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NEURAL CONTRIBUTIONS TO MAXIMAL MUSCLE PERFORMANCE

by

MATTHEW BUCKTHORPE

A doctoral thesis

Submitted in partial fulfilment of the requirements for the award of

Doctor of Philosophy of Loughborough University

May 2014

ABSTRACT

Neural activation is thought to be essential for the expression of maximal muscle performance, but the exact contribution of neural mechanisms such as the level of agonist, antagonist and stabiliser muscle activation to muscle strength is not fully understood. Explosive neuromuscular performance, including the ability to initiate (the electromechanical delay, EMD) and develop force rapidly (termed, rate of force development, RFD) are considered essential for the performance of explosive sporting tasks and joint stabilisation and thus injury avoidance. The thesis aimed to improve our understanding of the contribution of neural factors to muscle performance, with a specific focus on explosive neuromuscular performance. The work in this thesis utilised a range of approaches to achieve this aim. Initially, the association between muscle activation and rate of force development and EMD was established. Comparison of unilateral and bilateral actions was then undertaken. Finally interventions with the aim to both negatively affect and improve muscle strength, which included fatigue and resistance training (RT), respectively was undertaken and the neural contributions to changes in performance established. Agonist activation during the early phase of voluntary force production was shown to be an important determinant of voluntary EMD, explaining 41% of its inter-individual variability. Agonist activation was an important determinant of early, but not late phase RFD. Use of bilateral actions resulted in a reduction in explosive strength, which was thought to be due to differences in postural stability between unilateral and bilateral strength tasks. The level of stabiliser activation was strongly related to the level of agonist activation during the early phase of explosive force development and had a high association with explosive force production. Task-specific adaptations following isoinertial RT, specifically, the greater increase in isoinertial lifting strength than maximal isometric strength were due to training-specific changes in the level of agonist activation. High-intensity fatigue achieved a more substantial decline in explosive than maximal isometric strength, and this was postulated to be due to neural mechanisms, specifically decreased agonist activation. This work provides an in depth analysis of the neural contributions to maximal muscle performance.

Key Words: Rate of force development, explosive strength, strength training neural activation, electromechanical delay, electromyography, fatigue, muscle coordination, contractile properties.

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Buckthorpe MW, Hannah R, Pain MTG, Folland JP (2012). Reliability of Neuromuscular Measurements during Explosive Isometric Contractions, with Special Reference to EMG Normalisation Techniques. *Muscle & Nerve*, 46, 566-576.

Buckthorpe MW, Pain MTG & Folland JP (2013). Bilateral Deficit in Explosive Force Production Is Not Caused by Changes in Agonist Neural Drive. *PLoS ONE* 8(3): e57549. doi:10.1371/journal.pone.0057549.

Folland JP, **Buckthorpe MW** & Hannah R (2013). Human capacity for explosive force production: Neural and contractile determinants. *Scandinavian Journal of Medicine & Science in Sports.* DOI: 10.1111/sms.12131

Buckthorpe MW, Pain MTG, Folland JP (In Press). Central fatigue contributes to the greater reductions in explosive than maximum strength with high intensity fatigue. *Experimental Physiology.*

Buckthorpe MW, Erskine R, Fletcher G & Folland JP (In Press). Neural adaptations explain the task specificity of strength changes after resistance training. *Scandinavian Journal of Medicine & Science in Sports.*

Further work associated with the thesis:

Hannah R, Minshull C, **Buckthorpe MW** & Folland JP (2012). Explosive neuromuscular performance of males versus females. *Experimental Physiology*, 97 (5), 618-29.

PRESENTATIONS AND PUBLISHED ABSTRACTS

Folland JP, **Buckthorpe MW** & Hannah R (2011). Neural & Contractile Determinants of Explosive Force Production. Medicine & Science in Sports & Exercise 43 (5), 101. American College of Sports Medicine, Denver, Colorado, 2011.

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Buckthorpe M, Pain MTG & Folland JP (2012.) A bilateral deficit in explosive, but not maximal force production. *In The Biomedical Basis of Elite Performance, Queen Elizabeth the 2nd Conference Centre*, London, pp.0-0.

Buckthorpe MW, Pain MTG, Folland JP (2012). Fatigue induces a greater decline in explosive than maximal force production with evidence for neural and contractile mechanisms. In *Proceedings of the 16th Annual Congress of the European College of Sport Science*, Bruges.

Conference Proceedings associated with this thesis:

Hannah R, **Buckthorpe MW**, Minshull C & Folland JP (2010). Explosive neuromuscular performance of the knee extensors in untrained males and females. *Journal of Sports Sciences*, 29 (2), S78. British Association of Sport & Exercise Sciences, Essex.

Hannah R, Minshull C, **Buckthorpe MW** & Folland JP (2011). Muscle-tendon unit stiffness does not independently influence voluntary explosive strength. In Proceedings of the 15th Congress of the European College of Sport Sciences, Liverpool.

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CHAPTER 1

Introduction

Movement is achieved through the production of muscle force acting about a bone support system. It is the amount ofmuscle force and duration over which this is maintained which governs the change in velocity and resultant displacement of a body of mass and thus, resultant movement. Furthermore, active stabilisation of joints via muscular actions allows for the maintainance of posture and prevention of joint injury. The ability of the neuromuscular system to produce force is defined as strength (Siff, 2001). There are different types of muscle strength; the ability of the neuromuscular system to produce its maximal voluntary force (MVF) irrespective of any time domain, termed maximal strength; and the ability to develop force rapidly (typically defined as the rate of force development, RFD), termed rate of force development (RFD) or explosive force production. RFD is considered functionally more important than MVF production during certain explosive functional movements, such as sprinting or re-stabilizing the body following a loss of balance (Aagaard et al., 2002a; de Ruiter et al., 2004; Tillin et al., 2010, 2013). These movements involve contraction times which are significantly shorter than the time available for the development of MVF (typically in the order of more than 300 ms, Thorstennson et al., 1976). For example, ground contact time during sprint running is typically in the order of 80-120 ms (Kuitunen et al., 2002), whilst recent evidence suggests that injuries such as an anterior cruciate ligament rupture occur as early as 50 ms after ground contact (Krosshaug et al., 2007), and therefore, limited time available for force production to re-stabilise joint complexes and avoid injury as the result of mechanical perturbation.

The electromechanical delay (EMD) represents the time delay between the onset of neuromuscular activation and the initiation of force production. It represents an important aspect of neuromuscular reaction time, during which there could be unrestrained development of forces of sufficient magnitude to damage ligamentous tissue in synovial joints (Huston and Wojtys 1996; Mercer et al. 1998; Shultz et al. 2001). Thereby, a short EMD as well as good explosive force production capabilities are important aspects of explosive neuromuscular performance, which are considered to be essential for the performance of explosive sporting actions as well as the prevention of sports injuries (Aagaard et al., 2002a; Minshull et al., 2007; Tillin et al., 2010, 2012a). Despite a body of evidence documenting the association of explosive strength on explosive dynamic muscle performance such as sprint running (Tillin et al., 2013) and vertical jump performance (de Ruiter et al., 2007; Tillin et al., 2013), there is little evidence supporting an association between explosive neuromuscular capabilities and injury risk. Understanding the factors which influence the expression of explosive

neuromuscular performance, and considering how the capability for explosive neuromuscular performance may impact on injury risk are important research topics to improve our understanding of how to prevent injuries.

Sale (1988) likened the expression of voluntary strength to a skilled act, where agonists must be optimally activated, while supported by appropriate synergist and stabiliser activation and opposed by minimal antagonist activation. It is not fully understood how agonist and antagonist activation influence the expression of strength. Furthermore, there is a paucity of research examining the influence of stabiliser muscle activation on muscle strength and thus, relatively little known on the topic.

The broad theme of this PhD was to consider the neural contributions to maximal muscle performance, with a focus on explosive neuromuscular performance indicators (EMD, RFD), and maximal muscle strength. There are different approaches one may take to understand the neural contributions to muscle strength. The first is to assess muscle strength and neural activation across a range of individuals and assess their inter-relations in order to determine the proportion of shared variance between measures of performance. Other investigational approaches include interventions which manipulate force through various approaches and document the concurrent changes in neural and/or morphological parameters. This can be investigated on a group and/or individual level. The thesis utilised a range of approaches to enhance our understanding of the neural contributions to maximal muscle performance.

Although explosive strength appears to be essential for sports performance and injury risk, there is little documented evidence of its reliability. Therefore the initial aim of the thesis was to investigate the reliability of neuromuscular measurements during explosive isometric muscular actions.

The exact contributions of neural mechanisms such as the level of agonist activation to EMD are not known. Voluntary EMD of the quadriceps has been shown to be 100% (16-25 ms) longer than electrically-evoked EMD (Zhou et al., 1996; Minshull et al., 2007), and this suggests a significant neural component to the delay. Chapter 4 investigated the contribution of agonist activation on the variability in EMD. This was determined by comparing two groups for EMD performance, and documenting the underlying neural and contractile mechanisms which differed between these groups. Neural and contractile performance indicators were related to EMD performance to establish the shared variance between measures.

Bilateral deficit (BLD) has been used to describe the phenomenon of a reduction in performance during synchronous bilateral (BL) movements when compared against the sum of identical unilateral (UL) movements. BLD is considered to be due to neural mechanisms, although the exact mechanisms are not fully understood. Although there has been considerable research attention into the BLD for MVF, there is little research into explosive force production, and currently no documented evidence that there is a neural deficit during BL explosive isometric efforts. Agonist activation is considered a major determinant of RFD, and therefore more substantial reductions in RFD could be expected during BL explosive tasks. If the addition of an extra limb causes a substantial reduction in explosive neuromuscular performance, even for relatively simple isometric single joint tasks, then it would suggest that there could be much more substantial reductions in neural activation for explosive force development during more complex tasks, which involve more complex motor control, across multiple joints and consisting of a high number of degrees of freedom in movement. Thereby, understanding the influence of BL actions of explosive force production would serve as an important stepping stone to unravelling the neural contributions to movement and joint stabilisation.

Muscles may take up differing roles, depending on the required joint motion. An optimal level of stabiliser activation (that is muscles acting as stabilisers to fixate joints, in order to allow allow agonists to appropriately function) is thought to be important for muscle performance (Sale, 1988; Folland & Williams, 2007a). However, there is an incredible lack of research assessing the influence of muscle stabiliser activation on movement quality. No study has examined if the variability in stabiliser activation between individuals influences the expression of strength. Furthermore, it is unsure how the levels of agonist, antagonist and stabiliser activation inter-relate with one another during explosive contractions. Chapter 6 assessed the level of agonist, antagonist and stabiliser muscle activation across the rising force-time curve during explosive isometric contractions and assessed the independent influence of each muscle group on the expression of explosive strength and further documented the inter-relationship of agonist, antagonist and stabiliser muscle activation.

An increase in strength due to neural mechanisms demonstrates that muscle activation patterns pre-training were sub-optimal and provides evidence for the contributions of neural factors to the expression of muscle strength. Resistance training (RT) is typically undertaken by athletes with the primary goal to enhance neuromuscular performance, and consequently improve athletic performance and reduce injury risk. Marked increases in strength during the

early phase of a training programme have been observed, and are thought to be due to neural enhancements (Aagaard et al., 2002a; Del Balso & Cafarelli, 2007; Cannon et al., 2007; Tillin et al., 2011). Most research has documented changes in isometric strength which involved constrained situations. Although, isometric measures of neuromuscular performance allow for a more precise delineation of the neural contributions to muscle strength and as such a more accurate interpretation of the contributions changes in in neural factors make to mechanical measures of muscle performance following an intervention, these tasks are inherently simpler and as such likely reduce the potential contribution of enhanced neural mechanisms following training could make on measured performance. Changes in strength appear highly specific to the nature of the training task. For example, conventional dynamic isoinertial RT has repeatedly been found to produce disproportionately greater increases in isoinertial lifting strength than isometric strength (Thorstensson et al., 1976, Rutherford & Jones, 1986). This specificity of training phenomenon is often taken as strong indirect evidence for neural adaptations to RT (Folland & Williams, 2007a). However, there is no actual evidence to indicate neural mechanisms are responsible for this task specific phenomenon. Functional tasks generally require isoinertial strength and thus establishing the neural contributions to changes in isoinertial strength following RT would seem important, but has received minimal research attention. Chapter 7 assessed the task specific adaptations in isometric (maximum and explosive) and isoinertial strength following short term (three weeks) isoinertial RT and documented the concurrent neural changes in agonist, antagonist and stabiliser activation.

Evoking reductions in force capabilties and establishing the mechanisms responsible for the deficits in force is an alternative method of investigating the neural contributions to explosive neuromuscular performance. Fatigue can be defined as a temporaray reduction in muscle strength following muscular efforts, which largely recovers after a period of rest and declines in neuromuscular performance as a result of fatigue is thought to be a conbtributory risk factor for sports related injuries (Hawkins et al. 2001) and therefore is a topic of considerable interest to the sporting community. However, most of the research investigating the influence of fatigue on the functional capacity of the neuromuscular system has focused on the decline in MVF, with little focus on explosive neuromuscular performance. As explosive neuromuscular performance is thought to be a component of injury risk, understanding how fatigue may achieve a reduction in explosive neuromuscular performance is an important research topic, which has yet to be established. Chapter 8 of this thesis documented the

influence of a fatiguing exercise protocol on EMD and explosive force and contrasted these changes with the decline in MVF during the same muscular actions throughout a fatiguing protocol. The neural and contractile contributions to impaired performance were concurrently assessed to establish their contribution to the decline in muscle strength.

The purpose of the thesis was to assess the influence of neural factors (agonist, antagonist and stabiliser activation) on maximal muscle performance, specifically on explosive neuromuscular performance. This was done by examining the relationship of agonist activation on EMD and agonist, antagonist and stabiliser EMG explosive force production throughout the rising force-time curve, as well as investigating the influence of bilateral contractions, RT and fatigue on explosive neuromuscular performance and establishing the role of neural factors to changes in muscle performance.

CHAPTER 2

Literature Review

The first section will provide a brief overview of the historical background of the area of sport and exercise science. After this, the review will present important information on the process of voluntary force production and consider the peripheral, neural and mechanical factors which influence the expression of voluntary force. The review will then present issues concerning measurement of explosive neuromuscular performance (section 2.2) before considering its association with performance of explosive sporting tasks and injury risk (section 2.3). Section 2.4 will consider the determinants of maximal and explosive strength and EMD, whilst the review will finish with consideration of the influence of fatigue on explosive neuromuscular performance and associated peripheral and neural mechanisms.

2.1 Roots and Historical Perspective of Exercise Physiology as a Field

Before we critically review the available literature present today, it is important to consider the development of the field of sport and exercise science as a whole, and in particular exercise physiology and acknowledge the contributions of reseachers in the development of the field. An initial question could be how old is the field of Sport and Exercise Science? The formal answer might be that of 40 years, since the formal linkage in the united kingdom of 'science' to 'sport and exercise' in the mid 1970s with the introduction of degree standard study (Winter, 2008). However, although not formally under the title of 'Sport and Exercise Scientist', the origins of the practice of the discipline can be considered to range as far back of the ancient times, to the great works of Galen and Hipprocrates. Like Hippocrate, Galen had an interest in sport and exercise, and was appointed as physician to the gladiator school in Pergamum by Roman Emperor Marcus Auerlius (A.D. 121-180, Winter & Fowler, 2009). Galen was a pioneer of the field of physiology, who implemented the enhanced current thinking about health and hygiene, which can be considered 'applied' exercise physiology. Hippocrates similarly to Galen, was appointed by the state as physicans to aid the welfare of athletes of his time. They both essentially developed the first sports medicine and science institutes, which can be considered similar to that available today. Although, we are now driven by scientific approach to the study of sport and exercise science, through multidisciplinary teams surrounding the athletes, our science and medicine institutes can be considered a reinterpretation of what was available nearly 2000 years ago (McArdle et al., 2007; Winter, 2008). Fast forward through time and the development of the field was the result of a strong relationshiop between the classically trained physician, academically based anatomists and physiologists and emergence of physical educators who struggled to gain the

credibility of the field as a whole. Finally, that the application of scientific method to the sport and exercise was characterised by the research of Hill in the 1960 and 70s.

