forces, and have higher conduction velocities (Hakansson, 1956; Kupa et al., 1995). As a result of recruitment of these larger motor units, increases in the average muscle fibre conduction velocity (Kupa et al., 1995), as well as increases in the power spectrum frequency measures have been observed (Bigland-Ritchie et al., 1981; Broman, De Luca et al., 1985; Moritani & Muro, 1987). The recruitment of these motor units has also been associated with an increase in surface EMG amplitude measures, such as integrated EMG and the RMS of the signal (Bigland-Ritchie et al., 1981; Komi & Tesch, 1979; Moritani & Muro, 1987). An increase in integrated-EMG and RMS values during fatiguing contractions has been also observed consistently in the literature. It has been proposed that these sEMG amplitude changes associated with fatiguing contractions, as with varied force contractions, are also related to recruitment of new motor units (Arendt-Nielsen & Mills, 1988; Edwards & Lippold, 1956; Maton, 1981).

Recruitment and fatigue

Early work by De Luca (1984) questioned the role of recruitment in the increases in sEMG amplitude measures seen with fatiguing contractions. De Luca reported increases in the amplitude of the EMG signal in the first dorsal interosseous muscle of the hand during a fatiguing contraction at 80% MVC, a level of contraction at which this muscle is known to have recruited all of its motor units. As the muscle was fully recruited at this level, De Luca believed that further increases in sEMG amplitude were most likely unrelated to recruitment of additional MUs.

Further work in this area has subsequently produced data from which it appears recruitment does occur with a constant force fatiguing contraction at contraction intensities at which full recruitment is not anticipated. Thomas and de Valle (2001), for example, investigated the reasons behind an increase in EMG amplitude that they observed during a sub-maximal (50%MVC) fatiguing contraction in the triceps of normal and spinal cord injured subjects. Possible reasons they identified for this increase in amplitude were an increase in motor-unit twitch amplitudes, an increase in firing rates, and the additional recruitment of new motor units. On inspection of their data, the authors found that twitch amplitudes varied in a non-systematic way throughout the contractions, and that firing rates did not change. They concluded thus that the increase in EMG amplitude was due to motor unit recruitment. However, in light of this common assumption, Dimitrova and Dimitrov (2003) cautioned against using increased EMG amplitude measures alone as an indicator of recruitment during a fatiguing contraction. These authors reported that the amplitude of the sEMG signal could increase with fatigue due to mechanisms other than recruitment, such as alterations to the width and other characteristics of the MUAP.

In addition to EMG amplitude changes, a number of authors have suggested other indirect evidence of recruitment in the form of central nervous system mediated decreases in MU firing thresholds (Christova & Kossev, 2001; Jensen et al., 2000). Christova and Kossev (2001), for example, reported a consistent decrease in the recruitment threshold of high threshold, dormant motor units in the biceps brachii during a sub-maximal fatiguing contraction. These authors suggested that a decreasing recruitment threshold would mean that the recruitment of additional motor units would occur as their respective recruitment thresholds dropped low enough for the motor units to be activated by the current activation stimulus in a constant force contraction.

Rather than rely on indirect methods, a number of studies have measured MU activity directly using indwelling electrodes. Jenson, Pilegaard and Sjogaard (2000), for example, examined recruitment in the supraspinatus muscle of eight normal female subjects during a fatiguing sub-maximal contraction (11-12% of MVC). As with earlier studies, they surmised that evidence for recruitment was seen in the 38% increase in surface EMG amplitude seen during the contraction. However, their results appear contradictory: while the authors reported a 38% increase in EMG

amplitude, they reported an increase of only 5% in the number of active motor units detected by the indwelling electrodes from the beginning of the contraction to the end of the contraction. While this indicates some recruitment did occur, it also reinforces that other mechanisms are also responsible for the increases in EMG amplitude measures seen with fatigue.

Christova et al. (2001) have also used indwelling electrodes to report observations of recruitment during a fatiguing muscle contraction. The authors monitored motor unit behaviour during a ramped isometric fatiguing task (50% MVC) utilising the biceps brachii (n = 7). Sixty-three motor units were observed in total. At the beginning of the muscle contractions, 40 motor units were active immediately across the seven subjects. As the contraction progressed to fatigue, a further 23 MUs across the subjects became active. Closer examination of these findings question the conclusion due to the small subject numbers, low number of motor units monitored, and the conclusion being based on grouped, rather than individual motor unit data.

Further evidence of MU recruitment with fatiguing contractions is provided by Gazzoni et al. (2001). This group of authors used high spatial resolution sEMG, utilising multiple electrodes in a linear arrangement, to identify MUs non-invasively during low-level contractions. During low-level sub-maximal (5, 10 or 15% MVC) contractions of the biceps brachii (n=5) sustained for 10 minutes, the authors observed evidence of additional motor unit activation. Using a classification system involving neural networking techniques, Gazzoni and co-workers were able to identify individual motor units at these low levels of contraction. The ability to identify individual motor units allowed them to discern newly recruited motor units as they became active.

Synchronisation

An increase in MU synchronisation has also been suggested as a central mechanism involved in the sEMG frequency shift seen in constant force fatiguing contractions (Blinowska, Verroust, & Cannet, 1979; Lippold, Redfearn, & Vuco, 1957). Synchronisation is defined as the tendency of a motor unit to discharge at the same time as other motor units (C. De Luca, 1984). Synchronisation is caused by inputs that are common to two or more motor units that make up a motor unit pool (Kleine, Stegeman, Mund, & Anders, 2001). This input may be descending (from the central

nervous system) or ascending (from peripheral muscle sensory fibres) (Kleine et al., 2001; Young & Hagbarth, 1980). During a fatiguing muscle contraction it is thought that synchronisation, along with an increase in cortical drive, may serve to maintain the force of a contraction (Kleine et al., 2001). Cross-correlation is one method that has been used to assess for motor unit synchronisation during a fatiguing contraction. Synchronisation reflects a higher uniformity of the sEMG signal, and thus a higher cross-correlation value. Using this method, an early study by Lippold et al. (1957) reported weak but consistent cross-correlation in the sEMG signal at approximately nine hertz, with this cross-correlation becoming more evident as the muscle fatigued. In contrast, using the same method De Luca et al. (1982) found only minimal evidence of cross-correlation, and additionally they found that any significant cross-correlation they observed was not time dependent. This, they concluded, indicated that synchronisation did not occur consistently during a fatiguing contraction. Despite this comment, other experimental studies have attributed significant changes in the sEMG power spectrum to synchronisation during low-level muscle contractions. Christensen and Fuglsang-Frederiksen (1988), for example, recorded EMG signals from the quadriceps muscles (rectus femoris, vastus medialis and vastus lateralis) during fatiguing leg extensions at 10%MVC sustained for 60 minutes. They hypothesised that a decrease in MDF seen in each muscle was due to a decrease in average muscle fibre conduction velocity, and an increase in MU synchronisation. A subsequent study by Krogh-Lund and Jorgensen (1992) looked at fatigue in the biceps brachii (n=11) during sub-maximal (15%) isometric contractions. These authors also surmised that MU synchronisation was responsible for a significant portion (up to 65%) of the sEMG frequency shift observed with fatigue. Later work by De Luc, Roy and Erim (1993) in one sense supports these authors, in that they demonstrate the occurrence of synchronisation in a number of different muscles during fatiguing contractions. However, they reported that significant synchronisation only occurred in approximately 1% of motor units examined. Though further modelling studies have demonstrated that synchronisation of motor units would have a significant effect on the sEMG power spectrum (Farina, Fattorini, Felici, & Filligoi, 2002; Kleine et al., 2001), empirical evidence of significant synchronisation occurring during a fatiguing contraction could not be found to further support these reports. This, and the work of De Luca et al. (1993) questions the role of synchronisation in any changes in the sEMG power spectrum that occur during fatiguing contractions.

2.6.1.3 Summary

It is clear that peripherally mediated changes in the MUAP, such as a slowing of muscle fibre conduction velocity and reduced membrane excitability, along with differences in muscle fibre proportions have been credited with fatigue-induced alterations in the sEMG power spectrum. Although less obviously, central mechanisms of muscle control, such as firing rate modulation, recruitment and synchronisation of motor units, have also been linked to the changes seen in the sEMG signal with fatigue. While these central mechanisms of muscle control may influence the sEMG power spectrum during fatiguing contractions to some degree, their role appears to be secondary to that of peripheral mechanisms. It is these peripheral mechanisms that appear to have the greatest influence on the sEMG spectrum during a fatiguing muscle contraction.

2.6.2 GEOMETRIC AND ANATOMICAL FACTORS INFLUENCING THE sEMG SIGNAL

As well as being affected by peripheral muscle and central control factors, the sEMG signal is also affected by a number of other factors, termed geometric and anatomical by Merletti et al. (2001). These include electrode location, electrode orientation, signal stability, and various sources of filtering including tissue thickness and inter-electrode distance. While these factors have important effects on the sEMG signal, the majority of them remain constant within an individual during signal collection if appropriate measures are taken.

2.6.2.1 Electrode location and orientation

The location of electrodes and their alignment with respect to the underlying muscle fibres influence the sEMG signal, evidenced by changes in sEMG temporal and frequency parameters due to changes in electrode position and alignment. With respect to alignment, modelling by Merletti et al. (2001) and Farina et al. (2002) has demonstrated notable changes in spectral parameters when electrodes were moved 5° and 10° away from the longitudinal axis of the muscle fibres being examined. An empirical study by Farina (2003) also showed how displacement of electrodes by 5mm, either by skin, electrode or muscle movement, altered EMG parameters by up to 20%.

Hogrel, Duchene and Marini (1998) have also investigated the effect that the position of the electrodes in relation to the tendon and innervation zone has on the sEMG signal. Using a linear array consisting of 15 electrodes, the authors demonstrated that the electrode pair mid-way between the distal tendon junction and the innervation zone of a muscle provided the most stable sEMG signal. The most stable signal was shown to be that with the lowest MPF and the highest RMS values. Further to the MPF and RMS values, the best electrode *pair* was shown to be that from which the highest cross-correlation co-efficient was calculated, in conjunction with physiological conduction velocity values. The authors also demonstrated graphically the significant increases in the variance seen in measures at electrode pairs close to the innervation zone, or a muscle-tendon junction (Hogrel et al., 1998).

2.6.2.2 Signal stability

Signal stability is recognised as a key issue in sEMG fatigue analysis (De Luca, 1997). The stability of the sEMG signal is affected by a number of factors. For example, movement of the electrode in relation to the muscle alters the population of motor units active beneath the electrode, as well as changing the position of the electrode in relation to tendon and innervation zones, and also in some circumstances the alignment of the electrodes with the fibres of the muscle of interest (De Luca, 1997). Changes in the length of the muscle, as occur during dynamic contractions, lead to alterations in conduction velocity (Arendt-Nielsen et al., 1992), but also to alterations in the number of active muscle fibres present under the collection electrodes due to the bunching or elongation of fibres that occurs with shortening and lengthening (De Luca, 1997). Any of these changes in the electrode-muscle relationship can thus change the characteristics of the recorded sEMG signal. Similarly, changes in the intensity of a muscle contraction can change the characteristics of the sEMG signal by altering the number and type of motor units active beneath an electrode due to the principle of recruitment and de-recruitment with varying load (Henneman & Olson, 1965). It is for these reasons that sEMG signals are most commonly collected during constant force isometric muscle contractions, during which movement between electrodes and muscle, as well as changes in contraction intensity are minimised.

2.6.2.3 Filtering

Surface electromyography signals are also affected by filtering. For example, the tissue type, and the amount of that tissue between the motor units and the recording electrodes, acts as a low pass filter (Hogrel et al., 1998; Merletti et al., 2001). It is known that the more tissue there is between an active MU and the electrodes, then the greater the reduction in the frequency content of the sEMG signal, an effect that is manifested in the sEMG power spectrum (De la Barrera & Milner, 1994). Further filtering as a function of inter-electrode distance has been shown by early mathematical models to affect the sEMG signal frequency content (De Luca, 1979; 1984). In an early empirical study, Moritani and Muro (1987) supported this theory in a study investigating the changes in MDF with changes in muscle force production in the biceps brachii. Using bipolar electrodes with a small (6mm) inter-electrode distance, the authors demonstrated an increase in MDF with an increase in muscle force, a phenomenon consistent with the well recognised size principle of motor unit recruitment (Henneman & Olson, 1965). In the same experiment, the authors also demonstrated that a sEMG signal collected with large inter-electrode distances (40mm) did not allow the detection of MDF changes during the same ramped force contractions. In a more recent study, Farina, Cescon and Merletti (2002) specifically examined the effect of inter-electrode distances on sEMG parameters. Small (5-10mm) inter-electrode distances were shown to increase the sensitivity of EMG parameters to changes in electrode location and alignment. Furthermore, the smaller inter-electrode distance offered a broader frequency spectrum, which, in turn, provided greater sensitivity to changes in the sEMG frequency parameters, similar to what was observed by Moritani and Muro (1987). From a practical point of view, the smaller inter-electrode distances also offered the ability to fit between the tendon and motor point of most muscles more easily.

2.6.3 SURFACE ELECTROMYOGRAPHY COLLECTION TECHNIQUES

There is a number of collection techniques that can be utilised in muscle fatigue analysis. Limitations exist with these techniques in the ability to extract meaningful and consistent information from the sEMG signal. The influence of some of these limitations has been reduced through the evolution of sEMG technology.

Traditional bipolar sEMG is limited by what is called poor spatial resolution. Spatial resolution of sEMG refers to its ability to detect localised muscle activity. This limitation in traditional bipolar techniques occurs because relatively large collection surfaces and inter-electrode distances are used in order to detect the very low voltages emitted by muscles during contractions. The signal resulting from this collection method is thus the spatial and temporal summation of the electrical activity from many motor units, both near to and distant from the collection sites (De Luca, 1997; Merletti et al., 2003). A proportion of that collected signal may also be from cross-talk generated by other muscles, such that De Luca and Merletti (1988) have demonstrated that up to 16% of the sEMG signal of the quadriceps may be from neighbouring muscles. Therefore, traditional sEMG signals provide only general information, and thus general conclusions, relating to muscle function.

A significant advance in sEMG technology that has improved spatial resolution has been the use of active electrodes. Active electrodes amplify the sEMG signal at the collection site, allowing the size of the electrode collection surfaces and inter-electrode distances to be reduced significantly. Significant benefits associated with active electrodes include the collection of a wider frequency bandwidth (Farina, Cescon et al., 2002), and a reduction in cross-talk from neighbouring muscles due to a narrower inter-electrode distance (De Luca, 1997). The application of spatial filters to active electrodes further enhances the signal quality and improves spatial resolution (Broman, Bilotto et al., 1985b; Disselhorst-Klug, Bahm, Ramaekers, Trachterna, & Rau, 2000). These spatial filters behave as high pass filters, which dampen signals from distant motor units of reduced frequency (due to tissue filtering), while the higher frequency signals from motor units closer to the electrodes (less affected by tissue filtering) are amplified. Therefore, the activity of motor units closer to the electrodes are distinguished from those more distant, including those from other muscles (Disselhorst-Klug et al., 2000).

A further development in electrodes has been the use of a linear array. In a very early example of electrode arrays, Masuda, Miyano and Sadoyama (1983) applied 15 active electrodes arranged in a linear fashion to the biceps brachii muscle. Using this electrode array, the authors were able to demonstrate the propagation of motor unit action potentials along a muscle. This allowed the researchers to increase the amount

and specificity of information from individual muscles. In this case they could investigate the anatomical properties of the biceps muscle, such as fibre direction and innervation zones. Innervation points were depicted on the resulting 15 channel EMG trace by the reversal of the MUAP polarity between two adjacent channels signifying a change in propagation direction away from that point**.** In subsequent studies the same authors (Masuda & Sadoyama, 1986; Sadoyama, Masuda, & Miyano, 1985) also demonstrated more direct measurements of muscle fibre conduction velocities, using these linear electrode arrays, where previous measures had required more indirect or invasive techniques (Broman, Bilotto et al., 1985b).

Guidelines have since been developed for the standardised placement of electrodes by the SENIAM project (Surface Electromyography for the Non-Invasive Assessment of Muscles). However, studies have shown variability in individual innervation zones (Merletti et al., 2001), and muscle fibre directions (Fukunaga, Kawakami, Kuno, Funato, & Fukashiro, 1997; Wickiewicz, Roy, Powell, & Edgerton, 1983) such that standardised points may have questionable validity. The development and refinement of linear arrays provides the ability to customise electrode placements and orientation, thus improving the quality and quantity of information extracted from EMG recordings.

The evolution of linear arrays has also included the development of two-dimensional electrode arrays that use a Laplace filter function (Huppertz, Disselhorst-Klug, Silny, Rau, & Heimann, 1997). The two-dimensional Laplace filter is based on the weighted sum of five small (mm diameters) electrodes arranged in a crosswise pattern. This arrangement can be replicated to create a linear array utilising the Laplace set-up The central electrode in the array is weighted by a factor of –4, while the peripheral electrodes are weighted at +1 (Figure 2.2). The two dimensional spatial filtering of this array significantly reduces signals from distant sources in all directions, while the weighting further amplifies the signals from motor units near the collection site, as well as producing a distinctive triphasic peak as single, local MUAPs pass beneath the electrode pins (Figure 2.2) (Huppertz et al., 1997). While still allowing the calculation of traditional sEMG frequency and amplitude parameters, conduction velocity, and the correct orientation and placement of the electrode, the resulting spatial resolution also allows the detection of single MUAPs, and thus the study of the behaviour of

individual motor units. This is an option that usually requires invasive EMG techniques (Disselhorst-Klug et al., 2000; Hogrel, 2003; Merletti et al., 2001).

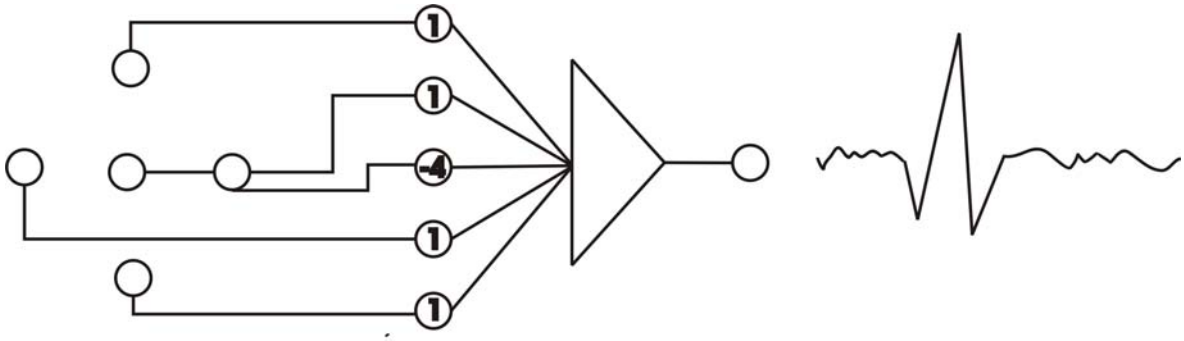

Figure 2.2. Two-dimensional Laplacian Filter

Schematic diagram of the two-dimensional Laplace filter arrangement showing the weighting of the peripheral and central pins, and the resulting signal that shows the tri-phasic peak indicating activity from a single motor unit. Figure reproduced with permission from Disselhorst-Klug et al. (2000).