2.2 Voluntary Force Production

2.2.1 Skeletal Muscle Structure

The main function of skeletal muscle is to produce force and act on the bone to which the muscle is attached to either stabilise joint complexes and thereby protect bony structures, maintain posture or joint position and enable movement. The word muscle derives from the latin musculus, a diminutive mouse, due to the way in which active muscle bears close resemblance to that of mice running underneath the skin. Human bodies are thought to contain 434 skeletal muscles. Each single skeletal muscle is composed of two main components: specialised contracting cells, myofibers, and a connective tissue framework formed by fibroblasts. Each myofiber is composed of myofilaments (comprising the contractile proteins, actin and myosin, see Figure 1) and a variety of structural proteins, all arranged in a regular configuration throughout the length of the myofibril, so as to form a series of contractile components, or sarcomeres. Each muscle fibre is surrounded by its own basal lamina, bordering directly on the endomysial connective tissue. The cell membrane of the muscle fibre possesses all the characteristics of cell membranes in general in which they have the capability to create and maintain a membrane potential which is vital to normal contractile function. The sarcoplasmic reticulum (SR) is the endoplasmic reticulum of the muscle fibre and functions as a calcium store. Transverse tubules or T-tubules are invaginations of the muscle cell membrane and run perpendicular to the muscle fibres, and along with the SR are integral to the contractile functioning of the muscle. Thousands of muscle fibres, each individually wrapped in a thin layer of connective tissue, the endomysium, are grouped together to form muscle fascicles. A layer of connective tissue, named perimysium surrounds each fascicle. Finally, a number of muscle fascicles are grouped together by another connective tissue sheath named epimysium. According to the sliding filament theory (Huxley, 1957), the interaction between the thick (myosin) and thin (actin) filaments causes the sarcomere and therefore the muscle fibre to contract and produce force. There are significant amounts of connective tissue in muscle (fascia, epimysium, perimysium, endomysium). The amount of connective tissue in muscle is ~ 13 % and this remains constant even with muscle hypertrophy (MacDougall et al., 1984). The component of connective

tissue which dictates its properties is collagen, although there is also elastin. Although, a main role of the connective tissue is to envelope, and thus forms skeletal muscle, tension in skeletal muscle fibres is transmitted to the skeletal via this connective tissue, at the ends of the muscle as it forms a tendon to essentially link muscle with bone. Therefore, when considering muscle and tendon, it is best to consider them as a single functional unit (i.e., muscle tendon unit, MTU).
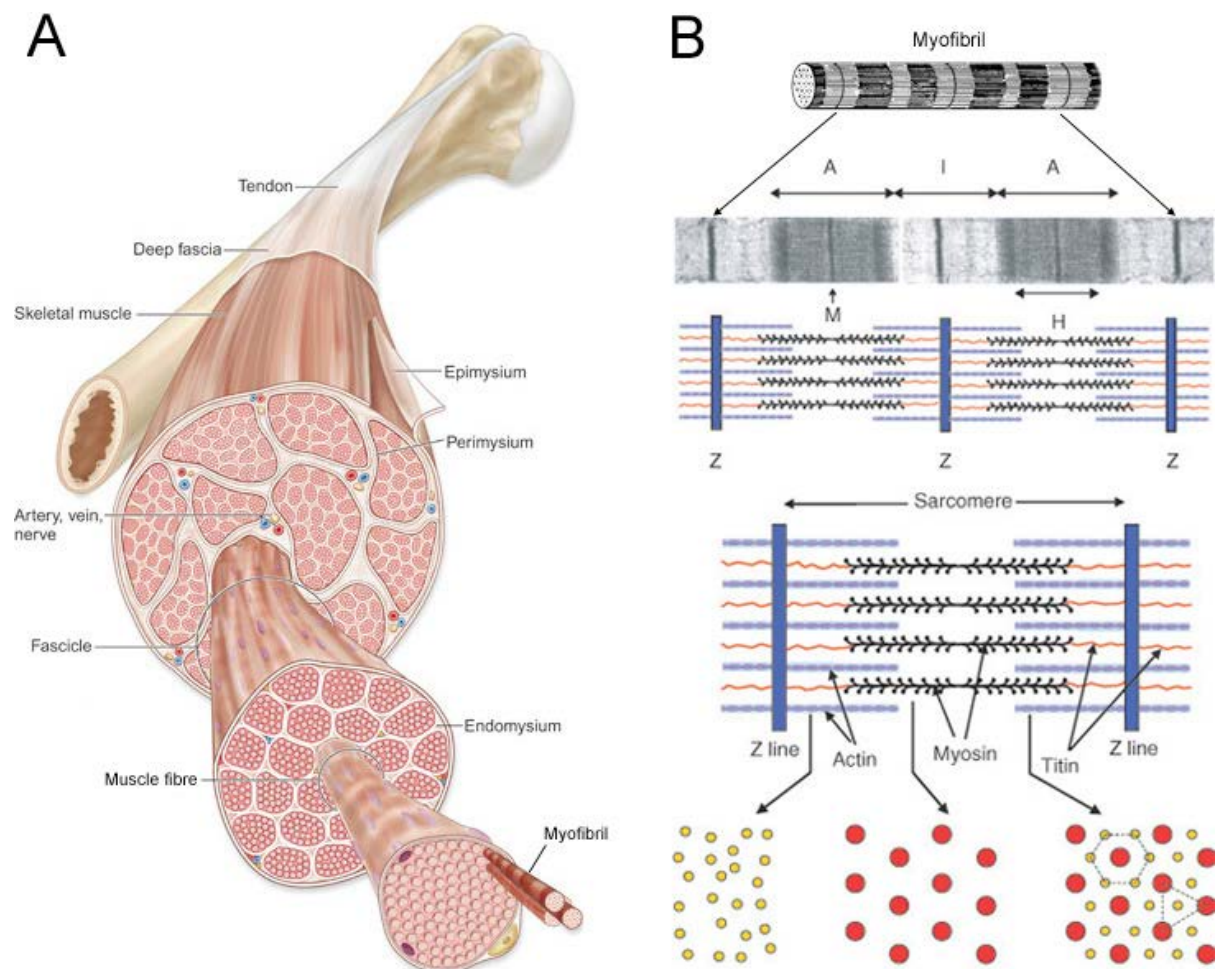
Figure 2.1 An overview of skeletal muscle structure from whole muscle to individual myofibrils (A) and of the muscle sarcomere and muscle proteins (B). Adapted from Jones et al., 2004).

2.2.2 Muscle Fibre Type Classifications

Skeletal muscle is composed of different types of muscle fibre with contrasting contractile and metabolic properties. Human skeletal muscle fibres can be characterised as slow twitch (type I) and fast twitch (type IIa or type IIx). At present, muscle fibre types are typically characterised according to their myosin heavy chain (MHC) isoform (I, IIa or IIx) and the greater muscle ATPase activity of muscle fibres containing purely MHC IIx allows them to contract faster than fibres comprising only MHC IIa, which in turn have faster contractile properties than pure MHC type I fibres (Bottinelli et al., 1996; Li & Larsson, 1996; Bottinelli & Reggiani, 2000; D'Antona et al., 2006; Degens & Larsson, 2007). Consequently, type II fibres can produce more force at a given velocity than type I fibres and therefore have a higher power output than type I fibres. The specific tension of the three different types of muscle fibres is a matter of debate however, in which some studies have reported higher specific tension of type IIa and IIx fibres (Bottinelli et al., 1996; D'Antona et al., 2006; Pansarasa et al., 2009), whereas others have not (Larsson & Moss, 1993; Ottenheijm et al., 2000; Gillier et al., 2009). Type I fibres are more oxidative than type IIa fibres, which in turn are more oxidative than type IIx fibres which primarily rely upon glycolysis for muscle ATP production. This can be seen as a logical consequence of the hierarchical order of recruitment, which will be discussed in section 2.1.6.

2.2.3 Muscle Architecture

Skeletal muscle can be characterised according to its alignment of the fibres relative to the tendon and aponeurosis. Essentially muscles can be grouped into two major types of fibre arrangements: parallel and pennate. Parallel muscles have their fibres arranged parallel to the length of the muscle. Generally, parallel fibres will produce a greater range of motion as they are equipped for greater muscle shortening. Pennate muscles have shorter fibres that are arranged obliquely to their tendon and aponeurosis. The pennation angle and muscle fibre lengths differ between muscles (Alexander & Vernon, 1975) and for the same muscle between individuals (Kawakami et al., 2006). It is possible using ultrasound to assess muscle fascicle angle of pennation of human muscles *in-vivo* (Kawakami et al., 1993).

2.2.4 Activation of Skeletal Muscle

A muscle is innervated by a number of motoneurons and each motoneuron innervates a specific group of muscle fibres. All the individual muscle fibres that are innervated by the same motoneuron form a motor unit, which will differ in size and type, according to the total number of fibres innervated and the contractile characteristics of those fibres. Each motoneuron innervates from as few as ten to up to possibly several thousand muscle fibres, referred to as small and large motor units, respectively. Correct timing and smooth and targeted execution of a movement is the result of close cooperation between sensory and motor systems. Although, the final executors are the muscles, muscles are referred to as the motoneurons 'slaves' as rested muscle obeys their commanding motoneurons completely. Each time a group of motoneurons launches their impulse trains, the corresponding muscle fibres respond in a predictable manner.

The motoneurons are responsible for the ultimate command of muscle contraction. There is considerable debate surrounding the term 'muscle contraction'. Although, commonly used terminology, it is important to note that the muscle when activated does not actually 'contract' (see Winter & Fowler, 2009). However, for the purpose of simplicity the commonly adopted term of muscle contraction will be used within the thesis, with appreciation of the debate concerning this use. Back to the process of muscle activation, irrespective of whether a reflex or voluntary contraction is performed, motor commands to the muscles have to be conveyed by motoneurons, earning for the latter the name "final common path". In turn motoneurons are influenced from several sources, including afferents from the periphery and descending tracts from supraspinal levels as well as local spinal circuitry. The term supraspinal alludes to a hierarchical organisation of motor control and comprises all areas of the central nervous system (CNS) that contribute to motor control, but have to do it through their influence on motoneurons only. Supraspinal motor areas of the CNS include the motor area of the cerebral cortex, the cerebellum, and various nuclei in the brain and brain stem. Some of these give rise to descending tracts that affect the motoneurons directly or- more often-indirectly through interneurons (Ästrand et al., 2003).

From the skeletal muscles afferent nerves report to the CNS about information on the state of the muscle, including tension, length, position and about changes in this state of muscle. These nerves are activated by special receptors, one of which is the muscle spindle. The muscle spindle is a sensory organ, and consists of intrafusal and extrafusal muscle fibres

which provides the CNS with information concerning muscle stretch. Golgi tendon organs are connected in series within extrafusal muscle fibres and insert between the muscle and tendon. Their primary role is to provide information concerning the active tension of skeletal muscle, and when stimulated, the afferent fibres have been found to cause neural inhibition of the corresponding muscle groups. Within joints, the ligaments and joint capsule contain different kinds of receptor and can provide information concerning joint position and movement of joints (Ästrand et al., 2003).

When a motoneuron is activated, a single electrical impulse (action potential) will be passed down the axonal branches to the neuromuscular junction. The action potential is caused by movements of Na^+ into the cell and K^+ out of the cell. The neuromuscular junction appears to provide uniform 1:1 action potential transmission under physiological conditions and therefore once activated always results in the release of acetylcholine from the synapse and depolorarisation of the sarcolemma (the muscle fibre plasma membrane). This is achieved via interaction of acetylcholine with its receptors on the post-synaptic membrane. The action potential is actively conducted down the transverse tubules (t-tubules) into the interior of the muscle. T-tubular membrane expresses high levels of L-type Ca^{2+} channels (or dihydopyradine receptors, DHPRs, voltage sensors) which change their conformation with an action potential, and result in charge movement. These voltage sensors are in close contact with the SR Ca^{2+} release channels (ryanodine receptors, RyR) and when activated result in a release of Ca^{2+} from the SR. Ca^{2+} release by SR gives rise to transient increase in myoplasmic free Ca^{2+} which binds to tropinin C on the actin filament, instigates movement of tropomyosin and exposes the myosin binding sites. The myosin heads then attach to the actin filament and, with the hydrolysis of adenosine tri-phosphate (ATP), the cross bridge power stroke occurs, drawing the Z-lines of the sarcomere closer together, causing the muscle to shorten. This whole process is called excitation-contraction (E-C) coupling, as the excitation by neural stimulation is coupled to the resulting muscle action. Finally, the muscle relaxes as elevated Ca^{2+} is pumped back into the SR by ATP driven SR Ca^{2+} pumps.

2.2.5 Mechanical Factors Influencing Force Production

The mechanical properties of skeletal muscle determine its performance. Mechanical properties will be defined and those properties of skeletal muscle that can be measured by parameters derived from mechanics: force, length, velocity and power. As will be discussed later in the thesis sports performance and joint stabilisation are determined by the ability of

the human neuromuscular system to apply force, and this application of force is determined by a number of factors which will be discussed herein. The mechanical properties to be discussed here include the force-length relationship and force-velocity relationship. The power-velocity relationship is an important mechanical factor influencing muscle performance, but will not be reviewed here as power is not assessed within this thesis. Furthemore, time dependent properties such as force depression, force enhancement and post-activation potentiation will also not be reviewed in this thesis. A large review of endurance time stress relationship (influence of fatigue) will be presented later in the thesis (section 2.7).

2.2.5.1 Force-length Relationship

The ability for muscle to develop force is critically dependent upon muscle length. The most basic form, the length tension relationship reflects the fact that tension generation in skeletal muscle is a direct function of magnitude of overlap between actin and myosin filaments. The greatest potential for force production occurs when the cross-bridges are formed with optimal overlap between actin and myosin filaments (optimal length). At this length cross-bridge interaction is maximal. When sarcomere lengths shorten below optimal, force production is impaired due to overlap between actin filaments from opposite ends of the sarcomere, whereas at muscle length longer than optimal force production is reduced due to less overlap between actin and myosin filaments (Gordon et al., 1966; Edman, 1966).