2.7 SEMG AND PREDICTION OF ENDURANCE TIME.

The performance of endurance tests in general requires maximal effort, and is by nature exhausting. Due to the sustained effort required, the results may be influenced by factors such as subject motivation and pain. For these reasons, it would be desirable to be able to estimate endurance time, or quantify fatigue without requiring a subject to perform a test until failure. As covered in an earlier section, an appreciation of the fatigue process can be gained from the changes observed in the sEMG signal during a fatiguing task. Furthermore, these changes appear to be due predominantly to peripheral mechanisms. Hence a number of studies have examined the ability to predict endurance time in limb muscles based on the rates of change in sEMG parameters. Early work by Badier et al. (1993), for example examined this concept using sEMG data collected from thumb and quadriceps muscles during isometric

contractions at intensities between 20% and 80% of MVC. The results from this study indicated that changes in the relative power in low (0-50Hz) and high (80-400Hz) parts of the sEMG power spectrum, when calculated across the entire contraction, were significantly correlated ($p < 0.05$) with the endurance times. No indication of the strength of the relationship was given. Later work by Merletti and Roy (1995) further investigated prediction of endurance times using the tibialis anterior muscle of six healthy subjects. Signals were collected during an isometric fatiguing contraction at 50%, 60%, 70% and 80% of MVC, with MDF as their sEMG parameter of interest. The study protocol required subjects to contract for 170, 150, 120 and 90 seconds respectively, regardless of whether or not they could maintain the contraction target. In addition to assessing correlations across the entire test, Merletti and Roy also examined how changes in MDF in the initial thirty seconds of a muscle contraction correlated with endurance time. The authors also compared two regression curves for their ability to model the changes observed in the sEMG parameters measured over time. It was reported that an exponential model best described the decline in MDF over the entire contraction time. However, the authors found that this model was inappropriate for prediction of endurance times based on only the first 30 seconds of sEMG data. In contrast, a linear model calculated over the first 30 seconds correlated highly ($R = 0.90$) with endurance time at the four levels of contraction tested. Mannion and Dolan (1996) also reported high correlations between endurance time and the linear slope of MDF, but this time for the vastus lateralis ($R = 0.91$) and rectus femoris ($R = 0.96$) muscles of ten healthy subjects. These authors also reported that this relationship was consistent at contraction levels between 20%-60%MVC. In addition to the 50%-80% in Merletti's and Roy's work, these findings suggest that prediction of endurance time using sEMG variables may be possible when collected from a broad range of sub-maximal isometric contractions.

Less favourable results using traditional sEMG parameters were found in a more recent study by Maisetti et al (2002). Using vastus medialis and vastus lateralis, these authors collected sEMG data during a sub-maximal (50%MVC) fatiguing contraction held to exhaustion. Using a linear model to describe the changes seen in sEMG parameters, they reported moderate correlations from VM and VL between the change in RMS ($R = 0.75, 0.71$), MPF ($R = 0.63, 0.60$) and CV (0.63, 0.54) and endurance time over the whole contraction. However, using only the first fifteen and thirty seconds of sEMG data, correlations fell below 0.35, indicating a poor ability to

predict endurance time using these parameters calculated from a short duration contraction. In addition to calculating correlations between the slopes of the sEMG parameters and endurance time, Maisetti and co-workers also examined correlations with endurance times of an index termed the *area ratio* (figure 2.3) (Merletti, Lo Conte, & Orizio, 1991) calculated from each parameter after 15 and 30 seconds. In contrast to the slope correlations for the short durations contractions, the authors reported high correlations with endurance time for area ratios calculated from FB1 after fifteen (r = 0.82) and thirty seconds (r = 0.82). These results suggested that the area ratio measure may improve the accuracy of endurance time prediction when using sEMG.

2.8 RELIABILITY

In general the reliability of parameters generated from a sEMG signal is recognised as poor. A number of studies have examined the reliability of these parameters measured from the quadriceps using a range of collection techniques.

Kollmitzer, Ebenbichler and Kopf (1999), for example, examined RMS and MDF in the three superficial quadriceps muscles (rectus femoris, vastus medialis, vastus lateralis) (N = 18). Kollmitzer and colleagues investigated the reliability of these measures within a session (3 minutes apart), between sessions on the same day (90-minutes apart), and between days (6 weeks apart). Measures were calculated for brief (5 secs) maximal and sub-maximal (50%MVC) voluntary contractions, as well as the rate of change of each parameter (slope) during a sub-maximal (50%MVC) contraction sustained to failure. Initial RMS values for vastus medialis were shown to have excellent reliability within a session only for the short duration sub-maximal contraction (ICC = 0.98). Reliability of initial RMS values between sessions was only poor to moderate for the same contraction (ICC = 0.54 at 90 minutes, 0.45 at 6 weeks). The reliability of the change in RMS during the fatiguing contraction was also poor (ICC = 0.34 at 90 minutes, 0.33 at 6 weeks). In comparison to RMS, initial values of MDF were shown to be a more reliable parameter in this study. The authors reported excellent reliability of initial values for a short duration sub-maximal contraction both within a session (ICC = 0.98), and between sessions (90 minutes – ICC = 0.85). However, ICC values for the 6-week re-test were only moderate to poor

(ICC = 0.49). Like RMS, the change in MDF during the fatiguing contraction was also reported to have poor reliability between sessions (ICC = 0.48 at 90-minutes, ICC = 0.14 at 6 weeks). In this study it is significant that only passive electrodes were utilised, which were placed on the quadriceps muscles according to standardised anatomical positions. No account was made for individual variation in muscle fibre angulation or innervation zone position. Slight anatomical variations between individuals, as well as variations in electrode placement within individuals between days may thus have contributed to the generally poor reliability figures reported.

A more advanced sEMG approach was used by Rainoldi, Bulock-Saxton, Cavarretta and Hogan (2001) with respect to the reliability of mean power frequency (MPF) and muscle fibre conduction velocity (CV) measures. The authors used a 16-bar linear electrode array to find the best position and orientation for a four-bar, two double-differential-channel recording electrode. They then examined MPF and CV initial values, and the initial slopes of these parameters (calculated over 15 seconds) during sub-maximal isometric contractions of vastus medialis sustained for 50 seconds. CV was calculated using a cross-correlation function, and the authors accepted cross-correlation values greater than 0.5 if the CV values were within physiological bounds. Using Fisher's test Rainoldi and colleagues reported excellent reliability for the initial values of both MPF ($F = 4.15$, $p<0.05$) and CV ($F = 7.03$, $p<0.05$). As with the work of Kollmitzer et al. (1999), this reliability did not carry over to the changes in these parameters with the fatiguing contraction, with both MPF and CV showing Fisher-values below the 3.0 threshold that indicates a reliable test (MPF, $F = 2.11$; CV, $F = 1.31$). As with the work of Kollmitzer et al. (1999), the reliability figures reported may have been influenced by the methodology used. Despite the use of a linear electrode array to position the collection electrodes for the first session, electrode placement for subsequent testing sessions was done according to ink marks left on the skin. Again, this methodology appears to leave room for measurement error between sessions.

A more recent study by Farina and Merletti (2004) utilised a complex multi-electrode (61 contacts) array, with inter-electrode distances (IEDs) of 5mm to investigate the effect on conduction velocity (CV) estimation reliability of 14 different combinations of IEDs, number of channels used, and electrode positions. Testing was done on the biceps muscle, and involved three 20-second sub-maximal (50%MVC) isometric

contractions per session, on three non-consecutive days. The optimal position of the electrodes was assessed for each session. The authors reported that the initial values of CV showed moderate reliability in general, with ICC values improving with three or more channels used for CV calculation (3 channels, IED 10mm ICC = 0.70). The overall change in CV was reported to have poor reliability for all methods of calculation, with each ICC being below 0.4. In general, the 10mm IED was shown to provide the least variance of CV measures, with variance improving with three or more channels used to calculate CV (values are the mean-SD%(±SE); 2 channels 22.9±2.3; 3 channels 14.1±1.0; 4 channels 7.8±0.5).

With respect to ratio measures within the power spectrum, Dolan et al. (1995) have examined the repeatability of two measures: the relative power in frequency band 1 (5-30Hz); and frequency band 2 (FB2). SEMG data was collected from the erector spinae muscles of ten subjects who maintained a static lifting position at 60%MVC for 46 seconds, and repeated the test one hour later. A further six subjects repeated the test several days later. Within day and between day ICC values for both initial and slope measures of FB1 were all reported as being above 0.9 .

While electrode placement and geometry are significant factors, the variability in sEMG estimates also appears to relate to the level of muscle contraction used. Rainoldi et al. (1999), for example, investigated the effect that different levels of muscle contraction have on variance and reliability of measures including MNF and CV. They found consistently that measures taken at 50% MVC showed less variance, and greater ICC values than those taken at 10%, 30% and 70% MVC (Rainoldi et al., 1999). These results are supported by Kollmitzer et al. (1999) who reported significantly lower variance of MDF at a contraction level of 50% when compared to MVC ($p < 0.05$).

From an analysis perspective, Merletti and co-workers (1991) suggested another method by which the inherent variability of the sEMG signal parameters could be reduced when examining the change in parameters over time. They called this the *area ratio* measure. The area ratio calculation has been applied to the time plot of other sEMG parameters, and can be described according to Figure 2.3. The initial value of the parameter is used to define the top of a reference rectangle. The time point within which the ratio is to be calculated further defines this rectangle. The

ratio is then calculated as the area B divided by the sum of the areas A+B. This ratio measure was shown by Merletti et al. (1991) to be much less sensitive than traditional sEMG measures to noise throughout a signal, while being more sensitive to noise that affects the initial value of parameters. This sensitivity to initial values appears of minor significance in light of evidence that the initial values of all parameters are highly reliable (Kollmitzer et al., 1999; Rainoldi et al., 1999). Expressed over time, the changes in the area ratio of a parameter is also described as regression free, meaning it is without the statistical violations of autocorrelation that affect the regression calculations of "raw" sEMG parameter values.

In order to calculate percentages of maximal voluntary contraction (MVC), a measure of maximum voluntary contraction is required. The repeatability of MVC measures has been shown to be excellent with ICC values consistently greater than 0.8 when the procedure used follows a warm-up period, chooses the best of three trials, and involves verbal encouragement of the subject (Kollmitzer et al., 1999; Rainoldi et al., 2001; Rainoldi et al., 1999). Further evidence of excellent reliability is reported by Kollmitzer et al. (1999). These authors reported excellent reliability in the form of standard error of the mean (SEM), showing within trial, between session and between days SEM measures of MVC ranging from 1.1% to 6.4%.

2.8.1 Summary

Regardless of the method of collection, the reliability of initial values of MNF, MDF, FB1 and CV is reported consistently as good to excellent within the literature regarding quadriceps. It is clear, however, that other sEMG measures, such as rates of change, display poor reliability, particularly between experimental sessions undertaken on different days. With respect to changes with time due to fatigue only the reliability of FB1 has been reported as acceptable for the quadriceps, with the reliability of other parameters being described as moderate at best. Examination of the literature suggests that recent reliability studies could have utilised sEMG protocols more effectively. In light of these studies, and based on the work of authors such as Farina (2001, 2004) and Merletti (2001, 2003), it would appear that the use of linear electrode arrays to optimise electrode location and replacement is an important concept that may result in optimising the accuracy and reliability of each of the sEMG parameters. With specific reference to the estimation of conduction velocity, the use of three or more collection channels appears desirable (Farina, Zagari, Gazzoni, &

Merletti, 2004). It also appears that variance in sEMG measures is reduced during a sub-maximal isometric contraction at 50% of the maximum voluntary contraction.

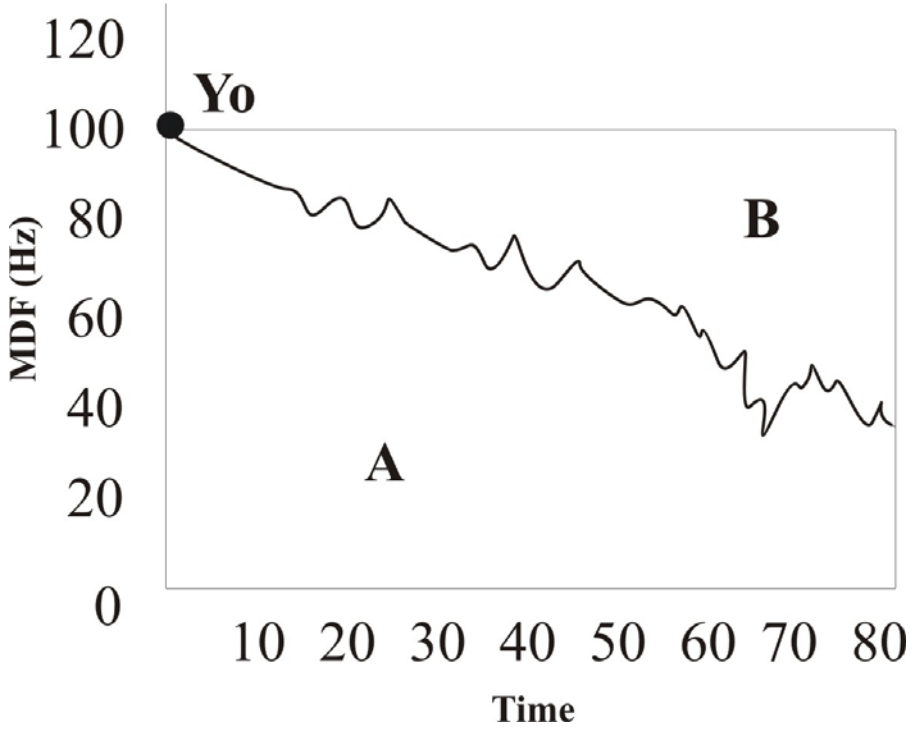

Figure 2.3. Area Ratio Calculation.

Representation of the areas considered for the area ratio calculation. The area (B) above the relationship between the surface electromyography (sEMG) parameter (e.g. median frequency, MDF) and time is divided by the sum of the area (A+B) as a rectangle delineated by the duration of the contraction and the initial value of the sEMG parameter. The value Y_o (filled circle) was computed over the first two seconds of the signal. Figure reproduced with permission (Maisetti et al., 2002a).

3.0 METHODOLOGY

3.1 SELECTION OF SUBJECTS

3.1.1 Subjects with healthy knees

Subjects with healthy knees were recruited via advertising at a local gym, advertising at a local exercise group for people aged over 60, and through word of mouth. Interested subjects were contacted and given an initial telephone screening that covered the following criteria.

Inclusion criteria:

- Two healthy knees

Exclusion criteria:

- Any history of stroke, significant knee injury or diagnosed osteoarthritis
- Knee pain at the time of testing
- Any physical condition that would prevent them participating fully in the experiment.
- Any medical condition that would place them at risk when working maximally and to exhaustion.
- Neurological condition (Parkinsons disease, Multiple Sclerosis).
- Dementia or other severe cognitive dysfunction.
- Inability to provide informed consent.

3.1.2 Subjects with osteoarthritis

Based upon pilot testing, to achieve a power level of 0.80 with an alpha level of 0.05, 26 subjects were required for the current study. Subjects with arthritis were recruited through advertisements in a local newspaper, contact with local general practitioners and physiotherapists, and through the Arthritis Foundation of New Zealand. Potential subjects were screened for the basic inclusion criteria by telephone.

Telephone inclusion criteria

- Unilateral knee osteoarthritis
- X-ray confirmation of the diagnosis

Those who met the criteria were invited to attend a further screening session at the Physical Rehabilitation Research Centre, School of Physiotherapy, Auckland University of Technology, where the following criteria were more rigorously assessed:

Inclusion criteria:

- Positive clinical diagnosis of osteoarthritis in one knee according to the clinical and radiographic algorithm used in the American College of Rheumatology 'Criteria for classification of idiopathic osteoarthritis of the knee' (Altman et al., 1986). (Appendix 1).
- Primary diagnosis of osteoarthritis of one knee, with a Kellgren-Lawrence x-ray score of 3 or greater (Kellgren & Lawrence, 1957)(Appendix 2).

Exclusion criteria:

- Osteoarthritis or injury to both knees
- Concurrent medical condition such as a previous heart attack that may preclude them from exhaustive or maximal physical exertion.
- Neurological condition (Parkinsons disease, Multiple Sclerosis).
- Dementia or other severe cognitive dysfunction.
- Unable to provide informed consent.

During the session subjects read an information document. This document explained the aims, rationale, methodology, potential risks, and potential benefits of the study. Subjects were encouraged to ask questions if they had any, and were assured that they could withdraw from the experiment at any stage, with no fear of repercussions. If the subject met the criteria, and was happy to participate, a consent form was signed and the subject admitted to the trial.

The use of the Kellgren-Lawrence classification, and the inclusion criteria of a score greater than 3 on this radiological scale is consistent with other studies examining similar patient populations with osteoarthritis of the knee (Fisher & Pendergast, 1997; Slemenda et al., 1997).

3.2 PROCEDURE

Subjects were required to attend one familiarisation session, and one testing session. There was no set time lag between these sessions. During the familiarisation session subjects were given an opportunity to experience the testing procedures involved. Based upon pilot testing it was deemed necessary to include a familiarisation session to improve the understanding and compliance of the subjects during testing. At the beginning of the testing session, subjects were asked to complete a WOMAC index (Version VA3.1) for knee osteoarthritis. This is a pathology specific subjective assessment of a patient's pain, stiffness and physical function. Its validity, reliability and responsiveness have been published widely (Bellamy, Buchanan, Goldsmith, Campbell, & Stitt, 1988; Bischoff-Ferrari, VonDechend, Bellamy, & Theiler, 2004; Theiler et al., 2002). The format used in the current study involved visual analogue scales, and a maximum score of 240.

3.2.1 Experimental Set-Up

Subjects were seated in a purpose built chair with their knee flexed to 90°. The chair was designed with no padding to reduce the impact of damping on the twitch interpolation protocol. The chair was adjusted to ensure that the back of the knee rested comfortably against the edge of the seat. Once comfortable, the subject was secured to the chair using a lap and chest strap. The lower leg, minus socks or other clothing, was strapped to a metal attachment in series with a strain gauge at the ankle level, just proximal to the lateral malleolus. Comfort was ensured by using a heat-moulded thermoplastic brace to secure the leg. This experimental set-up was similar to that used by Maisetti et al (2002).

Force measurements: a PST model, 250kg-maximum strain gauge (Precision Transducers Ltd, 7 Market Place, Glenfield, Auckland, New Zealand) was used to measure maximum voluntary contraction (MVC), and to provide a measure of 25%, 50% and 75% of that MVC during the twitch interpolation protocol, and 50% again during the endurance test. Force readings were in newtons (N), and were collected from the strain gauge via a custom-made amplifier by an Apple G4 personal computer sampling at 2000Hz. A real-time force trace was displayed on a computer monitor using a customised software program (Superscope II 3.0 GW Instruments,

Washington, USA). This provided visual feedback of the force being generated by the subjects during both the twitch interpolation protocol and the endurance test.

3.2.2 Warm-Up

Before being seated in the chair, subjects undertook a 5-minute warm-up on a stationary cycle at a self-determined level of light exertion. While seated in the chair prior to testing of maximum voluntary contraction (MVC), subjects performed an isometric warm-up protocol to help with familiarisation and to further reduce the chance of injury. This procedure involved five initial sub-maximal contractions, each held for five seconds, with a 10 second rest between contractions. The level of these contractions was self-selected, and increased with each contraction. Finally, the subject performed ten short, rapid contractions to self-selected levels. Subjects were asked if the warm-up was deemed sufficient. If the subject felt the need for additional warm-up exercise, a second set of isometric contractions was performed. The subjects then rested for 3 minutes before beginning maximum voluntary force testing.

3.2.3 Maximum Voluntary Contraction

The maximum voluntary contraction was defined as the highest force achieved by the subject over three trials, separated by a 2-minute rest (Maisetti et al., 2002b). The subject was asked to contract their quadriceps maximally, during which they received standardised verbal encouragement to facilitate this contraction (McNair et al, 1996, Barratta et al, 1999).

3.2.4 Interpolated Twitch Protocol

Figure 3.1 shows the physical set-up for the interpolated twitch procedure. A constant current stimulator (DS7A, Digitimer Ltd, Welwyn Garden City, England) delivered doublet pulses (10ms separation) to the quadriceps via 10cm x 10cm Dura-stick™ self-adhesive electrodes (Chattanooga Group, Inc. 4717 Adams Road, Hixson, Tennessee). The pulses were delivered at 300V, with a pulse width ranging from 100-500μs, and a current ranging from 80mA to 500mA (Allen, Gandevia, & McKenzie, 1995; Behm, St-Pierre, & Perez, 1996; Folland, Emsley, Martin, & Jones, 2000; Norregaard, Lykkegaard, Bulow, & Danneskiold-Samsoe, 1997; Rutherford, Jones, & Newham, 1986). The variation in stimulation parameters was utilised to gain a muscle contraction of at least 25% of a subject's MVC. The delivery of the pulse was controlled by the researcher via an Apple IIVx personal computer using a customised

software program to drive the stimulator (Superscope II 3.0 GW Instruments, Washington, USA).