2.2.5.2 Force-velocity Relationship

There are three types of muscular contraction concentric, the muscle shortens under tension; isometric, the muscle is under tension but does not change length; and eccentric, the muscle lengthens under tension. The influence of type of contraction on force production can be described by examining the force-velocity relationship. Experimentally the force-velocity relationship like the force-length relationship is a curve that represents the results of many measurements plotted on the same graph. In short, the *in-vitro* tetanic relationship shows concentric force lower than isometric force, with an hyperbolic decay in force with increasing velocity during concentric muscle contraction (Hill, 1938). As the velocity of muscle action increases less force is capable of being generated during that contraction. This can be explained due to actin-myosin cross-bridge cycling. As it takes time for filaments to attach and detach, as filaments slide pass one another faster and faster, force decreases, as there is a lower number of cross bridges attached. As force generation of the muscle is dependent upon

the number of actin-myosin cross-bridges, force production decreases as velocity increases. The cross-bridge theory which so eloquently describes the force-velocity relationship for concentric actions does not hold true for eccentric actions. The eccentric phase of the tetanic force-velocity relationship shows absolute tension that is greater than the maximal tetanic isometric tension and relatively independent of velocity, with a plateau at approximately 1.5-1.9 times isometric maximal tetanic force (Hill 1938; Edman et al., 1978; Harry et al., 1990). Although the force-velocity relationship was first defined in frog muscle (Hill, 1938), all human movement is fundamental limited by this muscle property (Thorstennson et al., 1976; Pain & Forrester, 2009). *In-vivo* the force-velocity relationship can be assessed by examining the force production of a joint through full of motion on an isokinetic machine at varying velocities and plotting the subsequent peak force output. The measurement *in-vivo* is more complicated than *in-vitro*, as muscles contain mixed fibre types (Faulkner et al., 1986), have different architectural characteristics (Wickiewicz et al., 1984; Herbert & Gandevia, 1995), and can be influenced by the level of voluntary neuromuscular activation (Pain & Forrester, 2009). In human's *in-vivo*, during MVCs the force-velocity relationship differs to that measured *in-vitro*, whereby eccentric maximum force is typically only 90-110% of isometric maximum force (Dudley et al., 1990; Pain & Forrester 2009).

2.2.6 Neural Aspects of Muscle Force Production

Motor unit recruitment and firing frequency represent two more or less parallel mechanisms of force regulation at the whole muscle level. Motor units are recruited in a systematic order during voluntary actions of increasing magnitude according to the size principle (Henneman et al., 1974). The increase in muscle force, with the activation of additional motor units, depends on the number of muscle fibres within that motor unit. Relatively small motor units that innervate type I fibres are initially activated at low force levels, whilst progressively larger α-motoneurons that innervate type IIa and IIx fibres are typically recruited after the slow twitch motor units at higher thresholds of force. The maximum force capabilities of motor units have been reported to vary up to 50 times (Enoka, 1995). Thus, the level of force produced during movement is largely influenced by how many motors, and specifically which motor units are activated.

Muscle force production is also influenced by the firing frequency of individual motor units. The motor unit firing frequency (MUFF) represents the rate of neural impulses delivered from the motoneuron to the muscle fibres. The effect of firing frequency can be understood

by examining the force-frequency relationship. A single action potential will cause the muscle to contract and then quickly relax, referred to as a twitch contraction (Enoka 2008). A single twitch represents the smallest contractile response, and usually does not occur in the development of human motion. If a second electrical impulse is delivered before the muscle has been allowed to completely relax, then the force output will be a summation of the two impulses. This summation of force, which will continue as long as consecutive impulses are elicited at a frequency that does not allow the muscle to relax, will eventually plateau, known as a tetanic contraction. The amplitude of the tetanus will depend on the frequency at which impulses are elicited (firing frequency), with higher firing frequencies inducing greater tetanic peak forces. The force-frequency relationship is sigmoidal, in that at low firing frequencies, small increments in the stimulation frequency result in large increases in force, but for firing frequencies above 40 Hz, much greater increments in frequency are required to produce relatively small increases in force (Figure 2.2). However, the specific shape of the curve will depend on the contractile speed of the motor unit. Due to the time characteristics of slow and fast motor units (fast motor units have much quicker contraction and relaxation time), slow twitch units will summate individual force impulses more readily than fast twitch units. Therefore, the activation required for the production of half or maximum force is usually less for slow than fast-twitch muscle fibres (Kernell et al., 1983; Botterman et al., 1986).
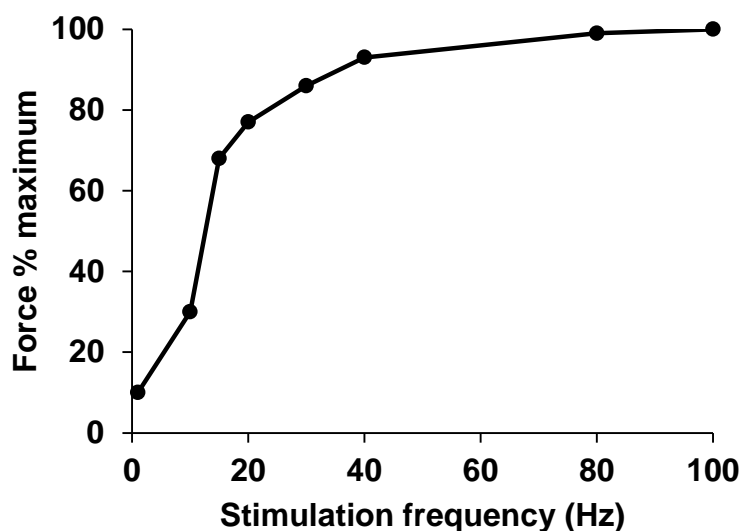
Figure 2.2 Force-frequency relationship recorded during electrically evoked contractions of the pollicis nerve. Force is reported as a percentage of maximum (Adapted from de Ruiter et al., 1999).

MUFF may not only influence contractile peak force, but also the RFD observed for whole muscle *in-vivo* (Grimby et al., 1981; Nelson et al., 1996). The MUFF at the onset of an explosive isometric contraction can be much higher than during the stable segment of a maximum voluntary contraction (i.e., 100-200 Hz vs. 20-30 Hz, Monster & Chan, 1977; Kukulka & Clamann, 1981; Bellemare et al., 1983; Van Cutsem et al., 1998). de Ruiter et al. (1999) reported that *in-vivo* muscle peak RFD of the adductor pollicis elicited via percutaneous electrical stimulation of the adductor ulnar nerve, increased with increasing firing frequency, with an initially steep increase in RFD at low firing frequencies (2-50 Hz), and a more gradual increase in RFD at higher firing frequencies (Figure 2.3). The muscle pRFD was achieved at 300 Hz. Thus, very high firing frequencies are required for explosive but not MVF production. Additionally, the frequency of the first impulses in an impulse train is particularly important, since an initial high-frequency doublet or triplet results in contractile force that is higher than would be expected from the frequency of the rest of the train of impulses. This is known as catch property of muscle (Lee et al., 1999) and likely serves to increase maximum RFD.
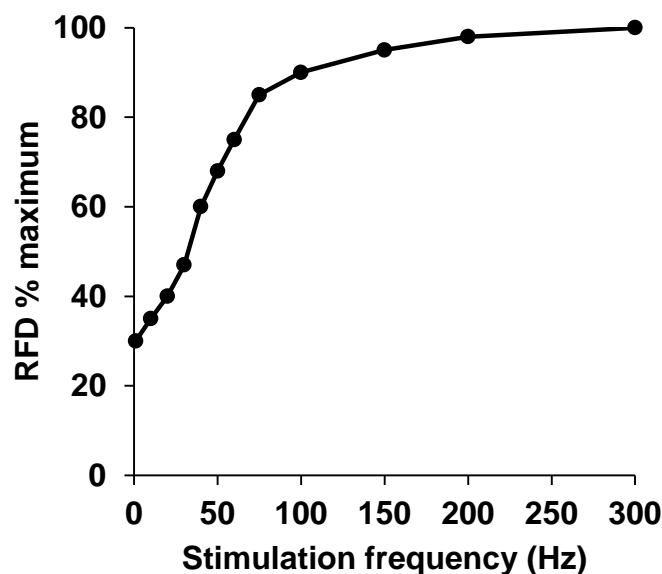
Figure 2.3 RFD-frequency relationship during electrically evoked contractions. RFD is denoted as a percentage of maximum (adapted from de Ruiter et al., 1999).

2.3 Measuring Maximal and Explosive Neuromuscular Performance

2.3.1 Maximal Strength

Muscle strength is specific to the muscle group(s) and situation in which it is measured. Muscle strength can be measured isoinertially (lifting), isometrically or isokinetically (Enoka, 2002). Maximum strength measured using an isometric or isokinetic dynamometer is commonly reported as the peak force/torque achieved over the course of a series of maximal contractions. There is inherent variability in the production of force and therefore, multiple maximal efforts (typically 3-4 attempts) are performed during a testing session to ascertain a participant's true MVF. MVF is typically defined as either the peak instantaneous force achieved during a measurement session (e.g., Tillin et al., 2010; Hannah et al., 2012, Figure 2.4) or as the highest mean force over a given period time during the MVC (i.e., average force over 500 ms). Isoinertial strength is commonly measured as the maximum weight that can lifted during a lift, named one repetition maximum (1RM) and reported as an absolute value or relative to body mass.
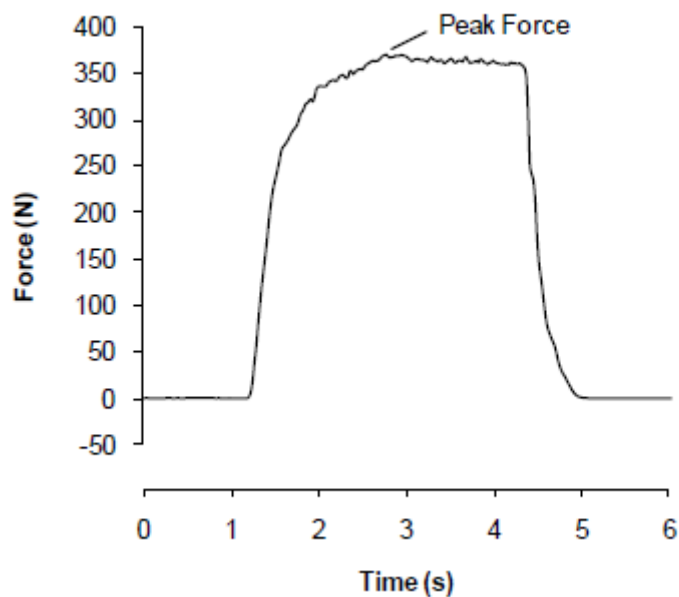
Figure 2.4 A force time curve recorded during an isometric maximum voluntary contraction of the knee extensors.

2.3.2 Explosive Neuromuscular Performance

EMD is commonly considered the time difference between muscle activation (typically assessed using surface EMG) and force onset (Zhou et al., 1996, Figure 2.5). This delay essentially encompasses the time between the muscle being 'turned on' and the actual time force can be initially recorded. If the researcher is concerned with the electromechanical delay (and not the delay in processing time, transmission of neurons down the synapse, the time between the motor end plate receiving the signal and the transmission throughout the sarcomlemma then the more commonly investigated method is to measure onset of muscle activation with surface EMG and then the recorded force with either a strain gauge or force plate (e.g., Tillin et al., 2010). EMD has been suggested to be due to several neuromechanical processes, specifically the time involved in: the propagation of the action potentials along the muscle fibre membrane; excitation contraction-coupling; and the stretching of the series elastic component (SEC) by the contractile component (Cavanagh & Komi, 1979). Estimates of EMD from force and EMG measurements during voluntary contractions range from ~16 – 50 ms for a range of lower limb muscles (Zhou et al. 1995b; Kubo et al. 2001; Minshull et al. 2007; Tillin et al. 2010; Wu et al. 2010). EMD can also be elicited through evoked contractions from electrical stimulation of efferent nerves as well as reflexively through electrical stimulation of peripheral afferent nerves (Zhou et al., 1995). The different methods as well as muscles investigated likely explains an aspect of the the discrepancies in recorded values between studies.
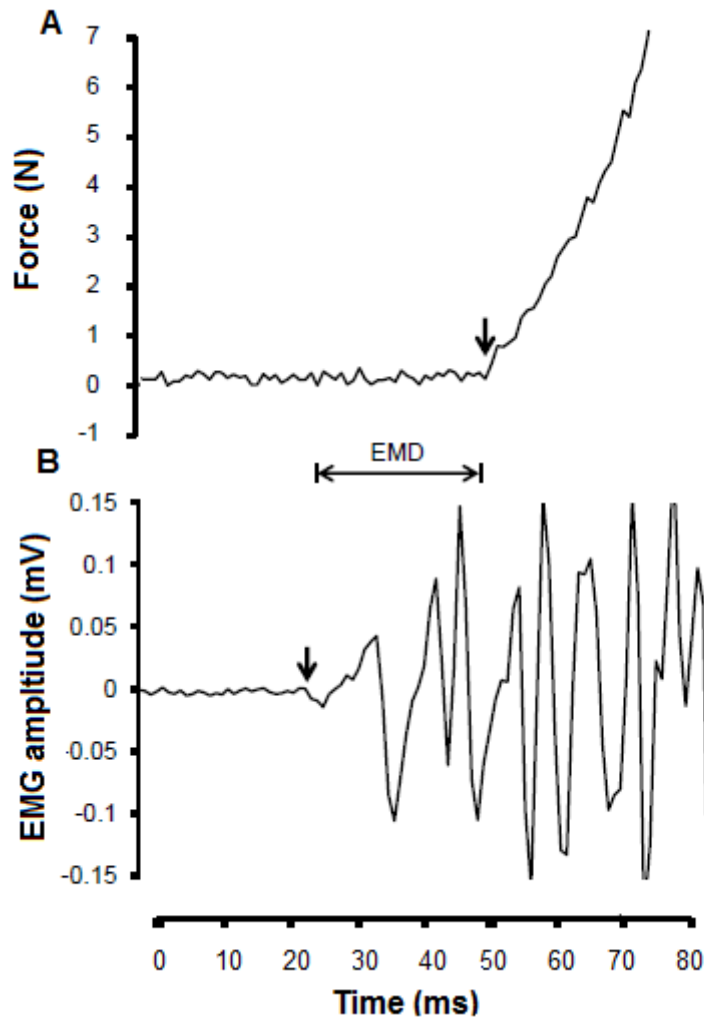
Figure 2.5 Force (A) and agonist EMG (B) during the early phase of an explosive isometric contraction of the knee extensors. EMD is reported as the time delay between onset of electromyographic activity and force, assessed through manual identification of the signals.

Explosive force is a measure of the capability to increase force from a low or resting level as quickly as possible. It is typically measured isometrically using isometric dynamometer (e.g. Aagaard et al., 2002a), isometric strength testing rig (e.g. Bosjen-Moller et al., 2005; de Ruiter et al., 2004; Barry et al., 2005) or on a force plate (e.g. Gruber & Gollhoffer et al., 2004; Nuzzo et al., 2008; Tillin et al., 2012). Some studies have attempted measurement of RFD during dynamic actions such as a vertical jump (Thorstennson et al., 2009; Tillin et al., 2012b), however, this situation is experimentally problematic, as the mechanics of the system interact with velocity in a non-linear manner. Based on Newtonian physics (force = mass × acceleration) and the force-velocity relationship (discussed above in section 2.1.5.2), an increase in force during the early phase of a dynamic action will result in an increase in

acceleration and therefore a higher velocity, which will then comprise potential force production during latter time points of the dynamic action, thereby influencing the observed RFD throughout the dynamic contraction.

Explosive neuromuscular force production is commonly quantified as the force (or torque) produced at specific time points from contraction onset (Tillin et al., 2010, Figure 2.6), or the RFD over a particular time period (i.e. the slope of the force time-curve; (Aagaard et al., 2002a; Barry et al., 2005)), or the force-time integral (area beneath the force-time curve, Aagaard et al., 2002a). RFD can be defined as the peak slope of the force-time curve, referred to as peak RFD (Jakobi & Cafarelli, 1998; Sahaly et al., 2001; Del Balso & Cafarelli, 2007). However, this reflects a single time point and does not account for variance in the time in which peak RFD (pRFD) actually occurs. Recently, assessing explosive force/RFD in distinct 50 ms time points/windows has been advocated to provide a clearer understanding of the underlying determinants of RFD (Tillin et al., 2010).
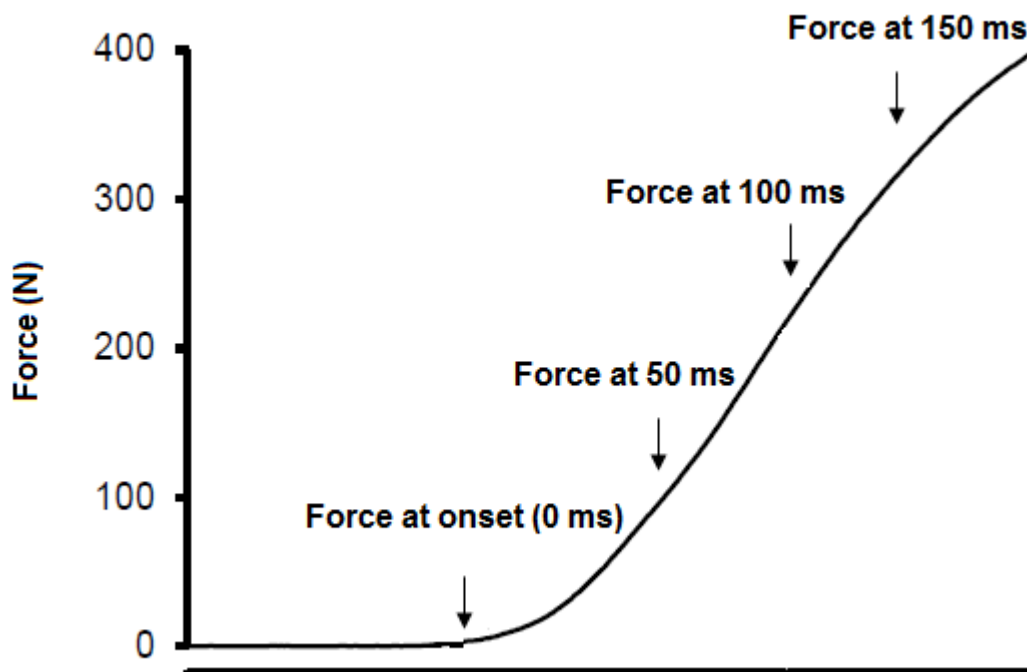
Figure 2.6 Force-time trace depicting the initial 150 ms of an explosive isometric contraction of the knee extensors.

There are certain methodological factors which need to be considered when assessing measures of explosive neuromuscular performance, namely both EMD and RFD. Firstly, Wilkie (1949) identified the influence of system compliance on muscle RFD in which RFD was shown to be lower when a compliant spring was introduced between participant's wrist extensors and the force transducer. Therefore, a compliant measurement system will likely reduce the initial force production and potentially lengthen EMD and reduce early phase RFD, but not necessarily influence MVF. Thus, a stiff measurement system is needed for accurate interpretation of isometric explosive neuromuscular performance. As previously described, force and velocity are interrelated, and therefore velocity can have a strong impact on subsequent force production. Consequently, explosive force production is typically measured isometrically. The identification of force and EMG onsets is pivotal to the valid and reliable assessment of EMD and explosive force. The majority of studies determining EMG and force onset for explosive contractions have used automated methods of onset such as absolute force thresholds (e.g., 7.5 Nm, Andersen & Aagaard, 2006) or mathematical algorithms (e.g. 3 standard deviations of mean baseline force, de Ruiter et al., 2004). However, manual (visual) identification of signal onsets is considered the "gold standard" method for identifying signal onsets (Staude, 2001; Allison, 2003; Moretti et al., 2003; Pain & Hibbs, 2007; Pulkovski et al., 2008) and has been shown to be able to identify signal onsets up to 60 ms earlier than automated methods (Allison 2003; Pain & Hibbs, 2007; Pulkovski et al., 2008). Therefore, automated detection methods are considerably less valid and relatively insensitive than manual (visual) identification procedures. Furthermore, the level of pre-tension in the muscle prior to performing an explosive contraction has been shown to subsequently affect the level of force that can be applied over the initial rising phase of contraction (de Ruiter et al., 2006) and so explosive contractions should be performed from a relaxed state (i.e. no pre-tension). Counter-movement (a drop in baseline force) exerts a strong influence on subsequent recorded values of explosive force production (Grabiner et al., 1994; Kamimura et al., 2009), and therefore a consistent baseline force should be ensured.

Although MVF has been documented widely to have excellent reliability (ICC > 0.95; coefficient of variation [CV] < 4%, Thorstensson et al., 1976; Strass, 1997; Kollmitzer et al., 1999; de Ruiter et al., 2004; Place et al., 2007) the between-session reliability of RFD has received less attention. The between-session reliability of RFD in the plantar flexors has been documented, but only in the early phase of the contraction (5- 40% MVF, Clark et al., 2007). Others have noted some reliability data for knee extensor RFD during intervention or

comparative studies (Clark et al., 2007; Place et al., 2007; Tillin et al., 2010) but there has not been a comprehensive attempt to assess the reliability of RFD measurements.

Whilst functional human movement involves the coordinated actions of multiple joints, given the number of technical considerations it can be seen why explosive force production has typically been assessed in isolated muscle groups such as knee extensors (e.g., Aagaard et al., 2002a; de Ruiter et al., 2004), ankle plantar flexors (e.g., Del Balso & Cafarelli, 2007) and elbow flexors (e.g., Barry et al. 2005). A few studies have assessed RFD in multiple joint situations such as an isometric squat (e.g., Nuzzo et al., 2008; Tillin, 2013) or leg press (Gruber & Gollhoffer, 2004). However, this situation is not ideal for understanding the underlying neural and morphological factors which influence the expression of explosive strength between participants. Therefore, measurement of isometric explosive strength in a single joint isometric situation minimises the potential number of confounding variables that can influence RFD measurement, and allows for a more controlled situation in which the determinants of RFD or peripheral and neural contributions to changes in explosive strength following an intervention can be more appropriately examined.

2.3.3 Measuring Neuromuscular Activation

Unfortunately, there is no gold standard technique for the measurement of neuromuscular activation. However, several methods have been used to measure neural activation and/ or motoneuron excitability. These include electromyography (EMG, intra-muscular and surface), the interpolated twitch technique (ITT), mechanomyograms, trans-cranial magnetic stimulation (TMS), magnetic resonance imaging (MRI) and measurement of V-waves and H-waves. This section will mainly review the two methods that have been most commonly used to quantify neural activation at MVF, and during explosive contractions; EMG and ITT. Furthermore, a relatively new measure of overall neural efficacy during explosive contractions, defined as percentage of voluntary to octet force/force-time integral (de Ruiter et al., 2004; Hannah et al., 2012; Tillin et al., 2012) will be discussed. Other methods available for assessing neuromuscular activation will be reviewed more briefly.

Electromyography measures the voltage potential generated across the sarcolemma of muscle fibres, in response to neural activation (MacIntosh et al., 2006). There are two available methods of EMG, intra-muscular and surface EMG. Intramuscular EMG can be used to

measure the activation timing and firing rate of single motor units. The main advantage of single motor unit studies with intramuscular EMG is that the discharge properties of the motor neuron can be obtained from the analysis of the single motor unit discharge. This can be achieved because for every action potential generated in the motor neuron, a corresponding action potential will occur in all the muscle fibres within that motor unit, due to a high safety factor for action potential transmission at the neuromuscular junction (Bigland-Ritchie et al., 1979). Consequently, this is one of few methods that can provide unambiguous information about motor neuron behaviour during voluntary contractions in humans. Intramuscular EMG is not ideal for measuring large muscles groups (Tucker, 1993). It is best suited for analysis of deep muscles, or smaller muscles located near the skin periphery. The surface EMG signal comprises the sum of electrical contributions made by the active motor units (MUs) as detected by electrodes placed on the skin overlying the muscle (Farina et al., 2004). The information extracted from the EMG signal is often considered a global measure of motor unit activity, because of the inability of the traditional (2 or 3 electrode) recording configuration to detect activity at the level of single motor units. There are numerous limitations with surface EMG which need to be considered when interpreting the signal. Firstly, substantial cancellation of the EMG interference signal can occur due to out-of-phase summation of motor unit action potentials (MUAP). Therefore, it has been suggested that the EMG signal does not provide a true estimate of the total amount of motor unit activity (Day & Hulliger, 2001). For example, research has also shown that increased motor unit synchronisation can result in increased EMG amplitude (Yao et al. 2000) due to less cancellation of the EMG signal as a consequence of less out-of-phase summation of the MUAPs. Therefore, changes in EMG can not only reflect changes in muscle fibre recruitment and/ or firing frequency, but also changes in MUAP synchronisation. However, the positive curvilinear relationship between EMG amplitude and force output (Alkner et al., 2000; Kooistra et al., 2007; Disselhorst-Klug et al., 2009) supports its use as a global indicator of the level of neural activation.

Additionally, the overall interference signal is mediated by a multitude of intracellular and extracellular factors which all exert a significant influence on the pattern of spatial and temporal summation of the single action potential. These include factors such electrode placement, signal crosstalk from other muscles, subcutaneous tissue, blood flow, fibre diameter, muscle biochemistry amongst others (de Luca, 1997; Farina et al., 2004, 2006).

Consequently, normalisation of the EMG signal is considered essential for comparisons between participants as well as for repeated measurement sessions with the same individual.

The EMG amplitude during a task of interest has typically been normalised to the amplitude obtained from a reference contraction, although there is no general agreement as to the best normalisation method (Perry, 1992). One method during maximal contractions is to compare the peak EMG during an isometric MVC (Burden & Bartlett, 1999); however, this method becomes problematic when it is necessary to quantify activation during MVC across individuals and across trials for the same individual. For EMG during maximal contractions, EMG response to an evoked maximal compound muscle action potential (M_{max}) has also been suggested as an alternative normalisation method (Araujo et al., 2000; Gandevia et al., 2001). As the M_{max} response is not confounded by volitional activation, it may provide superior reliability to traditional normalisation techniques (i.e. to EMG during MVCs). The peak-to-peak amplitude of M_{max} (M_{max} P-P) has been used to normalise EMG during explosive and maximum voluntary contractions (Van Cutsem et al., 1997; Tillin et al., 2010, 2011). Although, the reliability of EMG normalised to M_{max} P-P has been investigated during MVCs (CV, 12.1-13.4%, ICC, 0.45-0.90, Place et al., 2007) there has been no investigation during explosive contractions. Recent research has also suggested that the cumulative area of the M_{max} (M_{max} Area) may provide a more reliable measurement parameter than M_{max} P-P (Tucker & Turker, 2007), but the reliability of this parameter in either absolute terms, or when used as a normalisation method for volitional EMG has not been assessed.

Surface EMG can be used to assess activation during the explosive phase of isometric contractions, by splitting EMG into distinct time windows. EMG amplitude should reflect the timing of force achieved during the contraction. As there is an EMD at force onset, EMG analysis should begin from the onset of muscular activation (onset of EMG amplitude), and adjusted for this EMD. This onset of neuromuscular activation should be determined manually using visual identification of EMG onset. Whilst the between-session reliability of EMG amplitude has been assessed during maximal, submaximal and sustained isometric contractions (Yang & Winter, 1983; Rainoldi et al. 2001; Mathur et al. 2005; Clarke et al., 2007), its reliability during explosive isometric contractions has not been documented. Similarly, to EMG at MVF, effective use of EMG requires a normalisation procedure. However, there is no consensus on the most appropriate procedure (Perry, 1992). Isometric maximum voluntary contractions (MVC) are the most widely used (De Luca, 1997) and

advocated (Burden, 2010) reference method. There is however, no standard procedure for assessing the EMG during MVCs for the purpose of providing a reliable reference for EMG normalisation. Some authors have used the peak EMG (Bruhn et al., 2006) irrespective of the time it occurs, whereas others have used EMG at MVF (Gruber & Gollhofer, 2004), but there appears to be no evidence as to which is superior. Additionally, there is no consensus on the optimal window length that should be used when processing the amplitude of the EMG signal during MVCs, and a range of window lengths has been reported (100 ms, de Ruiter et al., 2004, 2006; 200 ms, Gruber & Gollhofer; 500 ms, Place et al., 2007).

Alternatively, de Luca (1997) suggested that sub-maximal contractions at $\leq 80\%$ MVF might provide more stable EMG amplitude than MVCs, and there is evidence that the EMG amplitude during sub-maximal contractions exhibits superior between-session reliability (Yang & Winter, 1983; Rainoldi et al., 1999). Furthermore, normalisation to M_{max} in line with EMG at MVF has been suggested. Further research is required to begin to understand the most appropriate normalisation procedures for EMG during explosive contractions.

TheITT is commonly used to assess the completeness of skeletal muscle activation during voluntary contractions (for a review see Shield & Zhou, 2004). The theoretical basis for the ITT is that when a supramaximal electrical stimulus is applied to a motor nerve branch during voluntary contraction, those motor units not already recruited, or those motor units firing sub-optimally and not in refractory state will respond with a twitch response or twitch like increment in force (Belanger & McComas, 1981; Shields & Zhou, 2004). With increasing neural drive to the muscle, fewer motor units are available for recruitment, therefore, the twitch response becomes smaller and smaller with increasing activation. The superimposed twitch as a proportion to a control twitch elicited at rest is considered to represent the inactive proportion of muscle. An increment in force from the ITT during an MVC denotes sub-maximal neural activation. The conventional method of calculating voluntary activation with the ITT assumes a linear relationship between evoked twitch and voluntary force, in which voluntary activation is quantified as the ratio of the superimposed to control twitch, subtracted from 1 and multiplied by 100 (Pucci et al., 2006; Del Balso & Cafarelli 2007). Several investigators have questioned the use of the ratio of superimposed twitch to control twitch as a reliable method for measuring maximal activation (see de Haan et al., 2009). Firstly, the relationship between evoked twitch and voluntary force has been examined in numerous muscle groups and reported to be concave and asymptotic at medium and high

voluntary forces (Belanger & McComas, 1981; Behm et al., 1996; Suter et al., 1996). Furthermore, the method assumes the superimposed twitch to occur at MVF (Folland & Williams, 2007b), which is highly unlikely. Recently, alternative methods, presumed to be more valid have been recommended in which a measure of maximal voluntary activation may be obtained by calculating the difference between MVF and theoretical maximum force (TMF), where TMF has been extrapolated from an appropriate curvilinear model of the superimposed twitch force-voluntary force relationship (Folland & Williams, 2007b).

A limitation of the ITT is that it cannot be used during an explosive contraction. The ITT requires stable force production, to ensure correct application of the stimulus to the corresponding force level, ensure a stable level of voluntary activation and to also ensure that the superimposed force response can be accurately measured. Neither of these requirements occurs throughout the rising force-time curve during explosive force production. A potential alternative method which has recently been used, and considered to provide a quantifiable measure of voluntary activation during the early phase of explosive contractions (0-75 ms), is to contrast voluntary explosive strength in relation to the force production achieved in response to electrically evoked octet (8 pulses at 300 Hz) stimulation (de Ruiter et al., 2004; Tillin et al., 2012; Hannah et al., 2012) hereby termed 'Neural Efficacy'. The supramaximal octet is thought to elicit maximal RFD of the MTU (de Ruiter et al., 2004) and therefore, provide a valid and reliable reference measure by which to contrast voluntary activation capacity. It is possible that this measure of overall Neural Efficacy could provide a more valid and reliable measurement technique than surface EMG for assessment of neuromuscular activation during explosive contractions, however the reliability of either evoked octet RFD/ force or reliability of ratio of voluntary/evoked octet force has yet to be documented. A weakness of both the ITT and Neural Efficacy (voluntary to octet force) is that they can only be used to assess completeness of activation of the agonists during a single joint action. Human movement involves complex coordination involving multiple muscle groups across multiple joints, as well as antagonist, stabiliser and synergist muscle groups. Thus, these techniques can only be used to assess agonist activation during isolated single joint, typically isometric situations, but not functional, multiple joint or dynamic actions.