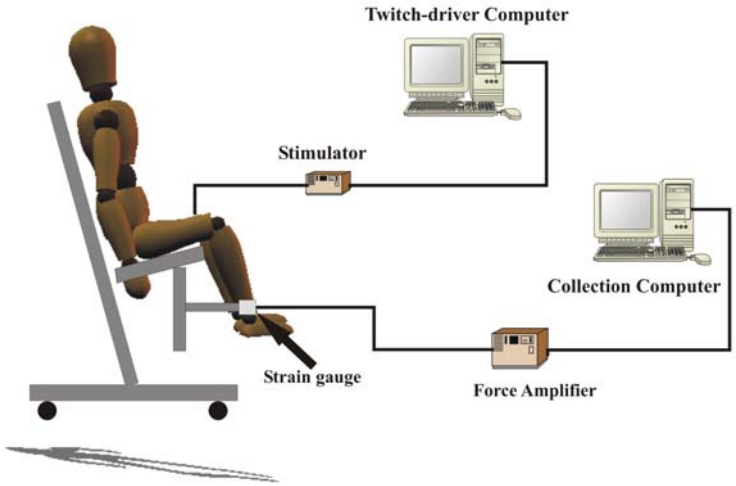

Figure 3.1. Interpolated Twitch Physical Set-up

Interpolated twitch experimental set-up used in the current study. The stimulating electrodes are attached proximally and distally to the subject's right thigh.

During the interpolated twitch protocol subjects were asked to perform six contractions: one each at 25%, 50% and 75% of the initial MVC, followed by three further maximal contractions. Sub-maximal contractions were maintained at the required force level until three pulses had been delivered. Maximal muscle contractions were maintained until a single pulse was delivered. Subjects rested for three minutes between each muscle contraction. Following the last voluntary contraction, three pulses, each separated by 10 seconds, were delivered with the subject at rest. The starting value (baseline) and the absolute size of the twitches were recorded. The values at each contraction level were averaged. The twitch sizes were normalised to the twitch delivered with the subject at rest. Based on a non-linear model-of-best-fit for the interpolated twitch data, the voluntary activation deficit for the quadriceps was calculated using the intercept on the x-axis of the graph of Normalised Twitch Amplitude versus Percentage of Voluntary Contraction. True maximum force was then calculated by adjusting the maximum voluntary contraction level for the activation deficit measured. This procedure is similar to that used by Behm et al (1996) and Norregard et al (1997). If a non-linear model did not intercept the x-axis, the calculation was made using a linear model including only the resting

and MVC interpolated twitch values (Rutherford & Jones, 1988). Previous studies have used a greater number of sub-maximal contractions in their interpolated twitch protocols (Berth, Urbach, & Awiszus, 2002; Machner, Pap, & Awiszus, 2002; Norregaard et al., 1997; Rutherford et al., 1986). Pilot testing found that fewer contractions reduced the effect of fatigue throughout the testing, and did not reduce the precision of the best-fit models.

3.2.5 Endurance Test

The subjects rested for an arbitrary period of 20 minutes following the interpolated twitch procedure before completing the endurance test. In this test, the subjects were asked to maintain a sub-maximal contraction (50% True maximum force – TMF) until exhaustion. Exhaustion was defined as when the subject could no longer hold with-in 5% of the 50% TMF target (Maisetti et al., 2002b), as demonstrated by visual feedback on the computer monitor. This exhaustion point was designated as T_{Lim}. Subjects were encouraged prior to and during the test using standardised verbal encouragement. Subjects were not informed of the results of previous subjects, or of any objective performance expectations of the experimenter. While undergoing the endurance test, surface electromyography data was collected from the vastus medialis muscle.

3.2.6 Electromyography

Skin preparation: The procedure recommended by De Luca (1997) was followed to ensure skin impedance was below 10kΩ. First, the electrode sites were shaved. The shaved area was then rubbed with an abrasive paste (Omni-Prep®, D.O. Weaver & Co., 565-C Nucla Way, Aurora, Colorado 80011 USA) until the skin was red, then cleaned with alcohol wipes. Finally, a small amount of conductive gel was wiped onto the shaved area. This was shown during pilot testing to improve the quality of the signal from the laplacian electrode.

Electrodes: Monopolar silver/silver-chloride (Ag/AgCl) passive electrodes (Red Dot, 3M Health Care, St. Paul MN, USA) were used for grounding purposes. Data collection was via an eleven-pin (1mm diameter) two-dimensional high-spatial resolution (HSR) laplacian electrode (Université de Technologie, Compiègne, France) with an inter-electrode distance of 10mm (figure 3.2).

Figure 3.2. Laplacian Electrode

The eleven-pin, three-channel Laplacian electrode, with amplifier, used for surface electromyography data collection.

Electrode placement: the ground electrode was placed on the bony surface of the anterior tibia approximately 1/3 the way from the tibial tubercle to the talocrural joint. The Laplacian electrode was secured over the vastus medialis muscle at a point at which the signals from at least two of the three recording channels were of similar amplitude, and delayed in time. This was deemed to indicate that the electrode was parallel to the underlying muscle fibres, and measuring mono-directional propagation (Farina et al., 2001; Farina, Zagari et al., 2004). The similarity of the channels was assessed during testing and also off-line by calculating the correlation co-efficient between each signal. This coefficient was required to be higher than 0.7 (Merletti et al., 2001).

***sEMG Data collection*:** Figure 3.3 represents the physical set-up for the sEMG protocol. The sEMG signal was first amplified (gain 100), differentiated, and filtered by the laplacian electrode. The EMG signal was then further amplified, differentiated and filtered (bandwidth 3-1000 Hz) by differential amplifiers (PSII series amplifier, Grass Instruments Company, Quincy Massachusetts, USA). Following analogue to digital conversion (InstruNet® 100B, GWI, Somerville, MA, USA) at a sampling rate of 10kHz, the signals were relayed to a software package (Protags V0.4, ©Jean-Yves Hogrel 1991-2004) operating on a personal computer.

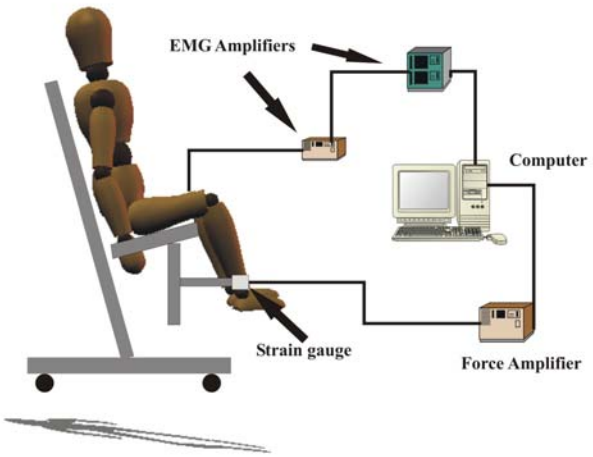

Figure 3.3. sEMG Physical Set-up

Electromyography (EMG) experimental set-up used in the current study. The Laplacian electrode is attached to the subject's right thigh (vastus medialis).

sEMG data analysis: The variables of interest were calculated offline using Protags V4.0 software. These variables were median frequency (MDF); mean power frequency (MPF); root mean square (RMS); the relative power in the signal between 5-30Hz (FB1); conduction velocity (CV); the relative slopes of each sEMG parameter calculated over the initial thirty seconds (T_{30}), and over the entire endurance test (T_{Lim}); initial and end values for each parameter; percentage change in each parameter over the entire endurance test. The means from the first and final two seconds of sEMG data were used as the initial and end parameter values, respectively. These initial and end values were also used in slope calculations. Slopes were expressed as the slope of the linear regression of normalised parameter versus absolute time, measured in percentage change per second (%/s). Percentage changes were calculated by expressing the end value of a parameter as a percentage of the initial value. Percentage change calculations were made using Microsoft Excel® software.

Intra-session reliability of sEMG parameters collected using the current study protocol was assessed during pilot testing. Using the experimental set-up described earlier, subjects contracted at 50% of their maximum voluntary contraction twice for five seconds. Each contraction was separated by a sixty-second period of rest. Surface electromyography signals were collected during the two five-second periods. The sEMG parameters of interest were calculated for each of the three channels of sEMG collected from the laplacian electrode in each period, and compared. A summary of results from this pilot study can be seen in appendix 3. In summary, the parameters MDF, MPF, RMS and CV were shown to have good reliability with all ICC values above 0.74, and most above 0.90 for all three channels of sEMG data. The lower confidence intervals for these parameters were above 0.67, with most above 0.80. The FB1 parameter demonstrated the poorest reliability, with acceptable ICC values between 0.74 and 0.83, but lower confidence intervals of between 0.36 and 0.57.

Also during pilot testing, time series sEMG-parameter data collected during fatigue tests were assessed using three curve fitting models: polynomial first order (linear), polynomial second order (non-linear) and exponential. Curve fitting was done in order to assess for patterns of fatigue that may have differentiated the subject groups. It was apparent that the changes in the sEMG parameters measured were not consistently linear over the duration of the fatiguing contractions. However, curve fit analysis showed that a linear model approximated the sEMG changes accurately over the first

thirty seconds of data than over the entire contraction. Thus it was thought that a linear model over the first 30 seconds of data may highlight any differences that were present in the rates of change of the sEMG parameters between the affected, unaffected and control legs. The use of a linear model to describe the changes in the sEMG parameters was consistent with Merletti and Roy (1995), Mannion and Dolan (1996) and Maisetti et al (2002).

2.3 STATISTICAL ANALYSES

Firstly, all data was checked for outliers and normality to ensure the appropriate use of parametric tests. Using t-tests, comparisons of age and body mass index were then undertaken to identify any differences in the two groups (OA and Non OA subjects).

With respect to initial and end values of the sEMG parameters, a two factor ANOVA (groups and time) with repeated measures on time was undertaken. Any main affects were investigated using paired (across legs) and un-paired t-tests (across the unaffected leg in the OA group and the control group leg).

For the variables maximal voluntary contraction, true maximal force, voluntary activation and endurance time, paired t-tests were used for the assessment of differences between the affected and unaffected legs of the group with knee osteoarthritis, while unpaired t-tests were used for comparisons between the unaffected and control legs.

Surface EMG variables measured over time were normalised to the initial value of the relevant parameter, and slope was calculated using linear regression. Thereafter, paired t-tests were used for comparisons of percentage change and slopes between the affected and unaffected legs of the group with knee osteoarthritis, while unpaired t-tests were used for comparisons between the unaffected and control legs.

Correlations were calculated between endurance times, and the relative slopes and area ratios for each parameter over the full duration of the endurance test in each group. Parameters were also assessed for correlations after the first thirty seconds of the test to assess whether endurance time could be predicted from sEMG data.

Alpha levels were set at 0.05. Where appropriate, to decrease the probability of a type 1 error, a sharpened Bonferroni correction was used to adjust alpha-levels for the rejection or acceptance of null hypotheses (Ottenbacher, 1991).

The above statistical tests were undertaken using the Statistical Package for Social Sciences V11 (SPSS Inc., Chicago, IL, USA), and GraphPad Prism® V4 software (GraphPad Software Inc, San Diego, California, USA).

4.0 RESULTS

4.1 INTRODUCTION

This chapter is divided into six main sections. The initial sections describe the data pertaining to the subjects, maximum voluntary contractions, true maximum forces, voluntary activation, and endurance times. The final sections cover the results related to surface electromyography. This includes a description of the initial and end values, percentage changes and rates of change, concluding with the findings relating to the prediction of endurance times.

4.1.1 Subjects

Data were collected from 26 subjects with unilateral knee osteoarthritis. This group included 15 males and 11 females aged between 35 and 78 (mean 63.6, SD 12.51). Details are presented in table 4.1. Kellgren-Lawrence x-ray scores were all greater than two. Data was also collected from 17 subjects with no known knee pathology. This group included 9 males and 8 females aged between 35 and 74 (mean 64.69, SD 9.52). Details are presented in table 4.2. There were no significant differences in age, or body mass indices of the group with knee osteoarthritis compared to the control group ($p > 0.05$).

Table 4.1. Osteoarthritis Subject Information

Details of subjects with unilateral knee osteoarthritis. BMI = body mass index. WOMAC = Western Ontario McMaster University Index, a pathology-specific subjective assessment for knee osteoarthritis.

Subjects	Number		Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m^2)	WOMAC
Male	15	Mean	64.5	171.1	75.6	25.7	
		SD	12.1	4.5	12.4	3.0	
Female	11	Mean	62.2	157.4	72	28.4	
		SD	14.7	4.5	9.6	4.4	
Overall	26	Mean	63.6	164.3	73.8	27.1	51.85
		SD	12.5	8.3	10.8	3.9	48.44

Table 4.2. Control Subject Information

Details of control subjects. BMI = body mass index. WOMAC = Western Ontario McMaster University Index, a pathology-specific subjective assessment for knee osteoarthritis.

Subjects	Number		Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m^2)	WOMAC
Male	9	Mean	66.9	179.1	75.5	23.5	0
		SD	7.4	6.3	8.2	1.2	0
Female	8	Mean	61.9	162.9	64	22.5	0
		SD	11.1	3.7	6.8	1.2	0
Overall	17	Mean	64.7	171	73.8	27.1	0
		SD	9.53	9.9	10.8	3.9	0

4.2 MAXIMUM VOLUNTARY CONTRACTIONS

Figure 4.1 shows the group means and standard deviations for maximum voluntary contractions. The affected legs generated maximum voluntary force of 436N, (SD± 159), the unaffected legs 505N (SD±154), and the control legs 556N (SD±173). Analysis showed a significant difference ($p < 0.05$) between the affected and unaffected legs of the osteoarthritis group. There was no significant difference between the unaffected leg and the control group.

4.3 MUSCLE ACTIVATION DEFICITS

Figure 4.2 shows the group means and standard deviations of the activation deficits for each leg. No significant differences ($p > 0.05$) were seen between the unaffected and affected legs. However, the unaffected leg of the osteoarthritis group (mean 8.2%, SD±7.2) had a significantly greater ($p < 0.05$) activation deficit than the control leg (mean 1%, SD±2.1%).

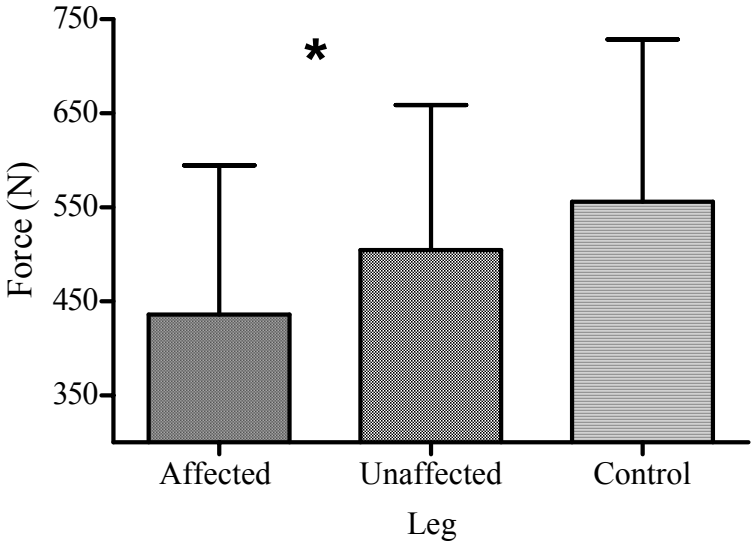

Figure 4.1. MVC Data

Means and standard deviations of maximum voluntary contractions (MVC) for the affected, unaffected and control legs. Force was measured in Newtons (N). *Significant difference in MVC between the affected and unaffected legs ($p<0.05$).

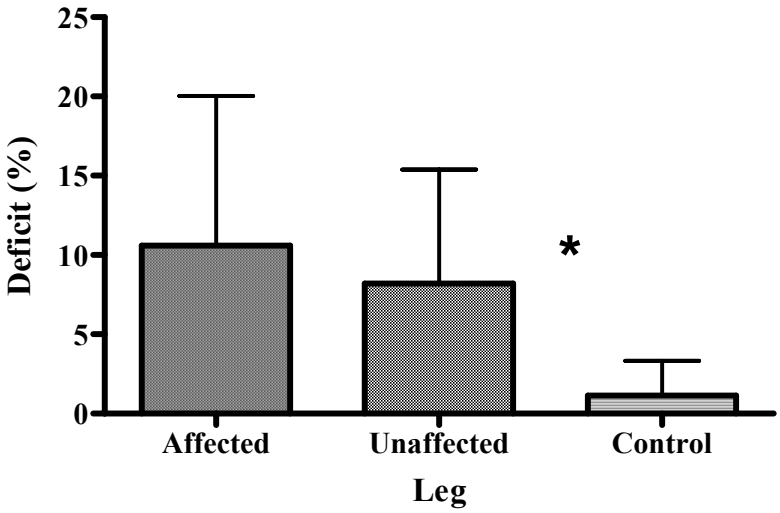

Figure 4.2. Activation Deficit Data

Means and standard deviations of the deficits in voluntary activation for the affected, unaffected and control legs. *Significant difference between the unaffected and control legs ($p<0.05$).

4.4 TRUE MAXIMUM FORCE

True maximum force refers to the figure generated when adjusting the maximum voluntary contraction with any activation deficit detected. Calculation of this figure is shown in appendix 4. Figure 4.3 shows the group means and standard deviations for the estimated true maximum force (TMF) capability of the quadriceps. Similarly to the maximum voluntary contraction data, the results indicate that the true maximum force capacity of the affected leg (491N, SD±177) was significantly lower ($p < 0.05$) than for the unaffected leg (551N, SD±172). There was no significant difference between the unaffected leg and the control leg (555N, SD±178).

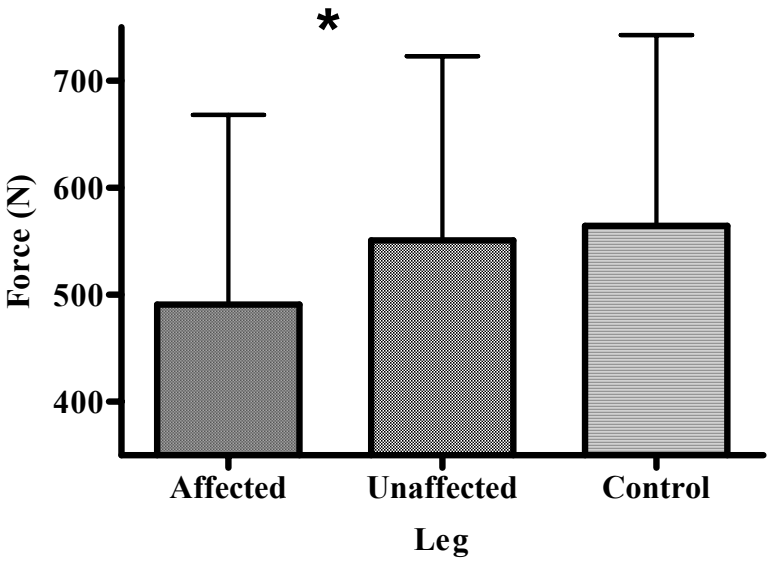

Figure 4.3. TMF Data.

Means and standard deviations of True Maximum Force (TMF) for the affected, unaffected and control legs. Force was measured in Newtons (N). *Significant difference in TMF between the affected and unaffected legs ($p<0.05$).

4.5 ENDURANCE TIMES

Figure 4.4 shows the group means and standard deviations of endurance times for each leg. No significant differences were observed between the endurance times of the groups. However a trend was observed, in that endurance times were higher in the affected (83s, SD 31, range 26-135s) and unaffected legs (79s, SD 33, 28-136s) compared to the control group (67s, SD 23, range 32-105s).