The quantitative evaluation of the neuromuscular excitability may be appraised by measuring the EMG responses elicited from electrical stimulation of peripheral nerves. Reflex studies produced through the stimulation of peripheral nerves have the potential to provide

information concerning the sites of adaptation following an intervention such as RT (i.e., spinal vs. supraspinal). There are two types of reflex techniques typically utilised, the H-reflex (Magladery & McDougal, 1950) and the V-wave (Upton et al., 1971). The H-reflex involves delivering a sub-maximal electrical stimulus to a peripheral nerve, which results in a characteristic reflex response (H wave), caused by a motoneuron discharge evoked by the activation of the Ia fibers from the muscle spindles. The maximal H-reflex response (H_{max}) is often normalised to the maximum compound action potential (M_{max}) and may be useful to assess motoneuron excitability *in-vivo* (Hugon, 1973; Schieppati, 1987). The V-wave is an electrophysiological variant of the H-reflex, and is delivered during an MVC and can be used to reflect the magnitude of efferent α-motoneuron output during voluntary muscle activation (Aagaard et al., 2002b).

Transcranial magnetic stimulation (TMS) involves painlessly activating neurons in the human cerebral motor cortex through the scalp. An index of responsiveness of the entire pathway from brain to muscle can be obtained from size of the compound muscle action potential, recorded with surface EMG. Use of TMS over the motor cortex elicits short-latency excitatory responses termed motor evoked potentials (MEPs). TMS has been used to interpret the level of supraspinal activation (Carroll et al., 2009) and it is thought that an increase in force is elicited from TMS then it can be assumed that the voluntary activation from the cerebral cortex was suboptimal. Voluntary activation using TMS is determined in a similar manner to the ITT, in which an MVC is performed, and this is compared to an extrapolated reference twitch (except down to a predicted resting twitch as opposed to up from a measured resting twitch for the ITT) in order to calculate voluntary activation. The size of the TMS is also influenced by factors within the spinal cord. Many corticospinal cells terminate on interneurons in the spinal cord; therefore the net output produce from TMS can be affected by synaptic efficiency and intrinsic responsiveness of the spinal inter-neuronal circuits (Carroll et al., 2011). Thus, it is inappropriate to use it as a measure of cortical activity. TMS is inherently a variant of the ITT, which involves delivering one or more stimuli during voluntary activation. It requires similar measurement constraints as the ITT including a stable force and activation value to elicit the electrical stimulation and thereby, it cannot be used to provide a reliable valid measure of activation during explosive tasks.

Mechanomyography (MMG) is a technique which measures mechanical (as opposed to electrical with EMG) activity of muscle using specific transducers to record muscle surface

oscillations due to mechanical activity of the motor units (Orizio & Gabbo, 2006). Islam et al. (2013) recently suggested that MMG is a useful tool for the assessment of voluntary activation. However, there is very little research currently available concerning its validity and reliability. Furthermore, the majority of research has been performed involving small sample sizes and healthy participants (Islam et al., 2013). Therefore, future work is required to determine if the method could provide an effective comprehensive measurement technique for the study of muscle function.

Muscle functional magnetic resonance imaging (mfMRI) is a further method of examining muscle activation. It refers to changes in the contrast properties of certain MR images that occur in exercising muscles. It measures the activity-induced increase in the nuclear magnetic resonance transverse relaxation time of muscle water, which is caused by osmotically driven shifts of fluid into the myofibrillar space (Meyer & Prior, 2000). These changes result indirectly from increased rates of cellular energy metabolism (Damon et al., 2007), and therefore provides an indirect measure of neuromuscular activity only. Unlike surface EMG, which provides a more global measure of neuromuscular activation (due to typically using two or three electrodes across the skin, and therefore localised to a specific region), noninvasively obtaining three-dimensional images of muscles (Kinugasa et al., 2006). The limited number of activities which can be performed within the MRI scanner, expense and time consuming nature of analysis, limit its applicability.

In summary, there is no gold standard measurement available for the assessment of neuromuscular activation. Numerous methods are available and each has their own strengths and limitations. Regardless of the method employed it is important to design rigorous studies to account for the measurements limitations. The majority of measurement techniques do not offer the opportunity to assess voluntary activation during explosive force development. Surface EMG and Neural Efficacy are techniques available for measurement of neuromuscular action during rising force development, but there is little or no evidence available on their reliability. Furthermore, given the issues associated with use of surface EMG, it is essential that the signal be normalised to a reference contraction to allow for comparison between individuals or for multiple between session analyses of the same individual. No study to date has assessed which methods may provide reliable reference points for this purpose, and this should be established.

2.3.4 Measuring the Intrinsic Contractile Properties of Skeletal Muscle

The contractile properties of a skeletal muscle describe its force response to a known electrically or magnetically evoked input stimulus. The contractile properties can be measured for a single motor unit, a skinned muscle fibre, or for a whole muscle group *in-vivo*. However, this review will focus on *in-vivo* measurement of contractile properties of human skeletal muscle. Whole muscle *in-vivo* contractile properties are assessed by eliciting impulses via percutaneous electrical stimulation (or magnetic stimulation) either directly over the muscle belly/ muscle motor points (Maffiuletti 2010), or over the nerve that innervates the muscle of interest (e.g., de Ruiter et al., 2004, Tillin et al., 2010). The intrinsic contractile properties can be determined via the force-frequency relationship, but this will not be reviewed here. Assessment of involuntary RFD in response to evoked contractions can give insight into the intrinsic capacity of the MTU for explosive force production without the influence of voluntary control and is therefore thought to reflect muscle morphology and tissue mechanics (Almeida et al., 1994; Harridge et al., 1996; Oda et al., 2007).

The contractile properties are typically assessed from single twitch supramaximal stimulation. Assessment of the force response typically includes measurement of isometric peak force, time to peak force, RFD (peak RFD or RFD over a given time period), and half relaxation time (time for the descending force-time curve to reach half peak force, Enoka 2008). It has been shown however, that maximal RFD can only be achieved at high frequencies of stimulation (Buller & Lewis, 1965). Twitch peak RFD is only 25-30% of the maximal RFD (de Ruiter et al. 1999), and therefore single twitch contractions may provide less insight into the intrinsic explosive capacity of the MTU than high frequency contractions such as an evoked octet (8 pulses at 300 Hz), which has been found to evoke the maximum capacity for RFD (de Ruiter et al., 2004, 2006). The reliability of an evoked octet contraction for assessing the MTUs maximal capacity for RFD is unknown and therefore, needs to be established. Furthermore, a body of research is required to determine the most valid method for assessing the contractile properties, in order to effectively document the morphological contributions to voluntary muscle performance.

2.4 Relationship of Neuromuscular Performance to Sports Performance and Injury Risk

2.4.1 Sports Performance

The evidence for an association of isometric maximum strength variables and sporting performance such as a vertical jump is equivocal (Young et al., 1999, 2001; Tillin et al., 2013). Isometric assessments of muscle strength are more controlled than isoinertial strength measurements and therefore allow for a more precise delineation of underlying neural and morphological contributions to performance. However, they have been criticised for a perceived lack of specificity and validity in relation to the dynamic muscle actions of athletic activities (Wilson & Murphy, 1996). Functional tasks generally require isoinertial strength, and thus isoinertial strength is of importance to sports performance. The evidence of an association with isoinertial maximum squat strength and explosive dynamic performance is also controversial. Nuzzo et al. (2008) compared the relationship between vertical jump height and 1RM squat strength in absolute and relative terms (normalised to body mass) and reported a good relationship between vertical jump height and relative, but not absolute 1RM. Similarly, Requena et al. (2011) reported moderate to strong correlation values between sprint and jump performance and relative 1RM squat scores, but non-significant findings with absolute 1RM scores. Taken together, it appears that relative and not absolute measures of isoinertial strength tasks (1RM squat) are important for athletic performance. This is not surprising when examining Newtonian mechanics as a body's ability to accelerate is dependent upon relative force capabilities (acceleration = force ÷ mass).

The human capability for explosive force production is considered by some to be more important than the capaability for MVF production during sports activities where time to develop force is limited (such as sprinting, jumping and punching) (Aagaard et al., 2002a; de Ruiter et al., 2004; Tillin et al., 2010). For example, Tillin et al. (2010) has reported that explosive power athletes with ability for explosive sporting actions (sprinters and jumpers) had two fold superior ability to express their available explosive force capacity during the early phase of contraction, but only 26% superior MVF capability. Thereby, suggesting the ability for explosive force development of the quadriceps is important for dynamic explosive sporting tasks. Two studies have assessed the relationship between explosive-isometric force production of the knee extensors and countermovement jump performance in small groups (N = 11), and whilst one reported a correlation between these parameters (de Ruiter et al., 2006)

the other did not (de Ruiter et al., 2007). The discrepancy in these results likely reflects similar thoughts when examining isometric and isoinertial strength parameters, that there are several muscle groups in addition to the knee extensors that contribute to jump performance. Likewise however, multiple joint explosive isometric force production has been reported to be either strongly related to jump height (Marcora & Miller, 2000), or unrelated to jump height (isometric squats, Nuzzo et al, 2008). Tillin et al. (2013) suggested that the assessment of RFD could explain the contrasting findings, in which assessment of isometric RFD at only one specific time point during the force-time curve may not have assessed the relevant force-time characteristics for jumping. Indeed, when examining the explosive force time characteristics over distinct time points from force onset (50, 100, 150, 200 and 250 ms) and relating force production at each time point to both countermovement jump and sprint performance, it was shown that multiple joint RFD in the early phase of contractions ($\leq$ 100 ms) was more closely related to acceleration capabilities during a sprint run (5-20 m, r = -0.54 to -0.63), whereas the ability for late phase RFD (> 100 ms) was more related to vertical jump performance (r = 0.51 to 0.61). The results of the study suggested that explosive force production during isometric squats was associated with athletic performance. More specifically, that sprint performance was most strongly related to the proportion of maximal force achieved in the initial phase of explosive-isometric squats ($\leq$ 100 ms), whilst jump height was most strongly related to absolute force in the later phase of the explosive-isometric squats (> 100 ms). Consequently, it seems that the time constraints of the task need to be considered when understanding the possible determinants of a sporting action.

2.4.2 Injury Risk

There is wide belief that the development of neuromuscular strength parameters is integral to injury prevention. Thereby, there is wide implementation of neuromuscular training programmes by athletes with a goal to decrease injury risk. There is good evidence of a positive effect of neuromuscular training interventions (i.e., RT, balance training, plyometrics) on the reduction in sporting injury risk (in team sports players (e.g. Caraffa et al. 1996; Mandelbaum et al. 2005; Olsen et al. 2005; Pasanen et al. 2008; Emery et al. 2010). In a recent analysis it was found that implementation of multi intervention programs was effective at decreasing the risk of lower limbs injuries (39%), specifically, knee (54%) and ankle injuries (50%). The rate of injury risk reduction was more pronounced in those individuals with a previous history of injury (Hubscher et al., 2010). Although, fundamental

and important research, the use of multi-aspect neuromuscular training programmes does not enable the identification of the extent of the various training components (e.g. balance, strength, flexibility) individual contribution to this injury risk reduction. It does provide excellent support for the benefits of neuromuscular training on injury risk minimisation, particularly for those with an increased risk following previous injury.

Tillin et al. (2013) reported that the time available for force development during a particular task, was a strong predictor of the type of neuromuscular strength required to achieve the goal. As recent evidence suggests that an injury such as anterior cruciate ligament rupture occurs within the initial 50 ms after ground contact (Krosshaug et al., 2007), it is possible that the ability for rapid force development may be of more importance than the ability to produce MVF for joint stabilisation and injury prevention.

EMD represents an important aspect of neuromuscular reaction time, as following mechanical perturbation after a loss of balance or trip or fall, there could be unrestrained development of forces of sufficient magnitude to damage ligamentous tissue in synovial joints (Huston and Wojtys 1996; Mercer et al. 1998; Shultz et al. 2001). Furthermore, given the limited time constraints to stabilise the knee before possible injury, it is likely an enhanced capability for very early phase RFD may help overcome potentially harmful forces, and limit damage to the surrounding structures. Thus, it is felt that EMD and RFD are important descriptors of explosive neuromuscular performance for injury prevention by facilitating the timely initiation and development of protective muscle forces (Shultz et al. 1999; Minshull et al., 2007; Blackburn et al. 2009). No research is available, as to whether there is an association between the ability to initiate and develop force and injury risk. However, evidence is accumulating that suggests that muscle strength and explosive force production capabilities are important determinants of the effectiveness of postural corrections during gait and following a perturbation that could lead to a fall or injury (Izquierdo et al. 1999; Pijnappels et al. 2008; Karamanidis et al. 2008; Wyszomierski et al., 2009; Sundstrup et al., 2009; Bento et al., 2010; Arampatzis et al., 2011). For example, both RFD and strength of the lower limb muscles appear to be associated with the ability to recover from experimentally-induced trips (Pijnappels et al. 2008) and slips (Wyszomierski et al., 2009). Furthermore, balance training has been reported to separately improve both RFD (Gruber & Gollhoffer, 2004, 2007) and decrease risk of joint injuries (Hubscher et al., 2010), but there is

no evidence at present for a causal relationship. It is important to begin to further understand the potential role of neuromuscular explosive performance on injury risk.

2.5 Evidence for Determinants of Maximal Muscle Strength

2.5.1 Morphological Contributions to Maximal Muscle Strength

2.5.1.1 Muscle Size & Architecture

A large muscle size is reflective of an increased number of sarcomeres in parallel and therefore, greater number of actin-myosin cross-bridges. Cross-sectional differences in strength are observable between different age groups (Always et al., 1996), gender (Castro et al., 1995) and throughout development (Kanehisa et al., 1995) and these differences are typically attributed to differences in muscle size. Muscle size can be evaluated *in-vivo* from ultrasound, computerised tomography and MRI. MRI is considered the superior method of determining muscle size due to its clearer resolution (Fukunaga et al., 2001; Folland & Williams, 2007a). Whilst MRI provides good resolution of ACSA of muscle, allowing for distinction between muscle, fat and connective tissue, this does not tell us anything about the alignment of the muscle fibres. Use of ultrasound may facilitate measurement of fascicle length and angle of pennation.

The pennate arrangement of muscle fibres allows a greater number of muscle fibres to be arranged in parallel, which theoretically should increase the force generating capacity of the muscle. However, with an increase in angle of pennation, the sum of forces resolved along the aponeurosis by the individual muscle fibres is reduced by a factor of cosine angle (Fukunaga et al. 1997). Thus, a trade-off exists between the increase in angle of pennation and thus muscle CSA and force generating capacity and the force resolved along the aponeurosis, in which isometric force per ACSA increases with increased angle of pennation until 45° (Alexander & Vernon, 1975), beyond which a further increase in angle of pennation would result in a net reduction in force capacity. Evidence from cross-sectional investigations relating indices of muscle size (such as anatomical and physiological CSA [PCSA], and muscle volume) and maximum strength suggest that muscle size explains ~ 50% of the variability in isometric maximum isometric force capabilities between individuals (Maughan et al., 1983; Bamman et al., 2000; Fukunaga et al., 2001; Blazevich et al., 2009).