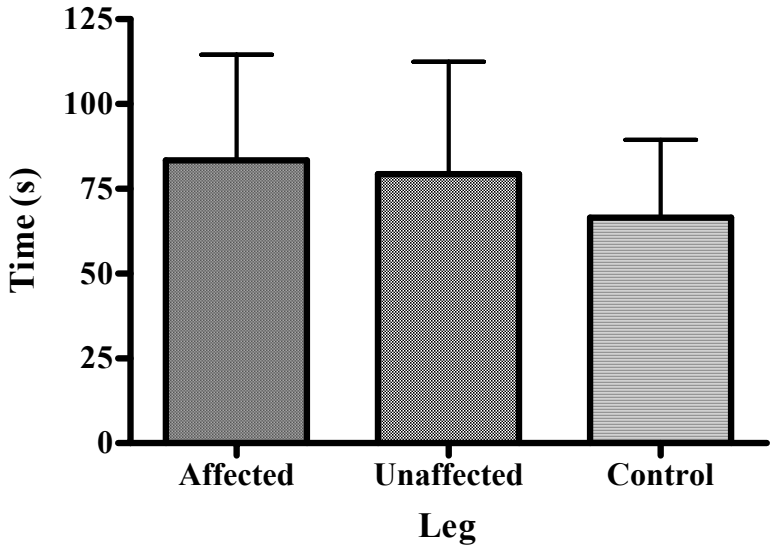

Figure 4.4. Endurance Time Data.

Means and standard deviations of endurance times for the affected, unaffected and control legs. No significant differences were detected between legs.

4.6 ELECTROMYOGRAPHY

A typical EMG trace collected during the endurance test is displayed in figure 4.5. Electromyographic signals from the affected legs of three subjects, and from the un-affected knees of four subjects with knee osteoarthritis were discarded due to poor signal quality. Data from a further subject with osteoarthritis was discarded due to the loss of the sEMG signal from their unaffected leg. All data was included from the control group.

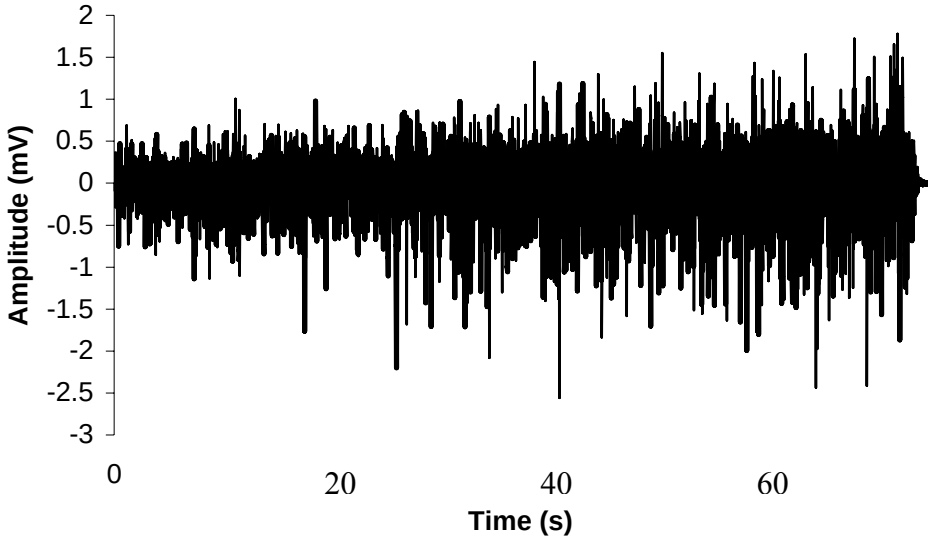

Figure 4.5. Raw sEMG Signal.

An example of a raw sEMG signal collected from the HSR laplacian electrode showing a gradual increase in signal amplitude during the endurance test.

4.6.1 Initial Parameter Values.

Figures 4.6-4.10 show group data for the initial and end values for MDF, MPF, FB1, RMS and CV, respectively. Irrespective of group, all parameters were significantly different ($p < 0.05$) at the beginning of the contraction when compared to the point of fatigue. MDF, MPF and CV decreased with time, while RMS and FB1 increased with time.

Initial values were also compared between groups. In comparison to the unaffected leg, the affected leg showed significantly lower ($p < 0.05$) initial values for MDF (figure 4.6), MPF (figure 4.7) and CV (figure 4.10). No significant differences were seen between the initial values of the unaffected legs and controls. The initial values

of the control leg trended higher than the affected and unaffected legs for MDF and MPF parameters, and lower for FB1.

4.6.2 Percentage Changes in sEMG Parameters

A significant difference ($p < 0.05$) was detected in the percentage change in conduction velocity between the affected (-14.3%, SD 15.2) and un-affected (-28.3%, SD 17.1) legs of the group with osteoarthritis. While this was the only significant finding, a trend towards greater percentage changes occurred for all parameters, except RMS, in the control legs and the unaffected legs in relation to the affected legs.

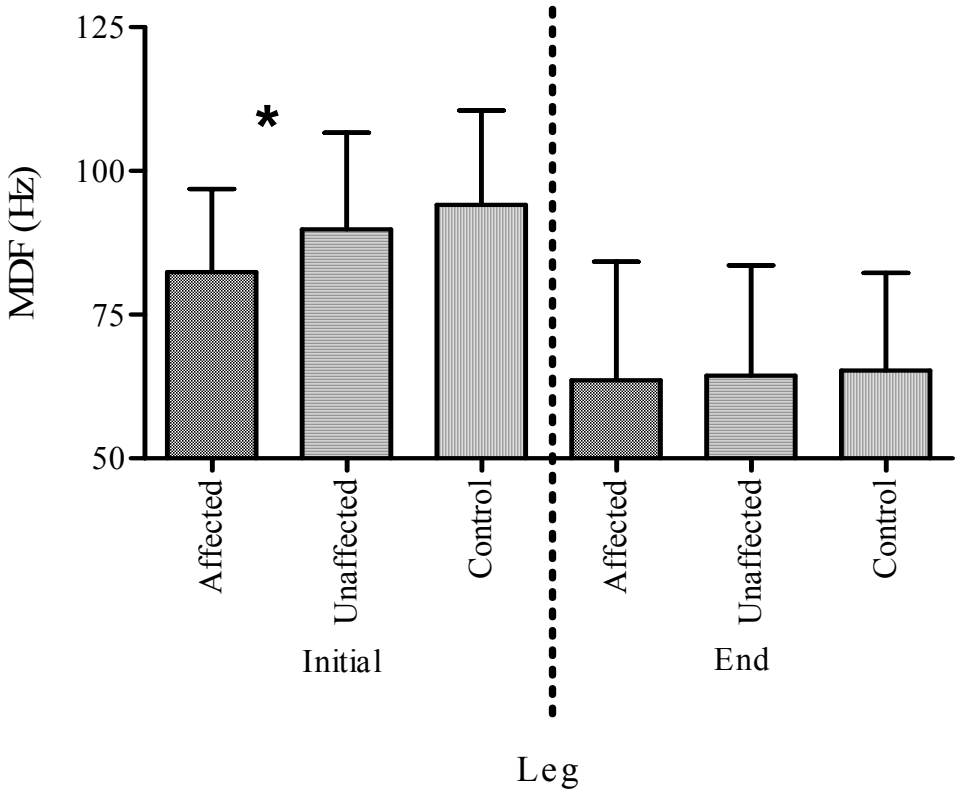

Figure 4.6. Initial MDF Data.

Bar graph of the initial and end values of the affected, unaffected and control legs for the sEMG parameter MDF: Median Frequency. * Significant difference between the initial values of the affected and unaffected legs, $P < 0.05$.

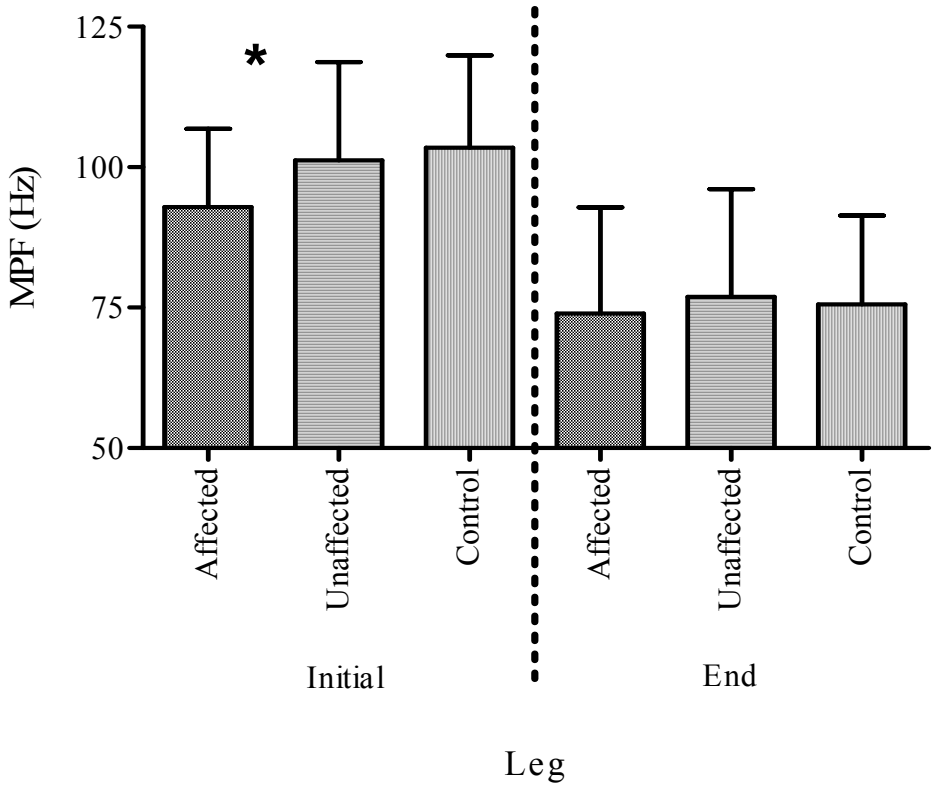

Figure 4.7. Initial MPF Data.

Bar graph of the initial and end values of the affected, unaffected and control legs for the sEMG parameter MPF: Mean Power Frequency. * Significant difference between the initial values of the affected and unaffected legs, $P < 0.05$.

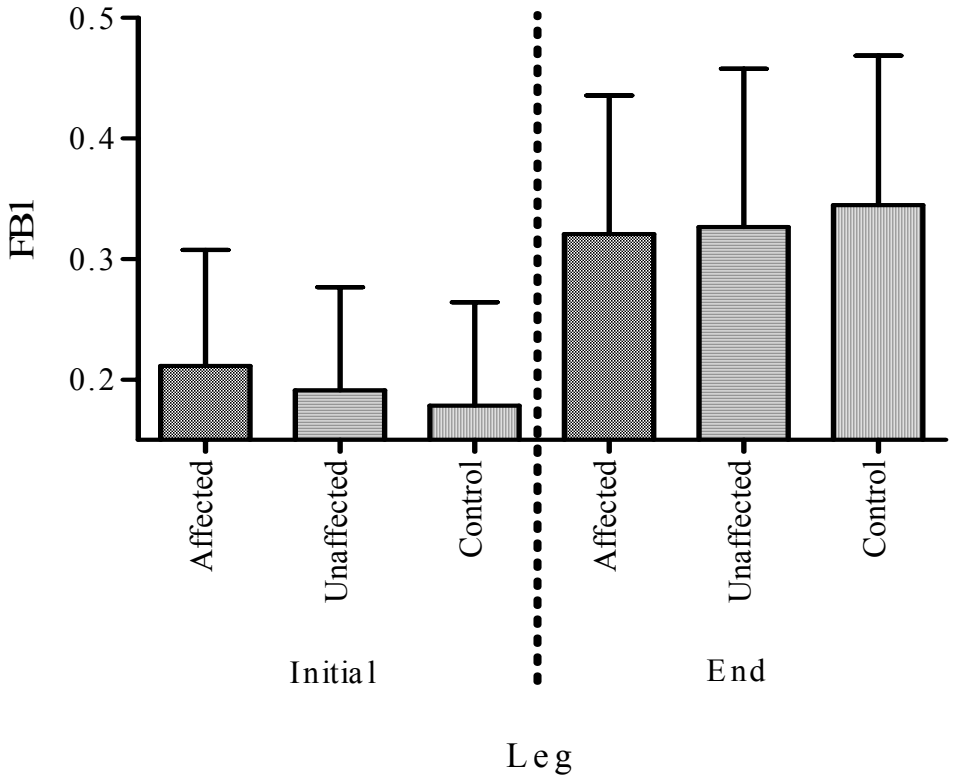

Figure 4.8. Initial FB1 Data.

Bar graph of the initial and end values of the affected, unaffected and control legs for the sEMG parameter FB1: Relative Power 5-30Hz.

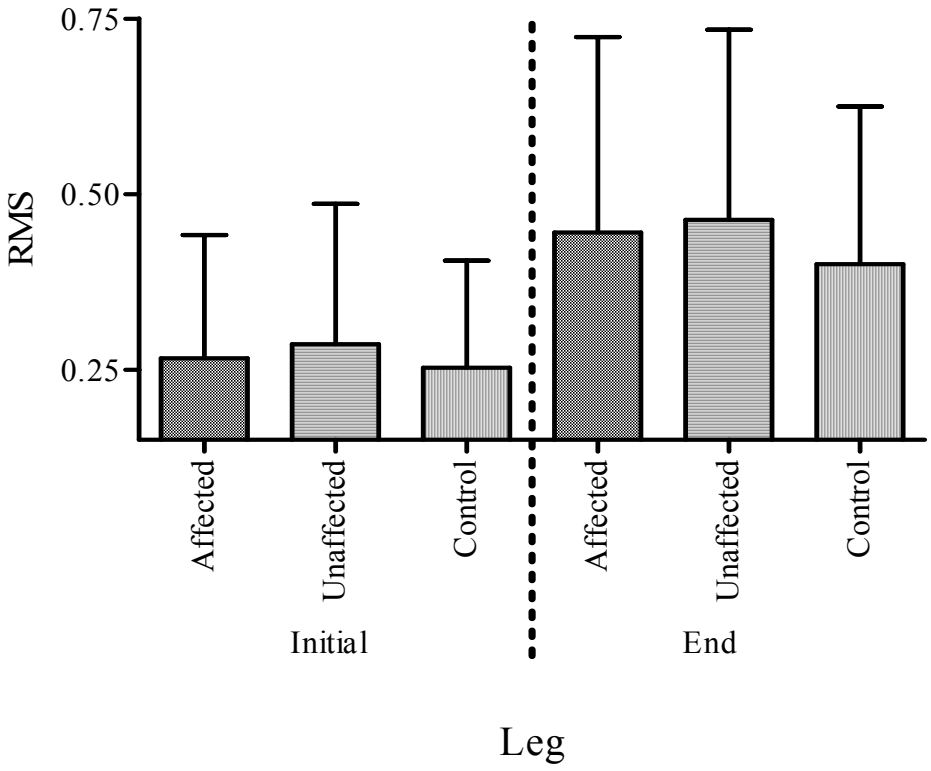

Figure 4.9. Initial RMS Data.

Bar graph of the initial and end values of the affected, unaffected and control legs for the sEMG parameter RMS: Root Mean Square.

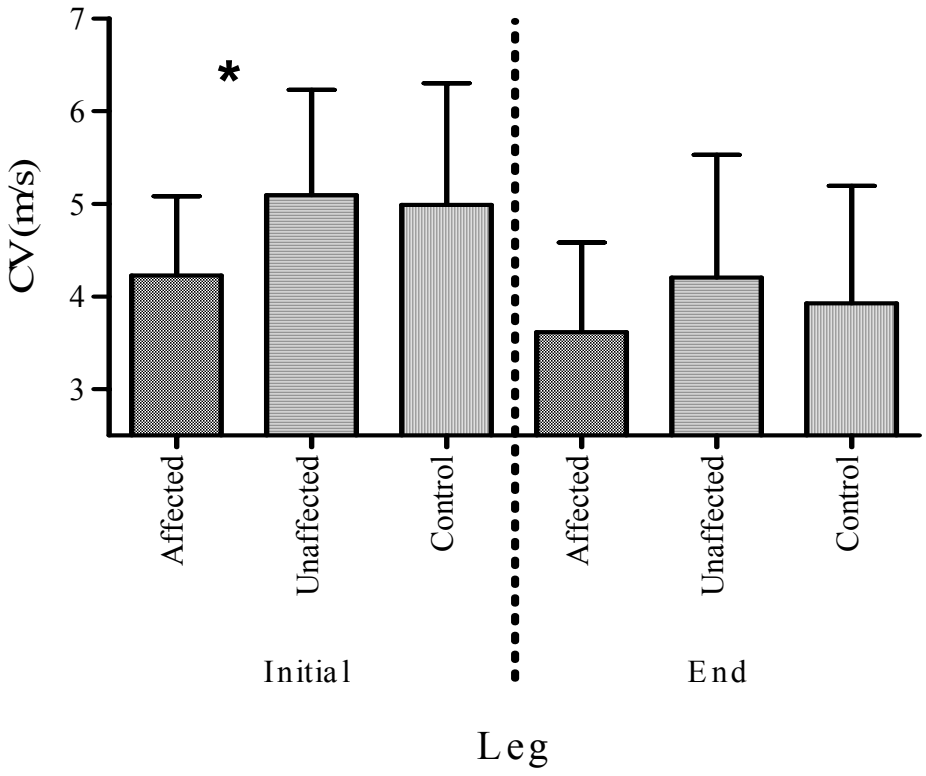

Figure 4.10. Initial CV Data.

Bar graph of the initial and end values of the affected, unaffected and control legs for the sEMG parameter CV: Conduction Velocity. * Significant difference between the initial values of the affected and unaffected legs, $P < 0.05$.

4.6.3 sEMG Parameter Relative Rates of Change.

Figures 4.11-4.15 show the relative slopes (%/s) of MDF, MPF, FB1, RMS and CV respectively, from the affected, unaffected and control legs. Analysis showed that the rate of change of FB1 after 30 seconds (figure 4.13) was significantly lower ($p < 0.05$) for the affected leg (0.180, SD±0.201) in comparison to the unaffected leg (0.297, SD±0.449). While not significant, the control group showed a trend towards greater rates of change than the affected and unaffected legs over the initial 30 seconds of the endurance test, particularly with respect to MDF (figure 4.11), MPF (figure 4.12) and CV (figure 4.15).

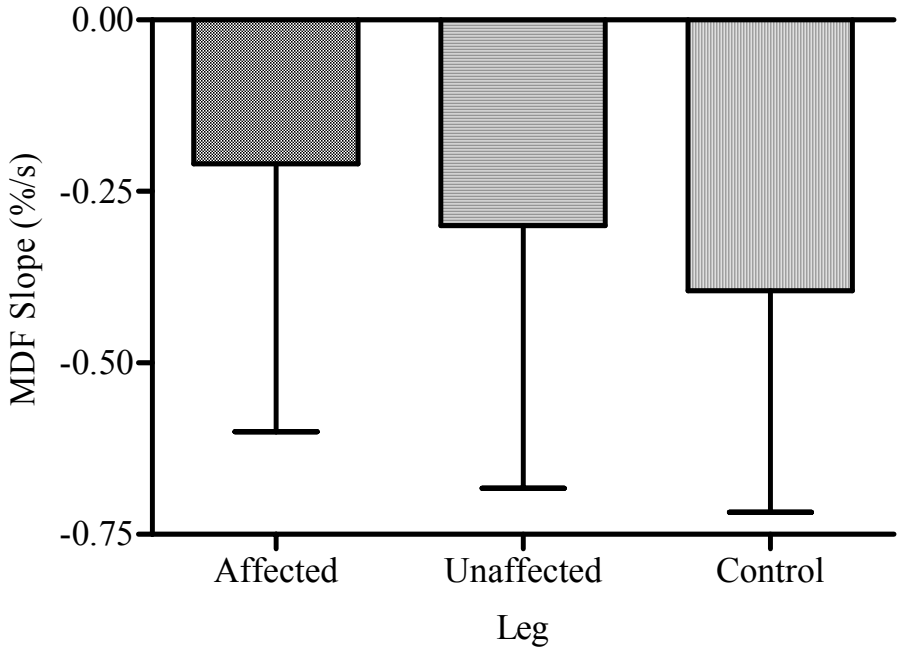

Figure 4.11. MDF Relative Slopes.

Bar graph of the relative slopes (%/s) of the affected, unaffected and control legs for the sEMG parameter Median frequency (MDF) calculated over the initial 30 seconds of the endurance test. %/s: percentage change per second.

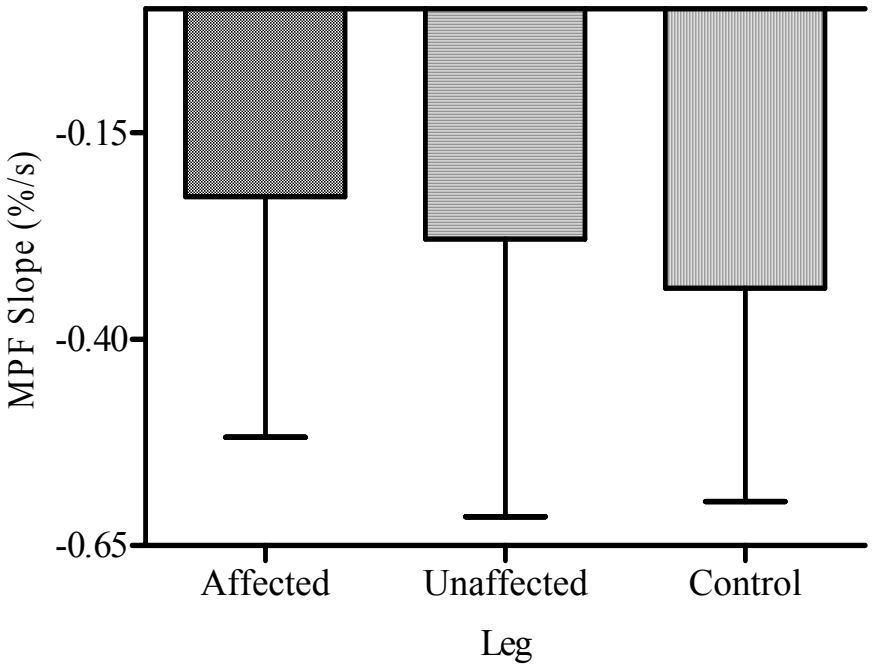

Figure 4.12. MPF Relative Slopes

Bar graph of the relative slopes (%/s) of the affected, unaffected and control legs for the sEMG parameter Mean power frequency (MPF) calculated over the initial 30 seconds of the endurance test. %/s: percentage change per second.

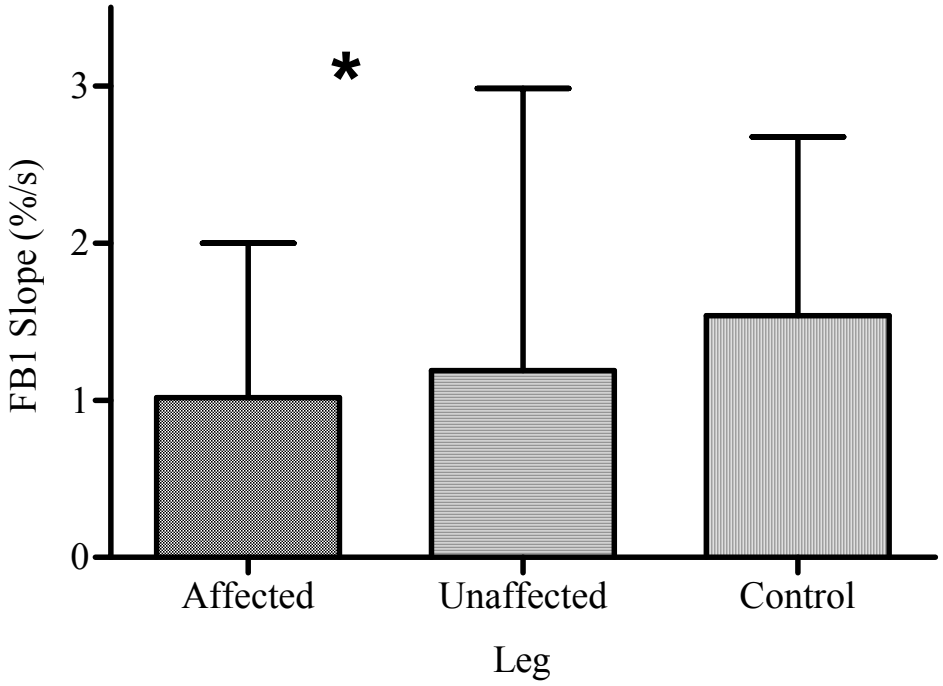

Figure 4.13. FB1 Relative Slopes.

Bar graph of the relative slopes (%/s) of the affected, unaffected and control legs for the sEMG parameter FB1 (relative power in the frequency band 5-30Hz) calculated over the initial 30 seconds of the endurance test. %/s: percentage change per second. *Significant difference between the affected and unaffected legs, $P < 0.05$.

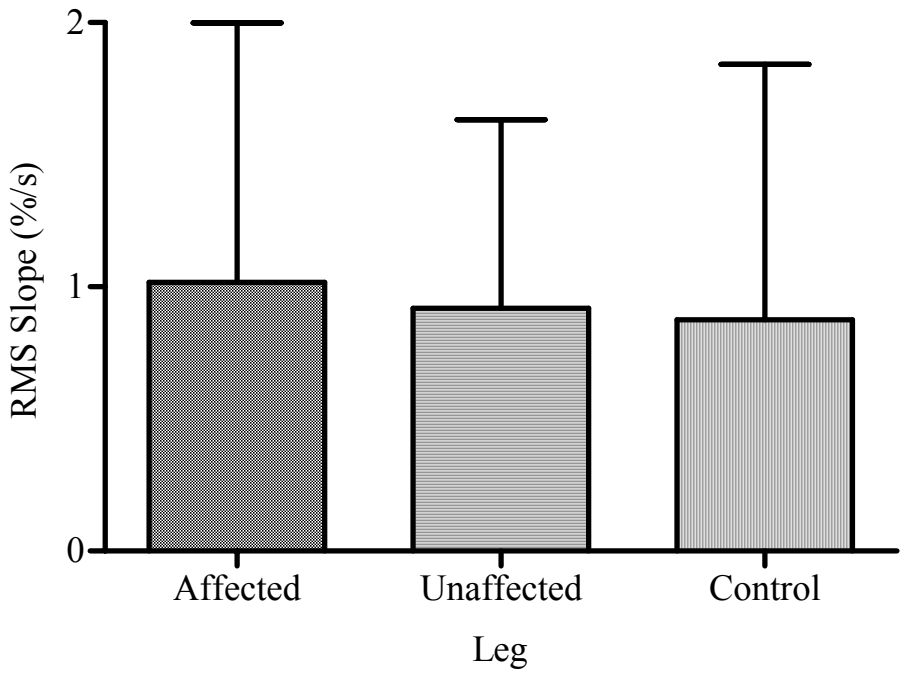

Figure 4.14. RMS Relative Slopes.

Bar graph of the relative slopes (%/s) of the affected, unaffected and control legs for the sEMG parameter Root mean square (RMS) calculated over the initial 30 seconds of the endurance test. %/s: percentage change per second.

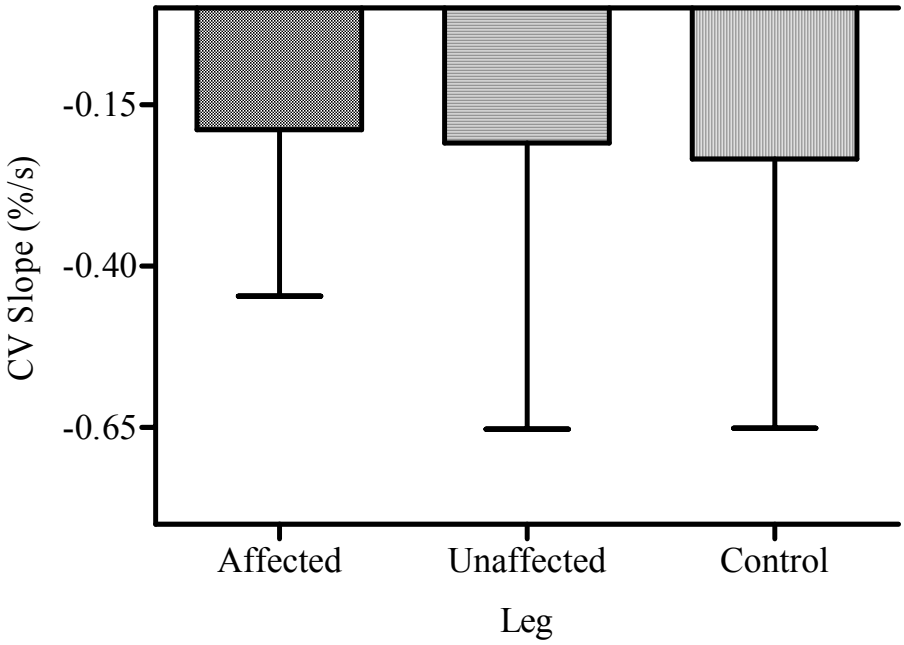

Figure 4.15. CV Relative Slopes.

Bar graph of the relative slopes (%/s) of the affected, unaffected and control legs for the sEMG parameter Conduction velocity (CV) calculated over the initial 30 seconds of the endurance test. %/s: percentage change per second.

4.6.4 Prediction of Endurance Time

T_{Lim}

Correlations were assessed between the endurance time (T_{Lim}) and relative linear slopes (normalised parameters versus time), as well as the area ratio of each parameter calculated over the duration of the contraction. These correlations can be seen in tables 4.3 and 4.4. Of note are the strong correlations observed for the relative slopes of MDF and MPF seen in the control group. There were no significant correlations with endurance time from the area ratio data.

Table 4.3. Slope vs. Endurance-time Correlations: T_{Lim}.

Correlation coefficients (r) of endurance time (T_{Lim}) with the relative slopes of the parameters of interest calculated over the duration of the endurance test.

**P<0.01, **P<0.001 indicate that the correlation was significantly different from zero.

GROUP	MDF	MPF	FB1	RMS	CV
Affected	0.3500	0.4548	-0.3684	-0.4034	0.3853
Unaffected	0.5274	0.4645	-0.1767	0.01376	0.6270**
Control	0.8057***	0.7891***	0.5568	-0.3949	0.5856

Table 4.4. Area Ratio vs. Endurance-time Correlations: T_{Lim}.

Correlation coefficients (r) of endurance time (T_{Lim}) with the area ratios of the parameters of interest calculated over the duration of the endurance test.

GROUP	MDF	MPF	FB1	RMS	CV
Affected	-0.006	0.1557	-0.0592	0.0671	0.0502
Unaffected	0.1047	0.1697	-0.1850	0.0990	0.0558
Control	0.0456	-0.0045	0.5374	-0.1946	0.1174

T_{30}

Correlations were also assessed between T_{Lim} and the slopes and area ratios of parameters over the initial thirty seconds (T_{30}) of the endurance test for the parameters that showed moderate to strong correlations over the full endurance test.

Tables 4.5 and 4.6 show the correlations with endurance time of the relative slopes and area ratios calculated over the initial thirty seconds of the endurance test. From the relative slope data, significant correlations were observed from the control group only. These were moderate to strong correlations for MDF, MPF, and CV. As for the correlations calculated over the full contraction, there were no significant correlations with endurance time from the area ratio data calculated over the initial thirty seconds of the endurance test.

Table 4.5. Slope vs. Endurance-time Correlations: T_{30}.

Correlation coefficients (r) of endurance time (T_{Lim}) with the relative slopes of the parameters of interest calculated over the initial thirty seconds (T_{30}) of the endurance test. *P < 0.05 indicates that the correlation was significantly different from zero.

GROUP	MDF	MPF	FB1	RMS	CV
Affected	0.2517	0.3002	0.0751	0.3067	0.0753
Unaffected	0.4004	0.3318	0.3961	0.3067	0.0753
Control	0.7412*	0.6427*	0.3142	0.4993	0.7215*

Table 4.6. Area Ratio vs. Endurance-time Correlations: T_{30}.

Correlation coefficients (r) of endurance time (T_{Lim}) with the area ratios of the parameters of interest calculated over the initial thirty seconds (T_{30}) of the endurance test.

GROUP	MDF	MPF	FB1	RMS	CV
Affected	0.0069	-0.0023	-0.0165	0.1142	0.0375
Unaffected	0.2264	0.1586	-0.3854	-0.1765	0.2718
Control	0.2802	0.4208	0.3589	-0.5182	0.0954

5.0 DISCUSSION

5.1 INTRODUCTION

This chapter is divided into five main sections. The first four sections discuss the results pertaining to maximum voluntary contractions, voluntary activation deficits, true maximum force estimates and endurance times. The final section addresses the comparisons, mechanisms and implications based around the changes observed in the surface electromyography data from the perspective of initial values, percentage changes, rates of change, and prediction of endurance time. Mechanisms and implications are presented together due to their common links to the changes seen in the sEMG signals.

5.2 SUBJECTS

The mean age, height and weight of the subjects with knee osteoarthritis from the current study were similar to those reported by much larger work by Hurley et al (1997) (n = 103; 60.73yrs; 165cm; 76.95kg), and Fisher & Pendergast (1997) (n = 90; 65yrs; 168.6cm; 85kg). These studies used similar inclusion criteria to the current work. The 18 control subjects in the current study were also similar to the 25 control subjects used by Hurley and co-workers (65.6yrs; 164cm; 74kg), and the 104 control subjects used by Fisher and Pendergast (68.2yrs; 166.2cm; 70.3kg).

The mean WOMAC score from the subjects with knee joint osteoarthritis in the current study was 51.85 (SD±48.44) from a possible score of 240. This represents a mean of 21% of the total score available. This was notably lower than the values reported in the work of Hassan, Mockett and Doherty (2001). In seventy-seven subjects with knee joint osteoarthritis, these authors reported a median WOMAC score of 51, from a maximum of 96, representing 53% of the total score available. Other work by Barker, Lamb, Toye Jackson and Barrington (2004) has reported normalised WOMAC scores ranging from 45.6%-64.7% of the maximum score in 123 subjects with moderate to severe knee joint osteoarthritis (Kellgren-Lawrence scores $\geq$ 2). This suggests that subject in the current study may have been less disabled as the result of their knee osteoarthritis, compared to other studies.

5.3 MAXIMUM VOLUNTARY CONTRACTION

The current study reported a mean of 435N (SD±158.7) for maximum voluntary contractions of the legs affected by osteoarthritis. Mean values in comparable studies are lower, with means between 147N and 226N, and ranges between 40N to 800N (Hassan et al., 2001; Hurley & Scott, 1998; Hurley et al., 1997; O'Reilly, Jones, Muir, & Doherty, 1998). The mean MVC for the unaffected leg (mean 504N; SD±154) in the current study was also higher than the 300N reported in the unaffected legs of a similar subject group with unilateral knee osteoarthritis used by O'Reilly et al. (1998). Also, the current study recorded a mean MVC for healthy subjects of 555.8N (SD±172.8). Again, this was higher than the values reported for healthy control subjects of a similar age in other studies, which ranged from 225N to 335N (Hassan et al., 2001; Hurley et al., 1997; O'Reilly et al., 1998).

The disparity in MVC values between the current and comparison studies may have resulted from a number of factors. Subject ages, the method of MVC measurement, and the angle of the knee joint during testing were all similar in the current and comparison studies. It appears likely, therefore that the differences in the MVC values reported between studies were due mainly to differences in factors such as activity levels and general fitness of the respective subject groups. These differences may have been the result of different attitudes and habits of the subjects towards physical activity. It is acknowledged, however, that because these factors were not examined in the current or comparison studies, the magnitude of their effect can only be speculated. A more definite point of difference noted between the current and comparison studies was the warm-up procedure used. In response to feedback from subjects and observation by the researchers during pilot testing, subjects in the current study exercised for 5 minutes on a stationary cycle in addition to the warm-up protocol utilised in the comparison studies. This more comprehensive warm-up may have been conducive to higher maximum voluntary contractions (Shellock & Prentice, 1985). Again, the precise effect of this difference in warm-up procedure can only be speculated on.

Regardless of the disparities in the MVC values reported between studies, the comparisons between maximum voluntary contractions in the current study show

findings that are consistent with those reported in the literature to date. The results from the current study showed that the quadriceps of the knee affected by osteoarthritis were significantly weaker ($p < 0.01$) than the quadriceps of the unaffected knee during an isometric maximal voluntary contraction. This is consistent with the findings of O'Reilly et al. (1998) who also reported a significant difference in isometric quadriceps strength between the affected and unaffected legs of 300 knee arthritis subjects. In light of the expected physiological changes due to the pathology and/or disuse, such as muscle atrophy (Berg et al., 1991; Young et al., 1982) and muscle activation deficits (Hurley & Newham, 1993; Pap et al., 2004), the differences between legs in the subjects with osteoarthritis observed in this and other studies are expected.

In addition to the significant difference between the affected and unaffected legs in the current study, there was also a trend toward a difference in MVC between the unaffected leg and the control leg (figure 3.1). While this finding did not attain statistical significance, this trend is consistent with the findings of much larger studies that have reported significant differences in voluntary strength of the quadriceps between control subjects and the unaffected leg of subjects with knee osteoarthritis (Hassan et al., 2001; Hurley et al., 1997; Pap et al., 2004).

5.4 VOLUNTARY ACTIVATION DEFICITS

Both muscle fatigue and the sEMG signal are affected by muscle activation intensity (Gerdle & Karlsson, 1994; Gerdle et al., 1997). It was therefore deemed important that all subjects in the current study activated at the same relative intensity to increase the likelihood that any differences in the behaviour of the sEMG signal detected between legs were due mainly to differences in the characteristics of the muscles tested. An important issue in this context is that subjects with knee osteoarthritis have been shown by a number of authors to have voluntary activation deficits in the quadriceps of their affected legs (Hurley & Newham, 1993; Pap et al., 2004). These voluntary activation deficits mean that a subject may produce a maximum voluntary contraction that is significantly less than their true maximum due to muscle inhibition of various origins (Young, 1993). In the current study, an interpolated twitch protocol was used to calculate and adjust for any activation deficits present. This was done by

setting a level of contraction for the fatiguing task equivalent to 50% of the true maximum force (TMF) for the quadriceps group, as calculated from the twitch interpolation procedure, rather than the maximum voluntary force (MVC).

In comparison to other literature, the activation deficits reported in the current study are low. Mean deficits in the control subjects in this study, for example, were 1% (SD 2.1), with a range from 0-7%. In comparison, Hassan et al. (2001) reported mean deficits in control subjects of 12.6% (range 5.7-19%), while Pap et al. (2004) reported mean deficits of 10.7% (SD 8.0). Activation deficits reported in the literature for the quadriceps of knees affected by osteoarthritis range widely from 0% to 95%, with most between 0 and 44% (Hassan et al., 2001; Hurley et al., 1997; Lewek, Rudolph, & Snyder-Mackler, 2004; Pap et al., 2004). The current study is again low in comparison, with a mean deficit of 10.6% (SD±9.4). Only one study could be located that examined the activation deficits in the unaffected legs of subjects with knee osteoarthritis (Berth et al., 2002). This study reported mean quadriceps deficits of 20.7% +/- 13.4 in comparison to the 8.2% (SD±7.2) from the present study.

There are several possible reasons for the discrepancies in voluntary activation deficits reported by various authors. Possible explanations for the differences in activation deficits lie in the methodology used for the twitch interpolation, including the stimulation method, and the model used to estimate the activation deficit. The stimulation for the twitch interpolation protocol may be delivered as a single square-wave pulse, or as multiple pulses separated by 10ms, such as the doublet utilised in the current study. Studies have shown that the variation in calculating activation deficits is lower when multiple twitches are used, relating this to a better signal to noise ratio offered by a multiple-pulse twitch (Behm, Power, & Drinkwater, 2001; Folland et al., 2000; Suter & Herzog, 2001). In light of these studies, it is possible that a proportion of the differences observed in activation deficits between the current study and other comparable literature may be due to the variation between pulse methods utilised.

Other reasons for the variation in activation deficits between studies may relate to the model used to estimate voluntary activation levels. Many studies, including the comparison studies above, have based their estimates of voluntary activation on the assumption of a linear twitch interpolation relationship (Rutherford et al., 1986).