Blazevich et al. (2009) recently assessed a range of anatomical factors including muscle volume, PCAS, ACSA, moment arm and knee extensor torque. The authors reported that muscle volume was the best predictor of knee extensor movement measured isometrically (R^2 = 0.60) and at 30° s^{-1} (R^2 = 0.74). The unexplained variability is likely explained by factors other than muscle morphology and likely includes the level of neuromuscular activity during force production.

Longitudinally, it has been consistently shown that increased strength in response to RT is associated with muscle hypertrophy (Narici et al., 1996; Hakkinen et al., 1998; Aagaard et al., 2001; Cannon et al., 2007), and declines in strength with rest or immobilisation, associated with atrophy of muscle (de Boer et al., 2007). The long term gains in maximal strength are thought to be primarily due to peripheral adaptations, as evidenced by parallel increases in muscle size and strength following the initial two months of RT, whereas the short term adaptations in strength are thought to be largely due to neurological changes (Narici et al., 1996). Various indices of muscle size (ACSA, PCSA, muscle volume) assessed by MRI, show significant changes after 8-12 weeks of regular RT (Folland & Williams, 2007a). However, increases in muscle size have been documented as early as four weeks (Seynnes et al., 2007). This adaptation appears to proceed in a linear fashion from onset of training for at least six months (Narici et al., 1996). Furthermore, training adaptations appeared to be influenced by muscle group with greater hypertrophic responses of upper body than lower body (Welle et al., 1996; Abe et al., 2000). Additionally, the extent of whole muscle growth appears to vary within constituent muscles of a muscle group, as well as along the length of each of the constituent muscles (Narici et al., 1989, 1996; Hakkinen et al., 2001; Folland & Williams, 2007a). Increased muscle size is thought to be due to the hypertrophy of muscle fibres (increased muscle fibre CSA), facilitating the increase in contractile proteins arranged in parallel (Folland & Williams 2007a). RT is thought to elicit an increased angle of muscle fascicle pennation, through increased packing of contractile proteins in parallel with muscle fibre hypertrophy (and potential hyperplasia) of pennate muscles (Folland & Williams 2007a) following training for maximum strength (Aagaard et al. 2001; Reeves et al., 2004; Blazevich et al., 2007).

Recently, it was reported that 9 weeks of RT of the knee extensors resulted in 26% increase in isometric knee extensor MVF production, but only a 6% increase in muscle PCSA (Erskine et al., 2009). Therefore, as well as changes in muscle size, other adaptations such as

muscle fibre type composition as well as neural contributions involving changes in muscle activation may be expected to influence adaptations in strength following RT.

2.5.1.2 Fibre Type

The exact role of fibre type proportion on the expression of muscle strength is not fully understood. The specific tension of the three different types of muscle fibre is a matter of debate in which some studies have reported higher specific tension of type IIa and IIx fibres (Bottinelli et al., 1996; D'Antona et al., 2006; Pansarasa et al., 2009), whereas others have not (Larsson & Moss, 1993; Ottenheijm et al., 2000; Gillier et al., 2009). There is evidence that fibre type proportion is an important determinant of force production at fast concentric velocities (Aagaard & Andersen, 1998) but less is clear of its role on maximal isometric strength.

Longitudinally, there is evidence of greater hypertrophy of type II fibres which increase in size earlier and greater extent than type I fibers (Hakkinen et al., 1981; Tesch, 1988; Staron et al. 1990; Folland & Williams 2007a). It is possible that the greater hypertrophic response and greater specific tension of type II fibers, contributes to the higher specific tension observed following RT (Folland & Williams, 2007a). Further work is required to understand the role of fibre type composition and maximal muscle strength.

2.5.2 Neural Contributions to Maximal Muscle Strength

Although it is clear that the nervous system regulates skeletal muscle function, the contribution of the nervous system in explaining the variability in muscle strength between individuals is not fully understood. For the nervous system to make a substantial contribution to muscle performance, or the prevention of injuries, there must be variability in the ability of individuals to appropriately activate their muscles, i.e. it is sub-optimal in untrained individuals, and/or it can be enhanced through training. Sale (1988) likened the expression of voluntary strength to a skilled act, where agonists must be maximally activated, while supported by appropriate synergist and stabiliser activation and opposed by minimal antagonist activation. Muscles are generally termed agonists when contracting concentrically they cause joint motion through a specified plane of motion. Antagonist muscles are usually located opposite of the joint from the agonists and have the opposite concentric action, and work in tandem with agonist muscles as a pair. Stabilisers surround the joint or body part and

contract to fixate or stabilise the area to enable another limb or body part segment to exert force and move. Synergists assist the action of the agonist muscles, but are not prime movers of the action and are known as guiding muscles (Thompon & Floyd, 2004).

2.5.2.1 Evidence for Maximal Agonist Activation

Quantifying activation capacity has been a controversial issue within neuromuscular physiology. The field is limited by the available technology and techniques, in that there is no considered gold standard measure of neural activation capacity. Early research using insensitive forms of the ITT concluded that untrained healthy participants can achieve 'maximal' activation during isometric single joint efforts (Gandevia, 2001). However, researchers have become more aware of the methodological issues associated with the use of the ITT. It appears that activation values vary as a function of muscle group, in which activation of the elbow flexors even in untrained participants has been reported to be maximal or close to maximal (> 98%, Allen et al., 1998; Gandevia et al., 1998), whilst the knee extensors are reported to range from 85-95% (Brown et al., 1990; Jakobi & Cafarelli, 1998; Kalmar & Cafarelli, 1999; Behm et al., 2002; Shima et al., 2002; Tillin et al., 2011). Furthermore, it appears muscle activation may also be joint angle specific. Becker and Awiszus (2001) demonstrated within the knee extensors that muscle activation was reduced with increasing knee joint angle to more extended knee joint positions, so that at a knee joint angle of 40° muscle activation was ~ 20% lower (~70% activation) than at a knee joint angle of 90. Therefore, it can be concluded that for isometric single joint MVCs, agonist activation is generally not maximal in untrained individuals, but does vary as a function of muscle group and joint angle.

There is evidence that there is inhibition during high force concentric contractions typical of heavy lifting/maximal strength tasks, resulting in reduced neural drive (Westling et al., 1991; Aagaard et al., 2000). Aagaard et al. (2000) found lower EMG amplitude during slow concentric contractions than for fast concentric contractions of the knee extensors, which was either partly (rectus femoris) or totally abolished (vastus medialis/ lateralis) following a period of 14 weeks of RT. Babault et al (2001) reported a lower level of activation for slow concentric than isometric contractions (89.7 vs. 95.2%).

Greater deficiencies in activation can be observable during maximal eccentric muscle actions. The force-velocity relationship measured *in-vivo* during maximal voluntary muscle actions has been shown to deviate markedly from that of isolated, *in-vitro* muscle preparations (Katz, 1939). Maximal eccentric contraction strength is equal to or up to about 40% higher than maximal isometric contractions recorded in the human quadriceps *in-vivo* (Westling et al., 1988; Dudley et al., 1990; Seger & Thorstennson 1994). In contrast maximal eccentric strength obtained from isolated muscle preparations *in-vitro* is 50-100% greater than isometric or slow concentric force (Katz, 1939; Edman, 1988). Armiridis et al. (1996) measured the force-velocity relationship during MVCs of the quadriceps and reported that the force generated during an eccentric contraction was not greater than the force achieved during concentric muscle actions. Superimposition of electrical stimulation onto the maximal voluntary contraction resulted in an increase in eccentric but not concentric force. Pain and Forrester (2009) examined the in vivo knee extensor force-length-velocity relationship of a group of athletes using a series of eccentric, isometric and concentric contractions and a muscle model, and contrasted it to the tetanic *in-vitro* force-velocity relationship. The authors also then divided the MVC forces by the normalised EMG data and generated corrected EMG amplitude-length-velocity data and EMG corrected force-length-velocity data. The *in-vitro* tetanic force-velocity relationship provided a significantly better fit to the EMG corrected forces compared to the actual measured MVC forces. Additionally, EMG corrected forces generated realistic *in-vitro* tetanic force-velocity profile. The authors concluded that neural factors are the major contributor to the difference between *in-vitro* and *in-vivo* maximal force, and declared a 58% increase in maximal eccentric strength is theoretically possible through the elimination of neural deficits. This is substantially different to the 0-15% window for improvements in isometric MVF, and highlights the significant contribution neural factors can make to the inter-individual variability in eccentric strength.

Improvement in neural function contributing to an increase in strength following RT indicates that neural function pre-training was sub-optimal, and that neural factors can explain some of the variability in muscle strength between individuals. Increases in maximal contraction force as well as maximal RFD will occur not only because of alterations in muscle morphology and architecture (Aagaard et al., 2002a), but also the result of changes in the nervous system. Large increases in muscle strength during the early phase of a RT program have been observed (Abe et al., 2000; Pucci et al., 2006; Del Balso & Cafarelli, 2007) and attributed to neural adaptations.

Early indirect evidence of the importance of neural adaptations in the gains in strength following RT arised from the observation that muscle strength increased disproportionately compared to the increase in muscle CSA (Moritani & de Vries, 1979; Narici et al., 1996; Aagaard et al., 2001; Erskine et al., 2008). Possible explanations for this discrepancy could be changes in muscle architecture, fibre type composition or neural activation. However, it appears, even when muscle fibre type and muscle architecture are accounted for, there still exists a discrepancy in changes in muscle morphology and strength following RT (Degens et al., 2009), and suggests a substantial neural contribution to changes in strength following RT.

Conventional dynamic isoinertial RT has repeatedly been found to produce disproportionately greater increases in isoinertial lifting strength (1RM, Thorstensson et al., 1976; Rutherford & Jones, 1986; Knight & Kamen, 2001) which suggests a considerable facility for neural adaptations that are specific to the training task (Sale, 1988; Folland & Williams, 2007a). These neural adaptations are associated with learning and improvement in muscle coordination (Rutherford & Jones, 1986; Laidlaw et al., 1999). However, there is no research which has examined how adaptations of muscle activation may contribute to task specificity and in particular the gains in isoinertial lifting strength, in contrast to more commonly studied isometric measurements. Functional tasks generally require isoinertial strength, and thus isoinertial strength is of importance, but the neural contributions to isoinertial strength have received limited investigation.

Evidence of adaptive changes in neural function with RT has been provided through the use of surface EMG. The research concerning changes in absolute agonist EMG during maximal isometric tasks following RT is controversial, with some studies reporting an increase (Hakkinen & Komi, 1983; Narici et al., 1989; Kubo et al., 2006), whilst others have reported no change (Carolan & Cafarelli, 1992; Garfinkel & Cafarelli, 1992; Narici et al., 1996; Kubo et al., 2001). The majority of these studies did not normalise EMG to M_{max}, and therefore the equivocal evidence could be associated with the methodological issues surrounding absolute EMG amplitude. However, the evidence is still equivocal when reviewing the literature that has normalised EMG to M_{max}, in which RT has been shown to elicit both an increase in agonist EMG to M_{max} (Van Cutsem et al. 1998; Pensini et al., 2002; Cannon et al. 2007) and no change (Rich & Cafarelli, 2000; Pucci et al., 2006; Tillin et al., 2011). With regard to isoinertial strength, Hakkinen et al (1998) demonstrated an increase in absolute agonist EMG during the knee extensor 1RM following six months' isoinertial RT, but no study has

investigated how changes in EMG normalised to M_{max} contribute to the improvement in isoinertial strength.

2.5.3 Specific Neural Mechanisms Influencing Force Expression or Adaptations to Training

Enhanced agonist activation following training could be due to increased muscle fibre recruitment and/or firing frequency. Definitive evidence of an increase in motor-unit recruitment with training would require demonstration of a population of motor units which were in-active pre-training, but can be recruited during voluntary contractions post training (Folland & Williams, 2007a). This is beyond the capabilities of current techniques and so changes in muscle fibre recruitment can only be inferred and so have yet to be demonstrated. It is clearly evident that enhanced agonist activation would require either enhanced firing frequency or recruitment or both.

2.5.3.1 Firing Frequency

Intramuscular EMG recording techniques can be used to examine MUFF of humans in vivo. Few studies have measured MUFF in response to training. Leong et al. (1999) reported higher MUFF in elderly strength trained individuals than age matched controls, suggesting long term training could enhance MUFF, or least in the context of the sample population, override the age related decline in MUFF. Changes in MUFF with RT are equivocal (Rich & Cafarelli, 2000; Pattern et al., 2001). Increased maximal MUFFs have been reported in response to a single session of RT (Rich & Cafarelli, 2000; Pattern et al., 2001). However, it was shown that MUFF returned to pre-training baseline levels by the end of training (Pattern et al., 2001). Other research has reported no change in MUFF in response to isometric training assessed using both submaximal (Rich & Cafarelli, 2000; Pucci et al., 2001) and MVCs (Pucci et al., 2006).

2.5.3.2 Synchronisation

Motor unit synchronisation is the simultaneous activation of numerous motor units. The evidence concerning the influence of synchronisation on MVF production indicates that RT athletes appear to demonstrate greater motor unit synchronisation than untrained individuals and that RT may enhance motor unit synchronisation during MVCs (Milner-Brown et al., 1975; Semmler et al., 1998). Theoretically, it is unsure how an increase in motor unit synchronisation would increase MVF production, as at firing frequencies equivalent to that during an MVC, there is no effect of synchronisation on force (Lind & Petrofsky, 1978).

2.5.3.3 Cortical Adaptations

Although numerous reports suggest central adaptations to RT, the specific sites of neural adaptations within the central nervous system are yet to be definitively identified. Motor skill training has been shown to be accompanied by changes in the functional organisation of the cerebral cortex (e.g. Martin & Morris, 2001) and it seems reasonable to presume RT may involve some reorganisation of the cortex. Research has shown decreases in corticospinal excitability following RT (Carrol et al., 2002; Jensen et al., 2005). Jensen et al. concluded it is likely that motor learning and RT produce differential neural adaptations. However, more recent research has presented evidence that strength training does enhance neural drive from the motor cortex as measured using transcranial magnetic stimulation (Griffin et al. 2007; Carrol et al., 2009; Lee et al., 2009).

2.5.3.4 Spinal Reflexes

Studies using the H-reflex at rest have demonstrated elevated H-reflex excitability in endurance athletes in compared to sprint and power athletes (Rochcongar et al., 1979; Casabona et al., 1990; Maffiuletti et al., 2001), as well as no influence of RT (Scaglioni et al., 2002). However, the method of assessing the H-reflex at rest has received criticism (Aagaard et al., 2002b), and it has been suggest that the H-reflex should be assessed during muscle actions. This is because low stimulation intensities will exert stronger afferent responses on the slow twitch type I fibers (Hugon, 1973). Elevated V-wave amplitudes have been found in sprinters and weight lifters compared to controls (Milner-Brown et al., 1975; Upton &

Radford, 1975) and increased V-wave amplitudes have been found in response to RT (Sale et al., 1983; Aagaard et al., 2002b).

Aagaard et al. (2002b) assessed the V-wave and H-reflex amplitudes (normalised to an M-wave) during maximal contractions and showed a 55% and 19% increase, respectively, in conjunction with a 20% increase in isometric MVF after 14 weeks of dynamic resistance training. The authors concluded that neural adaptations occurred at both spinal and supraspinal levels, involving an enhanced neural drive in descending pathways from higher motor centres as well as increased motoneuron excitability and/or changes in presynaptic inhibition. Further to this, Del Balso and Cafarelli (2007) reported a 57% increase in V-wave to M-wave amplitude with no change in H-reflex to M-wave amplitude at either rest or during contraction (10% MVC) in response to four weeks of isometric training that elicited a 20% increase in MVF. Taken together the research indicates that supraspinal mechanisms play an important role in the adaptations following RT and serve to enhance descending volitional drive to the muscle during MVCs. However, far more research is required particularly concerning the sites of adaptation during different types of muscle actions (i.e. eccentric and explosive contractions).

2.5.3.5 Antagonist Co-activation

Any co-contraction of antagonist muscles clearly reduces net force output of the joint, but it also impairs, by reciprocal inhibition the ability to fully activate the agonists. As a consequence, it is thought that the net torque about a joint would increase due to the removal of negative torque contributed by the antagonist muscles. The level of antagonist co-contraction (measured using surface EMG) produced during an isometric knee extension or flexion MVCs has been reported to be between 5 and 20% of the maximum values recorded from that muscle when acting as an agonist (de Ruiter et al., 2004; Kubo et al., 2004; Krishnan & Williams, 2009, 2010; Tillin et al., 2011). The level of antagonist activation appears to scale to the level of agonist activation, with increments in joint force production (Krishnan & Williams, 2009), and varies as a function of knee angle (Kubo et al., 2004). Strength trained athletes have been demonstrated to exhibit reduced coactivation of the hamstring muscles compared to sedentary participants when performing isokinetic contractions, with knee extensors muscles (Amiridis et al., 1996). Intuitively, a decrease in antagonist coactivation would seem desirable, as this would cause a greater net joint moment

(agonist joint moment minus antagonist joint moment). However, lower antagonist activation may not be optimal for joint integrity, and furthermore, antagonist co-activation may be important for optimal muscle performance. Firstly, antagonist activation is important to protect ligaments at the end-range of motion (Draganich & Vahey, 1990; More et al., 1993). It ensures homologous distribution of compression forces over the articular surfaces of the joint (Baratta et al., 1988) and it increases joint stiffness, thereby providing protection against external impact forces as well as enhancing the stiffness of the entire limb (Milner & Cloutier, 1993).

Carolan and Cafarelli (1992) found a small but significant decrease in hamstring coactivation after one week of RT with no further decrease throughout the remaining seven weeks of isometric training. The authors calculated the net force production of both agonists and antagonist muscles and concluded that the decrease in coactivation accounted for 10% of the increase in MVF following eight weeks of training. Hakkinen et al. (1998) reported a decrease in hamstring coactivation in elderly but not middle aged individuals in response to six months of RT. In contrast, Pucci et al. (2006) showed no change in hamstring co-activation, whilst Simoneau et al. (2006) and de Boer et al. (2007) reported increased dorsi-flexor co-activation after long term plantar-flexor RT in older individuals. Recently, Tillin et al. (2011) showed increased hamstring antagonist EMG amplitude normalised to EMG at MVF in young recreationally active individuals, but a reported a downward shift in the entire agonist-antagonist EMG relationship, representing reduced co-activation post training for any given level of agonist activation.

2.5.3.6 Stabiliser Muscle Activation

The effective stabilisation of joints is thought important for optimal force production (Sale, 1993; Folland & Williams, 2007a). Nozaki et al. (2005) highlighted the importance of controlling the adjacent joint on output of the primary joint. The authors demonstrated that even during relatively simple strength tasks (isometric knee extension), that there was a large variation, both between and within-participants in the ability to stabilise the adjacent joint torque through effective inter-muscular coordination. Although, mono-articular muscles attach to a bony process which allows for a rigid base of support, bi-articular muscles attach to complexes at different joints. Therefore, stability of the adjacent torque may influence expression of these muscles. There is little direct evidence available on the influence of

stabiliser activation on maximal muscle performance. Indirect evidence does come from the observation, that instability (created via use of unstable platforms) causes a reduction in force output, through increased co-contraction of antagonists and resultant decrease in net moment of force and altered muscle coordination (increased reliance on agonists to act as global muscle stabilisers, for a review see Andersen & Behm, 2005). Therefore, it is possible that low levels of stabiliser activation, even during relatively simple constrained tasks, could alter the expression of agonist muscle activation. Cacchio et al. (2008) assessed the training induced adaptations following either constrained or unconstrained path machine training on maximal and sub-maximal muscle activation patterns (activation of agonist, antagonist and stabiliser muscles during a chest press exercise task) and force production. The alterations in agonist, antagonist and stabiliser activation during the submaximal task were investigated and it was reported that the level of stabiliser activation increased following training, with a concomitant reduction in the level of agonist and antagonist activation. The authors suggested the unconstrained path training (which involved having to stabilise the joints without support from the machine) improved efficiency of motor control during the submaximal task, thereby allowing for a lower level of agonist activation, and reduced agonist effort during the submaximal task.

An interesting observation from the Cacchio et al. (2008) was the greater cross-over to unconstrained tasks for the constrained trained versus the unconstrained path to constrained movement. The authors suggested that the gains from unconstrained training included both strength adaptations but also motor pattern changes, but the motor patterns could not be utilised during the constrained activity. The finding does further highlight an important aspect of neuromuscular assessment, in which neural adaptations to training are often assessed in isolated isometric situation, which do not allow for adaptations in movement patterns, specifically possible changes in stabiliser activation to be accurately assessed. It is important to begin to fully understand the influence of morphological versus neural factors on functional performance, if we are to begin to fully understand how to optimally train our athletes or injured individuals.

The majority of research concerning the neural contributions to strength has been considered in isolated single joint situations. This is due to the fact that i) it allows for a more appropriate investigation of the mechanisms contributing to performance and ii) at present we are limited by the technology available for assessing neural activation (using the ITT and not surface EMG) in more complex neuromuscular tasks. It is unlikely that a controlled single joint

isometric situation fully reflects the neural requirements for functional performance. It is important to begin to understand the neural contributions in more functional/ complex situations if we are to fully understand the neural contributions to dynamic sports performance and injury avoidance situations.

2.5.4 Unilateral and Bilateral Contractions

One approach to bridging the gap between isolated single joint situations and functional movement, taking into account the limitations of current knowledge and available assessment techniques for the study of neuromuscular function is to consider bilateral single joint situations. Although not evident in all research studies there is considerable evidence that the human neuromuscular system is incapable of maximally activating both homologous limbs simultaneously (for a review see Jakobi & Chilibeck, 2001). Bilateral deficit (BLD) has been used to describe this phenomenon of a reduction in performance during synchronous bilateral actions when compared to the sum of identical unilateral movements. BLD in MVF is reported to range up to ~25% (Koh et al., 1993; Van Dieen et al., 2003; Magnus & Farthing, 2008), and therefore represents a potentially influential factor in the expression of bilateral muscle function. Understanding the BLD phenomenon may provide insight into complex neuromuscular control patterns. Many dynamic two-limb studies report a BLD, whereas isometric studies are more numerous and controversial (see Jakobi & Chilibeck, 2001). The mechanisms of the BLD are thought to be of neural origin, although the exact mechanisms explaining the phenomenon are unresolved. The primary explanation for BLD during maximum isometric and isokinetic contractions is reduced neural drive to the agonist muscles. However, the evidence is equivocal, with several studies documenting parallel reductions in force and agonist activation during bilateral tasks (Oda & Moritani, 1995; Van Dieen et al., 2003; Post et al., 2007), where as others have not (Schantz et al., 1989; Howard & Enoka, 1991; Herbert & Gandevia, 1996; Magnus & Farthing, 2008).

2.5.5 Summary of Determinants of Maximal Muscle Strength

In summary, the inter-individual expression of maximal muscle appears to be influenced by a multitude of morphological and neural mechanisms. Muscle size is thought to contribute ~ 50% of the variability in maximal isometric strength of single joints, and is thought to contribute

substantially to gains in strength following RT, particularly during the latter phases of training. Other morphological factors which may contribute to the variability in maximal strength include a possible higher specific tension of type II fibre and preferential hypertrophy of type II fibres following RT. Neural mechanisms including agonist and antagonist activation are thought to contribute to the variability in maximal strength tasks. A lower co-activation may increase force production, but could compromise joint stability. Changes in strength particularly during the early period of RT are thought to be primarily due to neural adaptations although the exact mechanisms are not fully understood. Stabiliser activation may contribute to the expression of maximal strength and could likely explain an aspect of the changes in strength following RT, but has received virtually no research attention.

Typically, the determinants of strength have been investigated within single joint actions. Use of isolated isometric assessment allows for more precise understanding of the determinants of strength, but has been criticised for its perceived lack of relationship to functional performance. It is thought that the human system is incapable of maximally activating both limbs during maximal isometric contractions. If a small increase in task complexity such as assessment of bilateral versus unilateral single joint MVF production causes decreased neural outflow and resultant performance, then it could indicate that the precise determinants of isolated single joint actions may not reflect functional muscle actions. Consequently, further work is required to progress the field in order to provide applied practitioners with appropriate evidenced based findings on the factors which influence muscle performance.

2.6 Evidence for Determinants of Explosive Neuromuscular Performance

2.6.1 Determinants of Electromechanical Delay

EMD has been suggested to include the time courses of the propagation of the action potential along the muscle membrane, theE-C coupling processes and the stretching of the series elastic component (SEC) by the contractile component (Cavanagh & Komi, 1979). Recent evidence from a study using high frame rate ultrasound imaging of the gastrocnemius medialis muscle fibres and myotendinous junction suggests that propagation of the action potential along the muscle membrane and E-C coupling account for ~52% of the delay, with the remaining 48% explained by the time taken to stretch the SEC (Nordez et al., 2009).

2.6.1.1 MTU Stiffness and Slack

The SEC is responsible for transmitting muscle force to bone. Tendons are extensible and lengthen due to application of force from the contractile apparatus. The extent of the deformation will depend on the mechanical properties of the entire MTU. The influence of tendon slack on EMD has been examined by several authors. Muraoka et al. (2004) measured EMD of the medial gastrocnemius in vivo by varying the ankle angle and corresponding tendon slack. The authors demonstrated that EMD was independent of joint angle where tendon slack was taken up by the muscle tendon complex. EMD obtained at the muscle tendon complex length with the greatest tendon slack, was greater than when tendon slack was taken up, suggesting that the extent of tendon slack was an important determinant of EMD. The results have been confirmed by Hug et al. (2011) and Lacourpaille et al. (2013) who measured EMD within the biceps muscle group. It is thought that the increase in EMD at short muscle lengths (i.e., shorter than the slack length) is probably explained by an increased time required for the muscle to take up the tendon slack (Lacourpaille et al., 2013).

In theory, increased tendon stiffness would be expected to benefit EMD, by increasing force transmission from muscle to bone, thereby decreasing the time from contraction to joint movement. Cross-sectional research has demonstrated EMD to be negatively related to MTU stiffness (R = -0.77; Wu et al., 2010). A reduction in tendon stiffness and lengthening of EMD has been reported following muscle unloading (de Boer et al., 2007), bed-rest (Kubo et al. 2000) and plyometric training (Grosset et al., 2009). Increased MTU stiffness following RT has been accompanied by a reduced EMD (Kubo et al., 2001), although this is not a global finding (Reeves et al., 2003). The contrasting findings concerning RT, could relate to the measurement differences between studies. Kubo et al. (2000) investigated stiffness of the vastus lateralis aponeurosis, whilst Reeves et al. (2003) investigated the patella tendon stiffness. Further work considering the role of tendon or MTU mechanics on EMD is sought.

2.6.1.2 Fibre Type Composition

As discussed previously, fast twitch fibres have greater shortening velocity and superior contractile RFD than slow twitch fibres (Bottinelli et al., 1996; Harridge et al., 1996; Li & Larsson, 1996; Bottinelli & Reggiani, 2000; D'Antona et al., 2006; Degens & Larsson, 2007). Therefore, a higher type II percentage of muscle fibres would be expected to shorten EMD by

decreasing the time taken to stretch the SEC. Significant relationships between muscle fibre type composition and EMD have been reported within the literature (% type II, $r = -0.72$, Taylor et al., 1997; % type I, r = 0.51; Viitasalo & Komi, 1981).

2.6.1.3 Neural Activation

As discussed, assessment of the intrinsic contractile capacity of the neuromuscular system is thought to reflect muscle morphology. Evoked EMD has been shown to be unrelated to voluntary EMD (Zhou et al. 1996; Mihsull et al., 2007), which suggests that the MTU intrinsic contractile capacity likely does not explain a significant proportion of voluntary EMD variance between individuals. This would indicate that factors other than muscle morphology (such as the level of neural activation) largely explain the variability in voluntary EMD. Voluntary EMD of the quadriceps has been shown to be 100% (16-25 ms) longer than electrically-evoked EMD (Zhou et al., 1996; Minshull et al., 2007), which suggests a significant neural component to the delay. A higher level of agonist activation during the early phase of contraction would be expected to stretch the SEC quicker and therefore decrease the response time. However, no study to date has actually assessed the contribution of agonist neuromuscular activation to the variability in voluntary EMD between individuals.

2.6.1.4 Summary of Determinants of EMD

It is clear that multiple factors likely influence the variability in EMD between individuals, in which morphological factors such as MTU stiffness and muscle fibre composition may play a role. It is likely that neural factors associated with activation of skeletal muscle also explain a proportion of the variability in voluntary EMD, but there is no evidence either direct or indirect to confirm this. Further work to understand the determinants of EMD is sought.

2.6.2 Determinants of Explosive Strength

2.6.2.1 Maximal Muscle Strength

Explosive force production at specific times is a composite function of a joint MVF capabilities multiplied by the relative percentage of MVF able to achieve at that specific time point. Therefore, if there was no variability in the relative capabilities to express the available force generating capacity, then it would be scaled purely to MVF. Andersen and Aagaard (2006) assessed the relationship between MVF and RFD throughout the rising force-time curve (0-10, 0-20, 0-30…0-250 ms) during isometric explosive MVCs of the knee extensors. The authors reported that the role of MVF on RFD depended upon the time period from force onset, in which voluntary RFD became increasingly more dependent on MVF as the time from onset of contraction increased. At time intervals later than 90 ms from the onset of contraction MVF accounted for 52–81% of the variance in voluntary RFD. However, MVF only accounted for 18-21% of the variance in initial voluntary RFD (0-40 ms). Similarly, Folland et al. (2013), reported that MVF was correlated increasingly strongly with absolute explosive force as time from force onset progressed (r = 0.59 – 0.95). Taken together there is good evidence that MVF appears to be an important determinant of later phase RFD, but less important for early phase RFD.

An increase in RFD would be perhaps the single most important functional benefit induced by RT. However, the efficacy of RT for improving explosive isometric strength is controversial, with some reports finding an improvement (Hakkinen et al. 1998, Aagaard et al. 2002a) and others no change (Andersen et al., 2010, Tillin et al., 2011). Tillin et al. (2011) recently investigated the influence of RT on explosive strength when specifically training for MVF. The authors instructed their participants to slowly increase force production during an isometric contraction of the knee extensors (over 1 second) up to 70% their respective MVF, before holding for three seconds at the target force. The authors reported an increase in MVF (20%) and EMG at MVF (26%), but no change absolute force achieved at either 100 or 150 ms after force onset following four weeks of RT. In addition the percentage of MVF achieved after 100 and 150 ms declined by 15% and 12 %, respectively. The authors concluded that the neural mechanisms associated with an enhanced MVF following training were specific to the high force non-explosive contractions performed during training. Similar findings were observed from Andersen et al. (2010) who investigated the adaptations in early (0-100 ms) and late phase RFD (> 200 ms) in response to RT and demonstrated that conventional RT

using loads that could be lifting 6-12 times over a progressive RT programme for 14 weeks resulted in no change in absolute RFD and a decrease in relative RFD during the early phase of contraction. Absolute RFD over the later stages of force development increased 11%, with no change in relative RFD during this time period. Taken together, it appears that RT may evoke differential adaptations in force production across the force-time curve.