However, the relationship between twitch size and muscle activation has been shown to be more accurately described by a curvilinear model (Behm et al., 1996; Bulow, Norregaard, Danneskiold-Samsoe, & Mehlsen, 1993; Norregaard et al., 1997). The validity of using a linear model to estimate voluntary activation has thus been questioned by these authors in light of the fact that the twitch relationship is curvilinear the majority of the time. Based on this literature, and on pilot data from the current study, the voluntary activation estimates in the present study were calculated using a best-fit model generated over five data points in the belief that this would offer greater accuracy and validity than a simple linear approach. The use of this approach may explain a proportion of the differences in VA estimates in this study in comparison to other studies utilising a two-data-point approach based on a linear model for the twitch interpolation relationship.

5.5 TRUE MAXIMUM FORCE

The true maximum force (TMF) gives the estimated maximal force output for a muscle given optimal muscle activation. In the current study the TMF of the affected leg (490.8N; SD±177.8) was significantly lower than the unaffected leg (551; SD±154). Other studies have also reported significantly lower values for TMF in legs with osteoarthritis in comparison to the unaffected leg in the same subject. O'Reilly et al (1998) for example reported a mean TMF of 300N for the affected legs and 333N for the unaffected legs of 300 subjects ($p < 0.005$). More recent work by Pap et al. (2004) showed a significant difference in TMF torque measures between 85 control subjects (141.8Nm, +/- 50.6) and 68 subjects with moderate knee osteoarthritis, (90.6Nm, +/- 43.7) as well as 154 subjects with severe knee osteoarthritis ($p < 0.001$). The differences between the affected and unaffected knees of those subjects with arthritis appear understandable, given the reduced muscle mass (Convertino et al., 1989; Young et al., 1982) and altered neuromuscular control (Hurley, 1997; Hurley et al., 1997) that are known to occur with such knee pathology.

5.5.1 Bilateral deficits

Although not a focus of the current study, an interesting observation from the data regarding voluntary activation deficits was that subjects with knee osteoarthritis showed a notable quadriceps activation deficit in both their affected and unaffected

legs. This is a finding that has been previously reported with regard to knee osteoarthritis (Berth et al., 2002), and knee ligament injuries (Urbach et al., 1999). The authors of these studies did not offer explanations for these bilateral deficits. Although the mechanisms were not examined in the current study, the causes of a bilateral activation deficit may be speculated on. A possible explanation for the deficits observed may have been an inhibitory mechanism mediated in the central nervous system. It is known that aberrant afferent input from a peripheral joint alters the excitability of alpha-motoneurons in the spinal cord and may lead to arthrogenic inhibition of peripheral muscles relating to that joint (Cervero, Schaible, & Schmidt, 1991; McNair, Marshall, & Maguire, 1996; Schaible, Neugebauer, Cervero, & Schmidt, 1991). In addition, work by Schaible et al. (1991) involving acute arthritis in cat knees has also suggested alteration in the motoneurons that control muscles in the limb contralateral to the arthritis. More recent work by Palmieri et al. (2003), however reports contrasting findings. These authors examined quadriceps inhibition bilaterally after acute infusion of fluid into one knee of eight otherwise healthy subjects. Using a Hoffman-reflex technique, they reported significant changes in the activation of the quadriceps at the affected knee, and no evidence of inhibition in the contralateral quadriceps. While apparently in contrast, the different findings of these studies may have resulted from the different sensory inputs used; one being pain, the other mechanical distension. The acute cross-sectional design of these studies must also be highlighted, in that chronicity of symptoms may be an important antecedent to the development of significant central nervous system changes (Flor, 2003), as may have occurred in subjects involved in the current study due to their osteoarthritis.

From a pathology perspective, the deficits may also have been the result of undiagnosed, asymptomatic osteoarthritis in the unaffected leg. If present, such bilateral pathology may be expected to cause not only activation deficits, but also a change in muscle morphology, and a corresponding decrease in strength. However, in the current study, there was no statistical difference in either the TMF or MVC between the unaffected and control legs. This would provide some indication there was no bilateral pathology.

5.6 ENDURANCE TIMES (T_{LIM})

In relation to other studies, the mean endurance time (66.5s, SD 22.9) recorded for the control group in this study was lower than those reported by other literature utilising similar protocols and healthy subjects. Early work by Hakkinen and Komi (1983) reported mean endurance times for sub-maximal (50%MVC) isometric quadriceps contractions of 89 +/- 27 seconds. More recently Maisetti et al. (2002) reported similar findings, with a mean endurance time of 78.8 seconds (SD 9.5). This suggests a reduced relative resistance to fatigue in the control subjects of the current study in relation to subjects used by Hakkinen & Komi, and Maisetti and co-workers. Because the current and comparison studies utilised similar methods to test endurance time, the differences between these studies may relate to a number of other factors. These factors are likely to include different morphological, psychological or physiological or training states, as well as age differences between the respective study groups. The current and comparison studies did not assess the first three of these factors, thus their influence on the differences observed cannot be determined. With respect to age, a comparison between studies shows that the subjects in the current study were notably older than the subject groups used by Hakkinen & Komi (1983) and Maisetti et al. (2002). While it is possible that these age differences played a role in the different endurance times between studies, literature examining the effect of age on relative fatigue resistance appears divided, with some showing a reduction, whereas others have reported an improvement (Allman & Rice, 2002).

While there were no significant differences in endurance times across the different legs in the current study, there was a trend towards greater endurance times for the affected leg in relation to the unaffected and control legs. No comparison studies were found that reported endurance times of sub-maximal isometric contractions from muscles that were associated with joint pathology.

5.7 SURFACE ELECTROMYOGRAPHY

The main purpose of this study was to examine changes in fatigue characteristics in the vastus medialis quadriceps muscle of subjects with osteoarthritis of the knee joint as measured using high spatial resolution (HSR) laplacian surface electromyography

(sEMG). Therefore, because of the relationship between sEMG and fatigue, one of the key findings of the current study was a significant difference in the behaviour of specific sEMG variables between the affected and unaffected legs of the subjects with knee osteoarthritis. Interestingly, a non-significant trend suggesting the same finding between the unaffected and control legs was also observed. These differences implied changes at a muscular and/or a central control level that differentiated the groups.

5.7.1 Initial values

Few studies have reported initial values of sEMG parameters collected from sub-maximal (50%MVC) isometric contractions of the quadriceps. In the studies reviewed, only MDF, and CV were reported. The MDF values from each group in the current study (82.4Hz, SD 14.4 Affected; 89.8Hz, SD 16.9 Un-affected; 94.1Hz, SD 16.5 control) were higher than those reported by these studies. Linssen et al. (1991), for example, reported MDF values of 77+/-13.5Hz from the vastus medialis of 12 healthy subjects. Mannion and Dolan also reported initial values of MDF, but only graphically, and only from the vastus lateralis and rectus femoris. The estimations from the graphs provided in this work showed mean initial values of 64Hz and 69.5Hz respectively. As well as MDF initial values, Linssen and co-workers also reported the initial values for conduction velocity in the same study. The mean value of 5.5+/-1.4m/s was higher than that recorded for the affected leg (4.2m/s, SD 0.9), and slightly higher than the unaffected (5.1m/s, SD 1.1) and control (5.0m/s, SD 1.3) legs in the current study. Despite the differences between the current and comparison studies, the values from the current study fell within the accepted physiological range for muscle fibre conduction velocity (De Luca, 1997).

In respect to parameter values of the group with osteoarthritis, the affected leg in the current study produced initial values for MDF and MPF that were significantly lower ($p < 0.05$) than those of the unaffected leg. No other literature could be found that reported initial values in pathological and contralateral muscles during sub-maximal isometric contractions. However, a number of studies have reported similar trends in sEMG initial values using maximal rather than sub-maximal contractions of the quadriceps muscles. Early work by McNair and Wood (1993), for example noted significantly lower MDF values collected from the vastus laterali of subjects following anterior cruciate ligament reconstruction in comparison to healthy controls. In light of these lower initial frequency values, McNair and Wood speculated that the

muscles of the affected leg had undergone preferential atrophy of type-2 muscle fibres. Similar findings were reported by McHugh and co-workers (2001) examining vastus medialis during maximal isometric contractions. Significant differences ($p < 0.01$) in initial MDF values were seen in subjects following anterior cruciate ligament rupture in comparison to normal controls. Similar to McNair and Wood, McHugh speculated also that the differences in frequency values suggested specific atrophy of type-2 muscle fibres in the affected legs.

5.7.2 Percentage changes

With regard to sEMG parameters, few studies have reported percentage changes during isometric fatiguing contractions of limb muscles. Even fewer have reported specifically in relation to the quadriceps. The percentage changes in MDF reported in the current study for the affected (-22%, SD±20.5), unaffected (-28%, SD±17.1) and control legs (-31%, SD±10.3) were similar to those of a comparable study by Mannion and Dolan (1996), who reported MDF decreases of 22% in vastus lateralis and 32% in rectus femoris quadriceps muscles during a sub-maximal (50%) isometric contraction. Values represented graphically in later work by Maisetti et al. (2002) were significantly lower than the current study. The graphs of Maisetti and co-workers indicate percentage decreases in MDF between 7.5% and 8.5% for the same endurance test used in the current study. These authors also indicated increases in the FB1 parameter of between 9% and 35% during the endurance test. The changes in FB1 recorded in the current study were notably greater in the affected (67%, SD±45.4), unaffected (88%, SD±104.3) and control legs (121%, SD±90) than those of Maisetti.

With respect to differences in percentage changes between legs in the current study, a significantly lesser rate of change in CV ($p < 0.03$) was observed in the affected leg in comparison to the unaffected leg. Few other studies appear to have made this comparison. In one such study, Duchateau and Hainaut (1991) have reported similar findings to the current work, but in the percentage change of MDF. These authors described significantly ($p < 0.05$) smaller percentage decreases in MDF from thumb muscles immobilised for six weeks (-14%) compared to control subjects (-28%). Duchateau and Hainaut surmised that these differences were the result of altered neural control, as well as reductions in the contribution made to the sEMG signal by type-2 muscle fibres. More recent work by McHugh et al. (2001) also suggested

similar findings. While these authors did not specifically comment on percentage changes, graphical evidence clearly showed less change in the MDF values collected from the VMO of subjects with anterior cruciate ligament rupture compared to those without this injury.

5.7.3 Relative Rates of change

The rate of change of sEMG variables has been used to quantify muscle fatigue for many years. Of the studies examining quadriceps fatigue with sEMG, it appears that only Mannion and Dolan (1996) have reported relative rates of change for the quadriceps during sub-maximal (50%) isometric fatiguing contractions. These authors examined rectus femoris and vastus lateralis quadriceps muscles with respect to MDF, and reported the results graphically. From the graphical data the values for vastus lateralis (-0.34%/s) were lower, while those from the rectus femoris (-0.47%/s) were similar to those reported in the current study for VMO in the unaffected (-0.49%/s, SD±0.45) and control legs (-0.55, SD± 0.27). It is of note that the MDF slope for the leg affected by osteoarthritis (-0.23%/s, SD±0.23) was noticeably less than the healthy legs in Mannion and Dolan's work. A non-significant trend reflecting the same relationship between MDF slope of the unaffected and control legs to that of the affected legs in the current study was also evident.

With respect to comparing rates of change in normal and pathological muscles, the current study revealed that the relative change of FB1 and CV calculated over the entire contraction were significantly ($p < 0.02$) less for the affected leg compared to the unaffected leg. Similar to the results from the current study, McHugh et al. (2001) described a significantly lesser rate of change in the MDF collected from the VMO of subjects following knee surgery and five weeks of immobilisation compared to that collected pre-operatively. Though these results are similar to those of the current study, it should be noted that sEMG data was collected from a maximal rather than a sub-maximal isometric contraction. Together with the work of McHugh et al. (2001), the observations of reduced rates of change in the affected leg of the current study differ from those of Portero et al. (1996). These authors reported no significant change in the rate of decrease in MPF taken from the tibialis anterior muscle during a sub-maximal (50%MVC) isometric contraction following four weeks of bed-rest. Also in contrast to the current study, Portero and co-workers described a significantly

greater ($p < 0.05$) rate of MDF decrease in the gastrocnemius muscle of the same subjects after bed-rest.

5.8 MECHANISMS

The current study did not examine the mechanisms responsible for changes in the behaviour of the sEMG signal. However, based on findings from other literature, it is possible to speculate on these mechanisms, and how they may relate to the changing fatigue characteristics of a muscle.

5.8.1 Initial values

Morphology and Physiology

Morphological and physiological factors must be considered when examining the differences seen in initial values between legs in the current study. With respect to morphological factors, past research has shown links between initial surface EMG frequency parameters and muscle fibre composition. In Linssen's human study mentioned previously, for example, initial values for MDF were shown to be lower in subjects with 95-100% type-1 muscle fibre composition in their quadriceps (69.0+/-14.2Hz) compared to control subjects with a more normal distribution of type-1 and type-2 muscle fibres (77.0+/-13.5Hz) (Linssen et al., 1991). These findings are consistent with those of Kupa et al. (1995) who reported a strong positive correlation ($r = 0.92$) between initial MDF and the percentage of type-2 fast twitch muscle fibres in rat muscle. Taken in light of these findings, the lower initial frequency values seen in the affected leg compared to the unaffected leg in the current study may, in part, be the result of specific atrophy of type-2 muscle fibres in the affected legs. Such changes in the muscles of the affected legs would most likely be the related to the knee osteoarthritis of these subjects.

Further support for a link between the initial frequency values and the proportions of type-2 muscle fibres in the current study is found in the well-established relationship between sEMG frequency measures and muscle fibre conduction velocity. Electromyography frequency measures have long been related to conduction velocity, in that changes in conduction velocity cause changes in the frequency content of a

sEMG signal (Arendt-Nielsen & Mills, 1985; Broman, Bilotto et al., 1985a; Sadoyama & Miyano, 1981; Stulen & DeLuca, 1981). This relationship is observed in the current study in the significantly lower initial values for both of the central tendency measures (MDF and MPF) *and* the conduction velocity in the affected leg compared to the unaffected leg. That the initial frequencies and conduction velocity were significantly lower in the affected leg than the unaffected leg suggests that any alterations in the muscle of the affected leg influenced these measures together. Such alterations would include the specific atrophy of type-2 muscle fibres in particular, resulting in a muscle with a reduced conduction velocity, as well as a lower frequency content to its sEMG signal (Kupa et al., 1995).

While differences in fibre-types may explain some of the differences between legs in the current study, the role of muscle fibre-types in the different initial conduction velocity and frequency values recorded in the current and comparison studies is not clear. In simple terms, the higher initial values in the current study, for example, imply that subjects in the current study may have had higher levels of type-2 muscle fibres compared to those of the comparison studies. However, this suggestion seems unlikely for several reasons. Firstly, increasing age has been related to decreasing proportions of type-2 muscle fibres (Deschenes, 2004). In this context, the age of the subjects in the current study (63.6yrs SD 12.51 and 64.69yrs SD 9.52) was notably higher than those used in the comparison studies of Linssen (Males 36+/- 11.3yrs; Females 30 +/-6.4yrs) and Mannion and Dolan (30+/-6.4yrs). Secondly, with specific reference to the affected leg in the current study, the muscle atrophy associated with knee osteoarthritis implies either a reduction in type-2 muscle fibres, or a generalised atrophy of all fibres. Both the age related and atrophy related alterations in fibre-type that are likely in the current study population imply a reduction in initial conduction velocity and frequency values in the affected legs (at least) of the current study in relation to the comparison studies. This situation is contrary to what was observed. This suggests that either muscle fibre-type differences were not responsible for the differences seen between the studies, or that any fibre-type differences present were outweighed by other factors separating the studies. For firm conclusions to be made on the role of fibre-type differences, further information on muscle morphology, for example, would be required to make a clearer distinction between factors.

In keeping with the context of fibre-type variation, differences in initial values between the current work and specifically that of Mannion and Dolan (1996) may be due to the different muscles that each study used. The vastus lateralis and rectus femoris muscles used by Mannion and Dolan are known generally to contain greater proportions of type-2 muscle fibres in comparison to vastus medialis (Johnson et al., 1973). Hence, it may be expected that higher initial MDF and CV values would be measured from these muscles compared to VMO. The observation that the current study in fact reported higher initial values from VMO than those reported from the vastus lateralis and rectus femoris suggests that other factors played a more significant role than the difference in muscles used, or that the subjects in the current study had higher levels of type-2 muscle fibres in their vastus medialis muscles than the subjects of the comparison studies due to natural variation.

In relation to other morphological factors that may have affected the initial values in the current study, skin and subcutaneous tissue thickness, for example, are also known to influence the sEMG signal (Farina, Cescon et al., 2002). Hence, the generally lower initial frequency values observed in the affected leg in relation to the unaffected leg may have been caused, to some degree, by either an increase in the proportion of subcutaneous tissue in relation to muscle on the affected side due to muscle atrophy, or an increase in intramuscular fatty tissue in the affected leg (Fuchs, Weishaupt, Zanetti, Hodler, & Gerber, 1999; Nordal, Dietrichson, Eldevik, & Gronseth, 1988). While either of these situations would reduce the frequency content of the sEMG signal through the increased filtering effect of the subcutaneous tissue, they would also reduce the amplitude of the detected signals (Farina, Cescon et al., 2002). The data in the current study satisfies the first of these conditions, by way of lower initial frequency values in the affected leg (figures 4.6 and 4.7). However, with regard to the RMS, there were no significant differences observed in the initial values collected from each leg, indicating no decrease in amplitude in the EMG signal from the affected leg (figure 4.9). On this basis, it can be surmised that differences in subcutaneous tissues may not have played a large role in the differences seen in initial values between legs of the subjects with osteoarthritis. However, as the current study did not control for skin or subcutaneous tissue thickness, their true impact on the sEMG values reported cannot be deduced.

Skin and subcutaneous tissue thickness can also be considered with respect to the differences in initial values between the current study and comparison studies. As discussed previously, greater tissue thicknesses are associated with greater filtering of the high frequency component of a sEMG signal. In relation to the comparison studies, it may be expected that subjects in the current study had greater levels of subcutaneous tissue due to their greater age (Das, Gabriely, & Barzilai, 2004; Kanehisa, Miyatani, Azuma, Kuno, & Fukunaga, 2004), as well as a reduction in activity levels that is expected in the case of the subjects with osteoarthritis due to their disability (O'Reilly et al., 1998). Subsequently, relatively higher levels of subcutaneous tissue in the subjects of the current study would be expected to manifest as lower initial sEMG parameter values in relation to the comparison studies. That the initial values in the current study for all legs were higher than the comparison studies suggests that factors other than skin and subcutaneous tissue differences played a more significant role in the different initial values seen between studies. In light of the multiple factors that can influence the sEMG signal, and because the current and comparison studies did not directly measure or control for skin or subcutaneous tissue thickness, their true impact on the sEMG values reported cannot be determined.

EMG methodology

In conjunction with morphological factors, differences in initial values between the current and comparison studies may be related to differences in the sEMG recording techniques used. In the case of the work of Linssen et al. (1991), the linear electrode array and recording technique employed was sufficiently similar to the set-up used in the current study to suggest that there would be no substantial differences in sEMG recordings as a result of the collection technique. The slight differences in the values reported may have been due to the use of a two-dimensional Laplacian set-up in the current study rather than the uni-dimensional linear array of Linssen et al. (991). However, in contrast to the HSR-EMG set-up used in the current study, Mannion and Dolan used passive electrodes with a large inter-electrode distance of 30mm. Furthermore, these were placed on the respective muscles using only general guidelines (SENIAM). This sEMG methodology used by Mannion and Dolan is likely to have resulted in greater filtering and a reduced sensitivity to spectral values. These factors may have sufficiently altered the frequency content of the sEMG signals in their study, and hence the different initial values reported compared to the current study.

Neuromuscular changes

Separate to changes in conduction velocity, the duration of the motor unit action potential (MUAP) has been shown to increase in a disused or immobilised muscle (Duchateau & Hainaut, 1987; Hoyer, Eickhoff, & Rumberger, 2000). Furthermore, changes in the duration of the MUAP are known to alter the sEMG frequency content, and are related to muscle membrane excitability and muscle fibre conduction velocity (Dimitrova & Dimitrov, 2003). As well as a change in proportions of muscle fibre-types, it is possible, therefore, that increases in the duration of the MUAP in the vastus medialis of the affected leg in the current study may be partially responsible for the lower initial frequency and conduction velocity measures observed in relation to the unaffected leg. However, because MUAP duration was not measured in the current study, further work directly examining this would be required to shed light on the role that a change in MUAP duration has on sEMG values in this situation. Furthermore, while a change in MUAP duration may have also had some role to play in the differences seen between the current and comparison studies, its effect cannot be deduced from the data available.

Along with an *increase* in the duration of the MUAP, the maximum firing rate of a muscle has been shown to *decrease* as the result of disuse and immobilisation (Duchateau & Hainaut, 1987, 1990). Although firing rates were not measured in the current study, the nature of knee osteoarthritis would suggest that the maximal firing rates in the current study would have been reduced in the VMO of the affected leg, at least. Several authors have demonstrated that firing rates influence the lower part of the sEMG power spectrum between 0 and 40 Hz (Farina, Merletti, & Enoka, 2004; van Boxtel & Schomaker, 1984). Other authors have also speculated that a decrease in firing rates may be a mechanism that, in conjunction with a prolonged action potential, reduces membrane stress and therefore prolongs the duration of a muscle contraction (Behm & St-Pierre, 1998). Hence, in the case of the affected leg in the current study, a lower firing rate may have contributed to both the lower initial values seen in the frequency measures (MDF, MPF), as well as the slightly longer (non-significant) endurance time from the affected leg. However, the work of Duchateau and Hainaut (1990) reported that up to the (reduced) maximum firing rate of a disused muscle, firing rates are the same as for a normal muscle. In addition to this, it has been suggested that the quadriceps are not fully recruited until they are generating

more than 75% of their maximum voluntary force (De Luca, 1984). Up to this point, firing rates do not change. Above this point, firing rates increase in order to generate more force from the fully recruited muscle. With this in mind, the quadriceps may not have been fully recruited during the sub-maximal (50%TMF) contraction in the current study. This being the case, the firing rates may not have been maximal, and thus not different in the affected and unaffected legs. Therefore, in the current study, differences in firing rates seem unlikely to have contributed to the differences seen in the initial sEMG values between the affected and unaffected legs, or in the differences seen with comparison quadriceps studies.

5.8.2 Magnitudes and Rates of Change

The changes in SEMG amplitude, frequency and conduction velocity parameters have been used as a method of indicating muscle fatigue for many years. With regard to the different magnitudes and rates of change in sEMG parameters, a number of mechanisms may be involved in causing the differences observed between the affected and unaffected legs in the current study, as well as variation in relation to the values reported by other studies.

Morphology and Physiology

From a morphological perspective, early work by Komi and Tesch (1979), for example, has indicated higher rates of change of sEMG spectral variables (MPF, MDF) in the quadriceps muscles of subjects with greater proportions of type-2 muscle fibres. Linssen and co-workers (1991) also related greater rates of change in sEMG parameters during isometric fatiguing contractions with higher proportions of type-2 muscle fibres. They specifically reported significantly greater rates of decrease in CV for subjects with a normal distribution of fibre-types in their quadriceps, in comparison to their subjects with congenital myopathies. While the differences were not significant, they also reported greater rates of change in MDF for the same muscles. The *in vitro* work of Kupa et al. (1995) also demonstrated different percentage and rates of change in MDF from rat soleus, diaphragm and extensor digitorum longus muscles. Greater rates of change were found to correspond to higher relative percentages of type-2 fibres in the three muscles tested. In light of these studies, it appears that a reduction in the proportion of type-2 muscle fibres in the affected leg may be a key factor in the differences in magnitudes and rates of change seen between the affected leg and the unaffected leg in the current study.

From a physiological perspective, other literature has associated type-2 muscle fibres with greater accumulation of lactic acid, and thus greater decreases in muscle pH during fatiguing contractions (Tesch, 1978; Tesch, Sjodin, Thorstensson, & Karlsson, 1978). Furthermore, Brody et al. (1991) have reported greater rates of change in spectral and conduction velocity parameters with decreases in muscle pH. With this in mind it can be appreciated that muscles with a lesser proportion of type-2 fibres may have smaller and slower changes in EMG variables due to smaller decrease in pH levels. This situation is consistent with that seen in the affected leg in comparison to the unaffected leg, and lends further support to the premise that a preferential atrophy of type-2 muscle fibres in the affected leg was a significant factor in the differences in sEMG variables observed.

An alternative suggestion is that preferential atrophy of type-1, rather than type-2, muscle fibres may have occurred in the affected leg of the current study. Preferential atrophy of type-1 muscle fibres intuitively results in a muscle that is more dominant in type-2 muscle fibres. As discussed in an earlier section, these type-2 fibres are known to utilise glycolytic/anaerobic energy systems when they function. With this in mind, it is known that isometric contractions have been shown to be hypoxic above 20% MVC (Edwards, Hill, & Jones, 1972). It may be possible that a muscle with a greater representation of type-2 glycolytic fibres is therefore better suited to sub-maximal isometric contractions, and hence result in reduced initial and change values seen in sEMG variables, such as those from the affected leg in the current study. However, this hypothesis appears unlikely for several reasons. As discussed earlier, other studies have shown a greater accumulation of lactic acid in type-2 muscle fibres during fatiguing contractions (Kupa et al., 1995)**.** Correspondingly, greater rates and percentages of decline in spectral and conduction velocity variables have been associated with greater muscle acidity (Brody et al., 1991). Furthermore, a muscle with higher relative proportions of type-2 muscle fibres would also be expected to produce higher initial conduction velocity values than unaffected muscles, as well as greater rates of change in the sEMG parameters (Kupa et al., 1995). Because the initial and change values for the affected leg were in fact lower than the unaffected leg in the current study, the likelihood of preferential type-1 fibre atrophy in the affected subjects involved in the current study is therefore questionable.

A further alternative to the preferential atrophy of type-2 muscle fibres relates to the generalised atrophy of both type-1 and type-2 muscle fibres reported by some authors as the result of immobilisation or disuse (Gerber et al., 1985; Veldhuizen et al., 1993). With respect to the generalised fibre-atrophy, an affected muscle would intuitively be expected to maintain the same relative proportions of each fibre type, while losing gross cross-sectional area. When it is considered that muscle fibre cross-sectional area has been related to conduction velocity, it can be appreciated that a generalised atrophy of all fibres could, as with type-2 fibre atrophy, result in a decreased global conduction velocity value. As demonstrated extensively within sEMG literature (Arendt-Nielsen & Mills, 1985; Brody et al., 1991; Broman, Bilotto et al., 1985a; C. J. De Luca, 1984; Gerber et al., 1985; Veldhuizen et al., 1993), such a general decrease in conduction velocity will also result in a decrease in the frequency content of the sEMG power spectrum, and correspondingly lower initial frequency measures. Such effects are consistent with the findings of the current study of lower initial frequency and CV values from the affected leg in relation to the unaffected leg. However, it might be expected that a muscle affected by generalised fibre atrophy would have the same relative physiological characteristics. Hence, at a given contraction intensity (such as 50% of TMF) such a muscle may produce lower frequency and CV values, yet have similar or the same rates of change in these parameters. In the current study, both the initial values and the rates of change of sEMG parameters were reduced in the affected leg in relation to the unaffected leg. Hence, in this context it appears that generalised muscle fibre atrophy may not be a factor in the differences in the behaviour of the sEMG signal observed between the affected and unaffected legs in the current study.

Other physiological changes associated with atrophy may also have influenced the rates and magnitude of change in the sEMG signal in the current study. Some authors have, for example, theorised that a decrease in muscle area may lead to improved perfusion within a muscle (Behm & St-Pierre, 1998). Such improvements in perfusion may result in improved relative resistance to fatigue by slowing the onset of metabolic changes, such as the reductions in muscle pH or membrane excitability that have been related to muscle fatigue and changes in the sEMG signal. However, the evidence from the literature investigating improvements in perfusion appears equivocal. Thus, its role in the sEMG changes seen in the current study, and others, is speculative at best.

A further contributing factor to the reduced magnitude of change, the slower rate of change, and the improved relative resistance to fatigue associated with the affected leg in the current study may relate to the additional recruitment of muscle fibres during the fatigue test. With regard to constant force contractions, it appears that recruitment occurs mainly during force levels below 30%MVC (Gazzoni et al., 2001), and that only a small amount of, if any, additional motor units are recruited (Christova & Kossev, 2001; Jensen et al., 2000). Despite notable activation deficits in the affected and unaffected legs, it is unlikely that any subjects in the current study contracted at such low levels of activation due to the correction implemented using the twitch interpolation protocol. Thus, with subjects contracting at or close to 50%TMF, it is questionable whether the recruitment of additional motor units influenced the sEMG signals in the current study.

5.8.3 Control Leg

The majority of sEMG comparisons made between the control and unaffected legs in the current study showed no significant difference between them. This implies that there were no differences in the vastus medialis quadriceps muscles between these legs. A conclusion along those lines appears reasonable considering the very similar demographics of the group with osteoarthritis of the knee and the control group, and the fact that these legs were ostensibly free of pathology. However, there appeared to be consistent, yet non-significant, trends between the control and unaffected legs that were along similar lines to those seen between the unaffected and affected legs. In light of the previous discussion points, these trends may therefore reflect differences between the unaffected leg of the subjects with knee osteoarthritis, and the control group. These differences may, for example, be due to any of the morphological or physiological changes discussed earlier. The validity of the trend seen in the current study would require further testing involving larger subject numbers, at least. Significant differences observed between the unaffected and control legs may indicate either predisposing factors to, or early indicators of, knee osteoarthritis, and that these differences may be detectable using surface electromyography. However, such a trend may also indicate that any differences between the unaffected leg of the subjects with osteoarthritis and the control leg may merely be the result of general deconditioning resulting from the disability related to the condition.

5.9 PREDICTION OF ENDURANCE TIME

Utilising sEMG parameters to predict endurance times has been undertaken with moderate success by several authors using healthy subjects and sub-maximal isometric contractions of limb muscles (Maisetti et al., 2002a; Mannion & Dolan, 1996; Merletti & Roy, 1996). The ability to predict endurance times using only a short duration sub-maximal test is appealing when considering several factors. With respect to the current study, these may include factors such as motivation and pain due to knee osteoarthritis, which may affect the performance and validity of a test held to exhaustion. In this context, a key finding from the current study related to the prediction of endurance time using the rates of change of various sEMG parameters. The ability to predict endurance times was expressed as the correlation between the relative rates of change of the sEMG parameters and endurance times. These relationships were investigated using the sEMG signal collected over the entire muscle contraction, with further correlations investigated over the initial thirty seconds of the endurance test for those parameters showing moderate to high correlations over the whole test.

Significant correlations for relative slopes were found in the MDF and MPF parameters from the control legs, as well as for CV from the unaffected leg, over the entire contraction (table 4.3). Using the area ratio measure, no significant correlations were seen with endurance time over the full test. Using a very similar protocol, Maisetti and co-workers also reported significant correlations for the same parameters as those in the current study, but with absolute slopes as well as area ratios taken from healthy subjects**.** Similar to the relative slope data of the current study, Maisetti and co-workers reported moderate correlations for the absolute slopes of MPF ($r = 0.63$). In contrast to the present study, Maisetti also reported a moderate correlation between RMS and endurance time ($r = 0.75$). Further contrasts to the present work occurred with regard to FB1, where Maisetti and co-workers reported that the area ratio of FB1 provided a moderate to strong correlation with endurance time when calculated over the full contraction ($r = 0.78$).

Significant correlations were also observed in a number of variables calculated over the first thirty seconds in the current study (table 4.5). Of these variables, the relative

slopes of MDF and MPF, together with CV, from the control leg showed moderate to high correlations with endurance time using only a short duration contraction. In contrast, the absolute slope correlations were similar to Maisetti et al. (2002), who reported poor correlations from the absolute slopes ($R < 0.35$) for all parameters over the first thirty seconds of their sEMG data. Area ratio calculations over the initial thirty seconds of the test in the current study provided no significant correlations with endurance time. This contrasted to the work of Maisetti, who reported strong correlations with endurance time for the area ratios from FB1 calculated over the first thirty seconds of their endurance test ($r = 0.82$).

It appears that the current study is the first to investigate the prediction of endurance time using sEMG in muscles affected by pathology in some way. With regard to endurance time prediction in the affected and unaffected legs in the current study, correlations for all parameters were poor or moderate when calculated over the entire contraction. When correlations were calculated for the relative slopes, as well as the area ratio index over the first thirty seconds of sEMG data in the current study, the ability to predict endurance times became even weaker in the affected and unaffected legs. Correlations for all variables calculated over the first thirty seconds of sEMG data from the affected and unaffected legs in the current study were poor. Along with the mostly non-significant correlations calculated over the entire contraction, this implies that the ability to predict endurance time using sEMG is compromised in some way in the quadriceps muscles of knees affected by osteoarthritis. Why this disparity exists is not readily apparent, although there are a number of possible reasons.

Examination of the results, for example shows large differences in the variation in relative slopes between the legs of the subjects with osteoarthritis and the control legs. These differences can be appreciated in view of the standard deviations of the relative slopes of MDF of each leg after 30 seconds. When calculated as a percentage of the mean slope, the standard deviation of the control leg was noticeably less (82%) than the unaffected leg (128%), and particularly the affected leg (187%). When the large variations of the MDF slopes for the affected and unaffected legs are considered it is appreciable that the correlations of rates of change to endurance times were low for the affected and unaffected legs. The broader implication from the greater variance in parameter rates of change in the osteoarthritis group is that the mechanisms of fatigue

may be more varied in the muscles of the affected leg than those of the unaffected (or control leg). The variation may be the result of different responses to knee osteoarthritis within the subject group. This could relate, for example to muscle fibre atrophy. As discussed earlier, the rate of change of sEMG variables has been related to muscle fibre-type proportions (Gerdle et al., 1997; Kupa et al., 1995). Also, it appears within the literature that has investigated muscle atrophy that immobilisation/disuse induces atrophy of type-1 or type-2 muscle fibres, or both. It is thus possible that some of the variation in rates of change seen in the affected and unaffected legs in the current study could be the result of different atrophic responses to knee osteoarthritis within individuals.

Differences in atrophic responses may also mean variation between individuals in the changes to other more intrinsic/physiological muscle factors. Behm and St Pierre (1998) have theorised, for example that improved calcium flux mechanisms and increased concentrations of intramuscular inorganic phosphate due to immobilisation can lead to improved fatigue resistance through an improvement in the efficiency of the contraction coupling mechanism. They also speculated that the increase in contraction coupling efficiency, along with a possible reduction in firing rates and an increase in motor-unit action potential duration may reduce membrane stress, and prolong a muscle contraction. As well as conceivably improving endurance times, it would appear reasonable to suggest that a reduction in membrane stress would slow the changes in membrane excitability that are linked to declines in sEMG spectral and CV variable (Dimitrova & Dimitrov, 2003). Variability in the extent of changes such as these between individuals may thus also contribute to the variation seen in the rates of change of the sEMG variables from the affected and unaffected legs in the current study.

To explain the increased variances in both the affected and unaffected legs, it is interesting to note that changes to the muscle that occurred would have had to occur bilaterally in subjects, despite the unilateral nature of the pathology in the current study. Such bilateral changes may have occurred for several reasons. As covered previously, knee osteoarthritis has been associated with a decrease in activity levels (Sharma et al., 2003). Hence, a decrease in activity is likely to manifest as bilateral deconditioning in some way. However, within such general decrease of activity, such as may have been the case in the current study, levels of activity may still be very

variable. Such variability in activity levels could naturally lead to variations in muscle morphology and physiology, and correspondingly varied sEMG signals. While activity levels were not examined in the current study, anecdotally there was a notable variation, with some subjects maintaining essentially normal levels of activity, while others were significantly disabled. Confirmation of this variation as a factor in the results of the current study would require further investigation.

As well as, or separate to any peripheral muscle changes, the increased variance in the rates of change in the osteoarthritis group bilaterally may have been caused in part by a central neuromuscular mechanism. Complex speculation as to the origins and mechanisms of such a central component are beyond the scope of this work.

6.0 SUMMARY AND CONCLUSIONS

Osteoarthritis is the most common form of arthritis, and leads to significant financial and social cost. Knee joint osteoarthritis is recognised as a significant subset of osteoarthritis, having the greatest economic impact due to productivity losses resulting from the disability (Felson et al., 2000; Yelin et al., 1998). Significant research has been entered into regarding the physiology and pathology of bones in relation to osteoarthritis. It is only more recently that increasing attention has been paid to the muscles associated with osteoarthritic joints. Much of this work has centred on strength deficits, with several authors surmising that these deficits are critical factors in not only the progression but also the development of osteoarthritis (Hurley, 1999; Slemenda et al., 1997). As well as a muscle's strength, its ability to resist fatigue is an important functional determinant. Despite this notion, minimal work has examined the effect of joint pathologies such as osteoarthritis on the fatigue resistance of the muscles associated with a diseased joint.

The purpose of this study was to investigate the fatigue characteristics of one quadriceps muscle (vastus medialis) using high spatial resolution electromyography during an endurance test. Results were compared between the affected and unaffected legs of subjects with knee osteoarthritis to determine differences within an individual. Results were also compared between the unaffected legs of the subjects with osteoarthritis, and the dominant leg of control subjects. It was anticipated that this would provide information on the fatigue characteristics of muscles affected by disuse/immobilisation that may allow more accurate prescription of rehabilitation programs, which are currently strength-focused. Finally, the ability to predict the endurance time of an individual using sEMG parameters collected during a sub-maximal short duration test was investigated. Success in this part of the study would mean that fatigue could be assessed in subjects with knee osteoarthritis without the need of exhaustive, potentially uncomfortable tests.

Twenty-six subjects with unilateral knee osteoarthritis, and seventeen subjects with no known knee pathology were evaluated. All subjects performed initial tests to evaluate maximum voluntary contraction (MVC), voluntary activation levels, and true maximum force (TMF). Endurance time was assessed during an isometric quadriceps

contraction at 50% of the true maximum force. Surface electromyography data was collected from the vastus medialis muscle of the quadriceps group during the fatiguing contraction.

The results from the MVC tests showed that the affected leg of the group with osteoarthritis was significantly weaker than the unaffected leg. With respect to voluntary activation, subjects with osteoarthritis presented with significant bilateral deficits. Calculation of TMF also found significantly lower true potential for force generation in the affected compared to the unaffected leg of the osteoarthritis group. Endurance time data showed no significant difference between groups, but a clear trend to greater endurance times in the affected leg was noted.

The EMG data from five subjects with osteoarthritis was not included due to the poor quality of their signals. Analysis of electromyography data collected during the endurance test showed significant differences between the affected and unaffected legs in initial values of MDF, MPF and CV, the percentage change in CV and the relative rate of change in FB1. These differences suggested improved relative resistance to fatigue in the legs with osteoarthritis. This added weight to the non-significant trend in endurance times. While there were few significant differences between the unaffected leg and the control leg, trends between these legs were similar to those between the affected and unaffected legs. Finally, significant correlations were seen between endurance time and the relative rate of change of MDF, MPF and CV calculated over the initial thirty seconds of the endurance test. This translates to a moderate success in the ability to predict the endurance time of healthy subjects using sEMG data collected from only a short duration contraction (30 seconds) and one muscle. This prediction ability did not manifest in either leg of the group with osteoarthritis.

Initial conclusions may be made in relation to the strength and activation data from the current work. Consistent with other osteoarthritis studies, muscle components relating to strength are altered in the leg affected by osteoarthritis in the current study in relation to the unaffected leg. This is evidenced in the MVC and TMF indices. It appears also that a central mechanism related to the unilateral osteoarthritis affects the ability of subjects with knee joint osteoarthritis to activate fully the quadriceps muscles of either their affected or unaffected leg.

In relation to the main purpose of this study, it can be concluded that there are differences in the behaviour of the sEMG signal between the vastus medialis muscle of the affected and unaffected legs of subjects with unilateral knee osteoarthritis. It appears likely that a significant contributing factor in the differences observed in the sEMG signals from these legs was a decrease in the proportions of type-2 muscle fibres in the vastus medialis of the affected leg. Furthermore, these changes in the behaviour of the signal also appear to indicate an improvement in the relative fatigue resistance of the affected leg in relation to the unaffected leg of the group with knee osteoarthritis. Such findings are consistent with other literature showing increased resistance to fatigue during isometric contractions in muscles with low proportions of type-2 muscle fibres (Hulten et al., 1975), as well as studies indicating improved relative fatigue resistance following knee injury (Halkjaer-Kristensen & Ingemann-Hansen, 1985), and knee surgery (McHugh et al., 2001; Snyder-Mackler et al., 1991). However, while the evidence from the literature supports the role of type-2 fibre proportions in explaining most of the differences in the sEMG signal seen in the current study, care may need to be exercised in making this assumption. In light of the many factors that may influence the initial values, percentage changes, and rates of change of sEMG variables, general caution should be exercised when making assumptions of fibre-type distributions based on sEMG amplitude, spectral and conduction velocity measures alone (Farina, Merletti et al., 2004). Thus, to further strengthen the findings of the current work in relation to muscle morphology, further investigations would be required.

Moderate success was seen with the prediction of endurance time in control subjects in the current work using the initial 30-seconds of EMG data from the endurance test. Interestingly, this relationship was not apparent in either the affected or unaffected leg of the subjects with knee osteoarthritis. The development of an endurance test utilising sEMG and a sub-maximal short duration muscle contraction is appealing with respect to painful knee conditions such as knee osteoarthritis. However, the inconsistency of the prediction relationship in the group with knee osteoarthritis questions the likelihood that such a test is feasible. Further investigation utilising different sEMG collection and analysis techniques may be required in this area to reduce parameter variability and perhaps develop different measurement indices.

Conclusions relating to the sEMG and strength indices may also be made in a clinical context. When it is considered that the strength measures in the affected leg were reduced, while the relative fatigue indices appeared to improve in the same leg, it can be hypothesised that the changes that occur within the vastus medialis muscle in relation to knee osteoarthritis may have a greater effect on the strength components of the muscle rather than those components related to fatigue-resistance. From a clinical perspective, this suggests that the strength focus of current rehabilitation programs is appropriate. Improvements in strength as the result of these programs also imply improvements in functional, or absolute, resistance to fatigue. Hence, it appears that the addition of a rehabilitation component specifically targeting relative fatigue resistance is not necessary; at least until a deficit in relative fatigue resistance is identified once strength deficits have been corrected.

7.0 RECOMMENDATIONS

Electromyography data from five subjects was excluded from analysis in the current study due to the poor quality of their signals. Because collection began only when real-time inspection indicated a satisfactory signal, the subsequent distortion of the signal is believed to have been due to electrode movement during the contraction. In most cases this appeared to occur when the bulging of the contracting muscle caused one or several pins of the rigid electrode to lift away from the skin. This occurred despite attempts to secure the electrode adequately. In light of this problem, future studies might use a flexible electrode, or an electrode with spring-loaded pins. Alternatively, studies investigating a single quadriceps muscle could use the broader and flatter vastus lateralis instead of the rounder vastus medialis muscle.

The use of high spatial resolution electrodes with Laplace filter functions is a significant advance in sEMG technology. While the current study utilised an eleven-pin, three channel electrode of this type, recent studies have shown that electrodes with a greater number of pins and channels allow more accurate calculation of sEMG temporal and spectral sEMG parameters (Farina et al., 2001; Farina, Zagari et al., 2004). More accurate estimation of parameters using such arrangements may allow clearer identification of any differences between subjects, as well as strengthening conclusions made on this basis.

Prediction of endurance time showed moderate to poor success in the current study. Prediction work by Maisetti et al (2002), Mannion and Dolan (1996) and Duchêne and Gouble (1990) has indicated that when there is a group of synergistic muscles acting across a joint, such as in the knee, the use of multiple muscles in fatigue analyses is desirable. Therefore, in relation to the current study, more success with prediction of endurance times may be found, particularly in the group with osteoarthritis, if further studies utilised vastus lateralis and/or rectus femoris muscles in addition to vastus medialis.

As is apparent in the discussion section of the current work, skin and subcutaneous tissue thickness are potentially significant confounding variables in sEMG fatigue analysis. Further studies in this area would benefit by controlling for these factors using tissue thickness measures and available sEMG models.

The activity level of individuals is also a potentially important confounding factor that should be investigated and controlled for in future work. Measurement of subjects' physical activity may take the form of the Habitual Activity Questionnaire (Baecke, Burema, & Frijters, 1982), or the Physical Activity Scale (PASE) for the elderly (Schuit, Schouten, Westerterp, & Saris, 1997).

The current work indicates that there may be differences in most sEMG measures between the unaffected leg of the subjects with osteoarthritis, and the control leg. The consistency of the trend across the study variables suggests that, with a larger subject group and methodological improvements, further investigation may show significant differences between these legs. Further study in this regard may investigate the potential to detect changes in muscles, using surface electromyography, which may be precursors to the development of knee osteoarthritis. Additionally, in light of the differences observed in the sEMG signal between the affected and unaffected legs, similar work could examine the ability to classify or diagnose knee osteoarthritis using surface electromyography, such as been done with regard to lumbar spine (Roy et al., 1989; Roy et al., 1995) and specific neuromuscular conditions (Disselhorst-Klug et al., 2000; Huppertz et al., 1997; Rau, Disselhorst-Klug, & Silny, 1997).

Finally, it is interesting to note the presence of a bilateral voluntary activation deficit in the group with knee osteoarthritis in the current study. Further investigation into

this area may look at the mechanisms behind this bilateral deficit, and also the relationship of this deficit to the onset of knee osteoarthritis. In this context, a voluntary activation deficit, such as in the unaffected leg of the current study, may prove to be an early sign of, or a precursor to, the development of knee osteoarthritis.

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9.0 APPENDICES

Appendix 1

Kellgren Lawrence Radiographic classification system (Kellgren and Lawrence, 1957).

Grade	Classification	Description
0	Normal	No features of OA
1	Doubtful	Minimal osteophytes, doubtful significance.
2	Minimal	Definite osteophytes with unimpaired joint space.
3	Moderate	Moderate diminution of joint space.
4	Severe	Joint space greatly impaired with sclerosis of subchondral bone.

Appendix 2

American College of Rheumatology: Criteria for Classification of Idiopathic Osteoarthritis of the Knee (Altman et al. 1986).

ESR = Erythrocyte sedimentation rate; RF = rheumatoid factor; SF OA = synovial fluid signs of OA (clear, viscous, or white blood cell count < 2000/mm^3).

Clinical and Laboratory	Clinical and Radiographic	Clinical
Knee pain + at least 5 of 9	Knee pain + at least 1 of 3	Knee pain + at least 3 of 6
Age > 50 years	Age > 50 years	Age > 50 years
Stiffness < 30 mins	Stiffness < 30 mins	Stiffness < 30 mins
Crepitus	Crepitus	Crepitus
Bony tenderness		Bony tenderness
Bony enlargement	+ Osteophytes	Bony enlargement
No palpable warmth		No palpable warmth
ESR > 1:40		
RF < 1:40		
SF OA		
92% Sensitive	91% Sensitive	95% Sensitive
75% Specific	86% Specific	69% Specific

Appendix 3

EMG Reliability Study Summary

MPF = Mean power frequency; MDF = Median frequency; RMS = Root mean square; FB1 = Relative power in 5-30Hz frequency band; CV = Conduction velocity; LCI = Lower confidence interval of the ICC value.

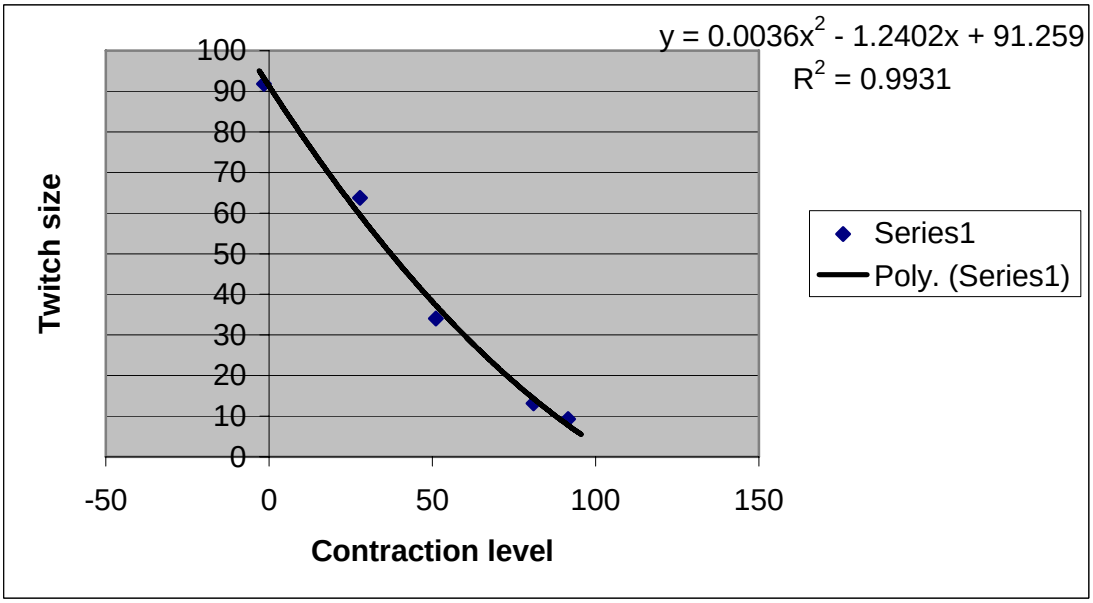
PARAMETER	TYPICAL ERROR (% mean)
MPF0	4.568379629
MPF1	1.613328322
MPF2	5.256434523
MDF0	6.843151542
MDF1	3.689333391
MDF2	8.158109139
RMS 0	10.24253948
RMS 1	12.12174613
RMS 2	10.3911369
FB1 0	23.01852038
FB1 1	16.90445687
FB1 2	26.45927567
CV1	-3.762986449
CV2	-3.327576994

Parameter	Mean		Correlation	F-stat	P	SM ICC	LCI
	Trial 1	Trial 2					
MPF C0	118.68	119.29	0.9083	0.0450	0.8350	0.9113	0.7564
MPF C1	127.87	128.28	0.9760	0.0930	0.7684	0.9690	0.9107
MPF C2	105.54	107.18	0.9494	0.5808	0.4587	0.9502	0.8617
MDF C0	100.09	103.49	0.8726	0.6951	0.4184	0.8739	0.6727
MDF C1	113.88	112.81	0.9070	0.2067	0.6567	0.9074	0.7485
MDFC2	87.83	86.51	0.9204	0.2029	0.6593	0.9190	0.7779
RMS C0	0.6829	0.6308	0.9649	2.7355	0.1204	0.9554	0.8698
RMS C1	0.7243	0.6578	0.9759	3.8444	0.0701	0.9568	0.8644
RMS C2	0.7158	0.6491	0.9836	5.6240	0.0326	0.9697	0.8870
FB1 C0	0.1774	0.1774	0.7921	0.0000	0.9975	0.8032	0.5036
FB1 C1	0.1228	0.1138	0.8321	1.1331	0.3051	0.8261	0.5687
FB1 C2	0.2799	0.2190	0.8270	4.9813	0.0425	0.7460	0.3624
CV 0-1	5.1850	5.1688	0.9410	0.0407	0.8436	0.9441	0.8275
CV 1-2	4.8757	4.8620	0.8839	0.0085	0.9277	0.8873	0.6959

Appendix 4

Calculation of True Maximum Force (TMF)

1. To calculate the TMF, use the best fit equation to find the x-axis intercept. See the graph below for examples of a polynomial second order model, and its R^2 value.
    a. For a linear equation it is as simple as re-arranging the equation $y = mx + c$ to get the intercept
        i. Intercept = c/m
    b. For a polynomial equation use the following formula based on $y = ax^2 + bx + c$
        i. Intercept = $\frac{-b \pm \sqrt{b^2 - 2ac}}{2a}$
        ii. If the polynomial doesn't intercept the x-axis, then the equation will give an error. If this occurs, plot a linear relationship using just the first and last twitch values (not the relationship plotted with all of the values).



2. The x-intercept of the best fit represents a percentage.
    a. I.e. 109 = 109%

3. Multiply the subject's MVC value by the x-intercept (as a percentage) to get their TMF.
    E.g. 400N x 1.09 = 436N TMF

Appendix 5

AUCKLAND UNIVERSITY OF TECHNOLOGY

TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

CONSENT FORM

Predicting muscle fatigue in individuals who have knee osteoarthritis

English	I wish to have an interpreter	Yes	No
Maori	E hiahia ana ahau ki tetahi Kaiwhakamaori/Kaiwhakapakeha korero	Ae	Kao
Samoan	Oute mana'o ia iai se fa'amatala upu	Ioe	Leai
Tongan	Oku ou fiema'u ha fakatonulea	Io	Ikai
Cook Island	Ka inangaro au I tetai tangata uri reo	Ae	Kare
Niuean	Fia manako au ke fakaaoga e taha tagata fakahokohoko kupu	E	Nakai

I…… consent to participate in the study, which has been explained to me to my satisfaction. **YES/NO**
I have read and fully understand the information provided and have asked questions of any procedure I did not understand. **YES/NO**
I am fully aware that taking part in this study is entirely voluntary, that I may withdraw from the study at any time, and my continuing health care will not be affected in any way. **YES/NO**
I understand that my participation in this study is confidential, and that no material that could identify me will be used in reports on this study. **YES/NO**
I agree that my data may be published provided my identity is not disclosed. **YES/NO**
I understand the compensation provisions in the event of physical injury to myself as a result of participation in this study. **YES/NO**
Do you wish to have a copy of the results? **YES/NO**

Name (Print): _____

Signature:_____Date:_____

Witness (Print): _____ Date:_____

Signature:_____

Investigators:

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Auckland University of Technology
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Appendix 6

WOMAC Index

Baseline: Name:_____ Trial no.:_____

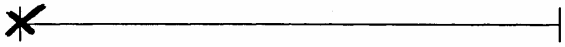
WOMAC OSTEOARTHRITIS INDEX VERSION VA3.1

Instructions to Patients

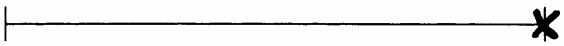
In Section A, B, and C questions will be asked in the following format. You should give your answers by putting an "*x*" on the horizontal line.

Examples:

1. If you put your "*x*" at the left of the line as shown below, then you are indicating that you have **no** pain.

No Pain  Extreme Pain

2. If you put your "*x*" at the right end of the line as shown below, then you are indicating that you pain is **extreme**.

No Pain  Extreme Pain

3. Please note:
   a. That the further to the right you place your "*x*" the **more** pain you are experiencing.
   b. That the further to the left you place your "*x*" the **less** pain you are experiencing.
   c. **Please do not** place your **"*x*" past the end of the line.**

You will be asked to indicate on this type of scale the amount off pain, stiffness or disability you have experienced in the last 48 hours.

Complete the questionnaire with respect to you knee.
You should think about your knee when answering the questionnaire. Indicate the severity of your pain, stiffness and physical disability that you feel is caused by arthritis in your knee.

Baseline: Name:_____ Trial no.:_____

Section A
PAIN

Think about the pain you felt in you knee due to your arthritis during the last 48 hours.

(Please mark you answers with an "*x*".)

QUESTION: How much pain do you have?	Study Coordinator Use Only
1. Walking on a flat surface. No Pain ——— Extreme Pain	Pain1_____
2. Going up or down stairs. No Pain ——— Extreme Pain	Pain2_____
3. At night while in bed. No Pain ——— Extreme Pain	Pain3_____
4. Sitting or lying. No Pain ——— Extreme Pain	Pain4_____
5. Standing upright. No Pain ——— Extreme Pain	Pain5_____

Section B
STIFFNESS

Think about the stiffness (not pain) you felt in your knee due to arthritis during the last 48 hours.

Stiffness is a sensation of **restriction** or **slowness** in the ease with which you move your joints.

(Please mark your answers with an "*x*".)

	Study Coordinator Use Only
6. How **severe** is your stiffness **after first awakening** in the morning? No Stiffness ——— Extreme Stiffness	Stiff6_____
7. How severe is your stiffness after sitting, lying or resting **later in the day**? No Stiffness ——— Extreme Stiffness	Stiff7_____

Baseline: Name:_____ Trial no.:_____

Section C

DIFFICULTY PERFORMING DAILY ACTIVITIES

Think about the difficulty you had in doing the following daily physical activities due to arthritis in your knee during the last 48 hours. By this we mean **your ability to move around and to look after yourself.** (Please mark your answers with an "***x***".)

QUESTION: **What degree of difficulty do you have?**	Study Coordinator Use Only
8. Descending stairs. No Difficulty ——— Extreme Difficulty	PFTN8_____
9. Ascending stairs. No Difficulty ——— Extreme Difficulty	PFTN9_____
10. Rising from sitting. No Difficulty ——— Extreme Difficulty	PFTN10_____
11. Standing. No Difficulty ——— Extreme Difficulty	PFTN11_____
12. Bending to the floor. No Difficulty ——— Extreme Difficulty	PFTN12_____
13. Walking on a flat surface. No Difficulty ——— Extreme Difficulty	PFTN13_____

Baseline: Name:_____ Trial no.:_____

DIFFICULTY PERFORMING DAILY ACTIVITIES

Think about the difficulty you had in doing the following daily physical activities due to arthritis in your knee during the last 48 hours. By this we mean **your ability to move around and to look after yourself.** (Please mark your answers with an "***x***".)

QUESTION: **What degree of difficulty do you have?**	Study Coordinator Use Only
14. Getting in or out of a car, or getting on or off a bus. No Difficulty ——— Extreme Difficulty	PFTN14_____
15. Going shopping. No Difficulty ——— Extreme Difficulty	PFTN15_____
16. Putting on your socks or stockings. No Difficulty ——— Extreme Difficulty	PFTN16_____
17. Rising from bed. No Difficulty ——— Extreme Difficulty	PFTN17_____
18. Taking off your socks or stockings. No Difficulty ——— Extreme Difficulty	PFTN18_____
19. lying in bed. No Difficulty ——— Extreme Difficulty	PFTN19_____

Baseline: **Name:**_____ **Trial no.:**_____

DIFFICULTY PERFORMING DAILY ACTIVITIES

Think about the difficulty you had in doing the following daily physical activities due to arthritis in your knee during the last 48 hours. By this we mean **your ability to move around and to look after yourself.** (Please mark your answers with an "*x*".)

QUESTION: **What degree of difficulty do you have?**	Study Coordinator Use Only
20. Getting in or out of the bath. No Difficulty |——— Extreme Difficulty	PFTN20_____
21. Sitting. No Difficulty |——— Extreme Difficulty	PTFN21_____
22. Getting on or off the toilet. No Difficulty |——— Extreme Difficulty	PTFN22_____
23. Performing heavy domestic duties. No Difficulty |——— Extreme Difficulty	PTFN23_____
24. Performing light domestic duties. No Difficulty |——— Extreme Difficulty	PTFN24_____

Appendix 7

Ethical Approval Notification

Please include the reference no. and study title in all correspondence/telephone calls.

Auckland Ethics Committees
Private Bag 92522
Wellesley Street
Auckland
Delivery Address:
C/O Ministry of Health
3rd Floor, Unisys Building
650 Great South Road, Penrose
Phone (09) 580 9105
Fax (09) 580 9001
Email: pat_chainey@moh.govt.nz

19 July 2004.

A/Prof Peter McNair
School of Physiotherapy
Auckland University of Technology.
PB 92 006
Auckland.

Dear Peter,

AKX/02/00/368 Predicting muscle fatigue in individuals who have suffered a stroke or a significant knee injury: PIS/Cons V#2, 15/01/2003: PIS/Cons V#3, 20/02/2003

Thank you for your progress report, received 8 July 2004.

The Chairperson of Ethics Committee X reviewed the report for this study.

The study has received ongoing ethical approval for the next twelve months. The next progress report is due 19 July 2005. If the study is not completed by this time, please note that a progress report is required to ensure ethical approval is continued as after that date, it would withdrawn if we haven't heard from you.

It should be noted that Ethics Committee approval does not imply any resource commitment or administrative facilitation by any healthcare provider, within whose facility the research is to be carried out. Where applicable, authority for this must be obtained separately from the appropriate manager within the organisation.

Yours sincerely,

Pat Chainey
Administrator, Committee X.

Accredited by Health Research Council