2.6.2.2 Fibre Type

RFD is higher in muscles possessing a higher percentage of MHC type II isoforms (Harridge et al., 1996), and therefore muscles possessing higher percentage of type II muscle fiberes would be expected to have higher RFD capabilities. The evidence for an association between fibre type proportion and RFD *in-vivo* is limited and where it has been performed equivocal. Viitasalo and Komi (1981) assessed the relationship between fibre composition (determined via histochemical analysis) of the vastus lateralis, and time taken to achieve different proportions of MVF (5% increments) during explosive voluntary isometric leg press contractions and reported a low to moderate significant relationship between the two ($0.34 < r < 0.48$, Viitasalo, Komi 1978). However, further research by the group reported no relationship between RFD and fibre composition (Viitasalo & Komi, 1981). Evidence from Harridge et al. (1996) supported a role of fibre type as a determianant of RFD, by reporting strong significant positive relationships between the two ($R = 0.999$). However, the study did examine three different muscles (triceps brachii, vastus lateralis, soleus), which have with an array of different morphological and mechanical differences not limited to just muscle fibre composition. Additionally, RFD was not normalised to either MVF or muscle size in this study. Thus, there remain considerable question marks over the role of fibre type composition on RFD *in-vivo*.

Andersen et al. (2010) reported a decrease in type IIx muscle fibre percentage and concomitant increase in type IIa muscle fibre percentage following RT which was accompanied by a decline in relative RFD. The change in RFD and type IIx fibre percentage were related to one another ($r = 0.61$). It is possible that the decline in relative RFD observed following training (Andersen et al., 2010; Tillin et al., 2012a) could in some part be explained by fibre type conformation changes observed with RT.

2.6.2.3 Tendon Stiffness

There is evidence to support an association between tendon stiffness and RFD during both single and multiple joint actions (Wilson et al., 1994; Walshe et al., 1996; Ditroilo et al., 2010). Bojsen-Moller et al. (2005) reported positive correlations between MTU stiffness of the vastus lateralis (measured using ultrasonography) and absolute RFD over the initial 100 (r = 0.65) and 200 ms (r = 0.68) of explosive isometric knee extensions. Furthermore, there was also a small to moderate relationship between tendon stiffness and relative (normalised to body mass) RFD (r = 0.55). There is a potential spurious relationship which may influence these findings, as both MTU (e.g. Muraoka, 2005; Seynnes et al., 2009) and RFD (Andersen & Aagaard, 2006; Folland et al., 2013) are both reported to be related to MVF capabilities. Therefore, further work to understand the influence of tendon stiffness and RFD when accounting for the influence of MVF is required.

2.6.2.4 Muscle Contractile Properties

Assessment of involuntary RFD in response to evoked contractions can give insight into the intrinsic capacity of the MTU for explosive force production without the influence of voluntary control and is therefore thought to reflect muscle morphology and tissue mechanics (Almeida et al., 1994; Harridge et al., 1996; Oda et al., 2007). A greater RFD of whole muscle *in-vivo* during electrically evoked contractions represents a greater capacity of the MTU for explosive force production, which should theoretically benefit RFD during an explosive voluntary contraction. Andersen and Aagaard (2006) documented the influence of the contractile properties on isometric RFD within the knee extensors. The authors reported a moderate relationship ($0.45 < r < 0.60$) between twitch peak RFD and voluntary RFD during the early phase (0-50 ms) of an explosive contraction. Twitch pRFD was not related to RFD when analysed over time periods greater than 50 ms. However, it has been shown that maximal RFD can only be achieved at high frequencies of electrical stimulation (Buller & Lewis, 1965). Twitch peak RFD is only 25-30% of the maximal RFD (de Ruiter et al. 1999), and therefore single twitch contractions may provide less insight into the intrinsic explosive capacity of the MTU than high frequency contractions such as an evoked octet (8 pulses at 300 Hz), which has been found to evoke the maximum capacity for RFD (de Ruiter et al., 2004, 2006).

2.6.2.5 Agonist Activation

There is strong evidence for a significant contribution of agonist activation to RFD. Theoretically a higher level of neural drive to skeletal muscle will benefit RFD (through higher motor unit recruitment and firing frequency). There is some preliminary evidence demonstrating that the level of agonist activation is a key determinant of RFD during the early phase of the contraction. Firstly, research has shown that maximal RFD can only be achieved when using high pulse rates during electrical stimulation protocols (Buller & Lewis, 1965). Secondly, de Ruiter et al. (2004) reported an 8 fold difference in the capability for individuals explosive force capacity when voluntary force production was compared to the muscle maximal capacity for force generation during electrical stimulation of 8 pulses at 300 Hz (10 to 83%). This difference in capability was largely explained by differences in agonist muscle activation measured via normalised surface EMG at the start of the contraction (torque time integral over initial 40 ms, $r^2 = 0.75$). Additionally, Tillin et al. (2010) compared a group of explosive power athletes (sprinters and jumpers) who were accustomed to performing explosive actions to a group of controls, and reported that despite the explosive power athletes displaying only a 26% higher knee extensor MVF than controls, the athletes exhibited a 2-fold greater absolute RFD over the initial 50 ms of the contraction from force onset. The observed mechanical differences were thought to be explained by the large differences in agonist activation (EMG values normalised to M_{max}), as the intrinsic contractile properties of the muscle (response to electrically evoked twitch and tetanic contractions) were similar between groups. The activation values during explosive force production are reportedly 30-40% when examined with either surface EMG normalised to EMG at MVF or Neural Efficacy (de Ruiter et al., 2004; Tillin et al., 2012a; Hannah et al., 2012). Taken together, it appears that the early phase of contraction is sub-maximal and can vary substantially across individuals.

The relationship between EMG and RFD in other studies has been reported to be less than that of de Ruiter et al. (2004). Klass et al. (2008) reported a shared variance between EMG amplitude up to peak RFD and peak RFD capabilities of 33%. Hannah et al. (2012) and Folland et al. (2013) reported a shared variance of 45-50% and 34% for normalised agonist EMG (to M_{max}) and RFD (both 0-50 ms from respective EMG and force onsets) respectively. Therefore, there is strong support for the role of agonist activation on explosive strength, although the exact contributions are not fully understood and appear to vary between studies.

There is some evidence that including an explosive strength component to RT (i.e., intending to lift the weight as quickly as possible) is sufficient to enhance early phase explosive strength and agonist neural drive (Van Cutsem et al., 1998; Barry et al., 2005; Del Balso et al., 2007; Gruber et al., 2007; de Hann et al., 2009; Tillin et al., 2012a). Behm and Sale (1993) reported similar adaptations in RFD for interventions using dynamic ballistic training using loads at 30-40% 1RM and isometric ballistic training and concluded that it was the intention to increase force as quickly as possible regardless of velocity that determined changes in RFD. Those studies that have required participants to develop maximum force as quickly as possible have shown increased RFD following training (Hakkinen et al., 1998; Van Cutsem et al., 1998; Barry et al., 2005; Del Balso & Cafarelli, 2007) with a concomitant increase in EMG.

Del Balso and Cafarelli (2007) reported a large increase in RFD (43%) and agonist EMG (49%) following four weeks of RT of the ankle plantar flexors. Similarly, Tillin et al. (2012a) investigated the influence of explosive strength training using short explosive type contractions which required the participants to increase force as quickly as possible (1-s duration, achieve at least 90% MVF) over four weeks of training of the knee extensors and reported a 54% increase in RFD over the initial phase of contraction (0-50 ms), which was accompanied by 42% increase in agonist activation and relative explosive force production. Furthermore, the early phase of activation is highly trainable with on average 2-3% increase per session or over 10% improvement per week during the early phase of training in untrained individuals (Tillin et al., 2012a). Thus, training the early phase of activation appears to provide significant benefit to RFD, within a short period of training time.

A key question missing from the literature however, is 'is the inclusion of intention to lift a weight quickly during isoinertial RT sufficient to achieve increases in agonist neural drive and explosive force capabilities?' Mechanical changes of explosive strength have been investigated following RT involving either isometric actions or ballistic type training (i.e., Van Cutsem et al., 1998; Rich & Cafarelli, 2000; Del Balso & Cafarelli, 2007). Therefore there is little documented support for the use of conventional RT to develop RFD. Athletic training and/or rehabilitation programmes adopt conventional type RT models, and therefore it is important to understand the influence of isoinertial RT on explosive strength. Aagaard et al. (2002a) has demonstrated an increase in both RFD and agonist EMG following conventional type RT. However, EMG was not normalised, and there was expected to be considerable hypertrophy of type II fibres, following 12 weeks of RT. Another study by the

same research group failed to elicit an increase in RFD and agonist EMG following a similar conventional RT programme (Andersen et al., 2010). No study has examined the influence of isoinertial RT on RFD following short term training. Given the considered importance of RFD for sports performance and injury prevention, understanding how to effectively train this component of athletic performance is essential for applied practitioners.

2.6.2.6 Antagonist Activation

The reported values of antagonist activation have been shown to be very low during the very early phase of voluntary explosive contractions (40-50 ms) of the knee extensors (1-2 %, de Ruiter et al., 2004; Hannah et al., 2012) and therefore would be thought to contribute minimally to the observed joint torque during this time period. There is little documented evidence of the level of antagonist co-activation during explosive force development for other muscle groups than the knee extensors, or during the latter phases of explosive force production (> 50 ms) is available. Furthermore, no study to date has attempted to document the contributions of antagonist co-activation to the variability in voluntary explosive force development observed between individuals. Longitudinally, no research studies have reported stable values of co-activation pre and post RT during explosive force production (Aagaard et al., 2002a; Barry et al., 2005).

2.6.2.7 Stabiliser Activation

No study has considered the importance of stabiliser activation to isometric RFD, or has it been assessed during explosive contractions. It is important to understand the role of stabiliser activation on explosive strength in order to comprehensively understand the determinants of explosive strength.

2.6.2.8 Unilateral and Bilateral Contractions

Despite considerable investigation of the BLD in maximal isometric and isokinetic strength tasks, only a few studies have examined whether there is a BLD in explosive force production, with equivocal findings and limited mechanistic evidence. A BLD in peak RFD has been

reported to range between 0-24% (Koh et al., 1993; Jakobi & Cafarelli, 1998; Sahaly et al., 2001; Van Dieen et al., 2003), and has been shown to be greater than the BLD in MVF in some (Koh et al., 1993; Sahaly et al., 2001; Van Dieen et al., 2003) but not all studies (Jakobi & Cafarelli, 1998; Sahaly et al., 2001). The primary explanation for BLD during maximum isometric and isokinetic contractions is a reduction in neural drive to the agonist muscles. Therefore, as the MUFF at the onset of an explosive isometric contraction can be much higher than at MVF (i.e., 100-200 Hz vs. 20-30 Hz, Monster & Chan, 1977; Kukulka & Clamann, 1981; Bellemare et al., 1983; Van Cutsem et al., 1998), it is possible that any reduction in agonist activation might exert a more pronounced effect on explosive force than MVF. However, at present only one study has actually assessed agonist neuromuscular activation during the explosive phase of an isometric contraction, and reported no change in activation, despite a 13% decline in peak RFD (Van Dieen et al., 2003). Therefore, a possible neurological basis for any BLD in MVF and RFD is equivocal or remains to be established. Given explosive force production is considered to be more important than MVF on performance of dynamic sports tasks (Aagaard et al., 2002a; Tillin et al., 2010), understanding how bilateral contractions influence RFD is an important step to furthering our understanding of the neural contributions to dynamic sports actions. If bilateral contractions exert a significant decline in RFD due to neural inhibition of agonist activation, then the underlying neural contributions of single joint RFD could be considered to not be fully reflective of bilateral explosive strength capabilties.

2.6.2.9 Summary of Determinants of Explosive Strength

In summary, it is evident that there is large inter-individual variability in the capability of the neuromuscular system to develop force rapidly. This inter-individual variability appears to be higher during the early phases of explosive force development. MVF appears to be the main determinant of late, but not early phase RFD. Agonist EMG and twitch properties appear to be important determinants of the development of force (50 ms), with increasing more of an important role for MVF as the time from onset increases. The individual roles of MTU stiffness and fibre type proportions are relatively unknown. It is likely that a stiff MTU system and high proportion of fast twitch fibres would benefit RFD, but there is lack of available evidence. When taken together it appears that the early development of force (50 ms) is a function of the ability to initiate and produce high levels of activation (agonist EMG),

and a high response to sub-maximal levels of stimulation (twitch force responses) and the late phase of RFD (100 ms plus) is related to MVF capability. More research is required to begin to further understand the determinants of RFD, particularly beginning to understand how these isolated determinants of single joint isometric RFD explain functional movement involving multiple degrees of movement freedom.

2.7. Muscle Fatigue

Fatigue can be defined as a reduction in muscle performance following muscle contractions, which largely recovers after a period of rest (see Allen et al., 2008 for a comprehensive review of muscle fatigue). The acute changes that occur with muscle fatigue negatively affect muscle performance, and therefore the ability to produce maximal and explosive force. Muscle fatigue is negatively associated with performance of explosive sporting actions (Mohr et al., 2003; Krustrup et al., 2006) and is thought to be an influential risk factors for sports related injuries (Hawkins et al., 2001). Voluntary contraction of skeletal muscle occurs following complex processes arising at the cerebral cortex, and eventually leading to activation of skeletal muscle following the E-C coupling process. Fatigue can arise at many different points in the pathway and can usually be defined as central and peripheral fatigue. Peripheral fatigue is defined as the loss of force caused by processes occurring distal to the neuromuscular junction. It is universally accepted that much of the fatigue arises in the muscles and therefore, a large volume of fatigue research has been studied using isolated muscle tissues. However, there is often a substantiated central component to fatigue (see Gandevia, 2001), defined as a progressive exercise-induced reduction in voluntary activation or neural drive to the muscle (Taylor et al., 2006).

Muscle fatigue is a complex phenomenon, which has important implications for not only athletic performance and injury prevention, but also daily living and health and disease and therefore significant scientific investigation has been conducted in the area. Thus, there is a considerable level of information available on the topic. A complete discussion of muscle fatigue is beyond the scope of this review. Therefore, the present discussion will consider the mechanisms responsible for the reduction in maximal muscle performance during isometric contraction, from either maximal electrically evoked contractions or voluntary contractions.

2.7.1 Effect of Fatigue on Maximal Muscle Performance

Muscle fatigue has been defined as 'an exercise-induced reduction in maximal muscle force'. The majority of research has considered the influence of fatigue on MVF production, with a lack of research addressing the influence of fatigue on functional muscle performance, or explosive neuromuscular performance. Muscle fatigue has been reported to negatively influence the performance of explosive sporting actions (for a review of fatigue and soccer, see Mohr et al., 2003). Furthermore, it is thought that muscle fatigue is an influential factor in sports injuries (Hawkins et al., 2001), but this link has never been experimentally reported. RFD is considered functionally more important than MVF during explosive movements, such as sprinting, jumping or restabilising the body following a loss of balance (de Ruiter et al., 1999; Aagaard et al., 2002a; Tillin et al., 2010). Therefore, an understanding of how fatigue affects explosive neuromuscular performance would seem important in understanding its influence on athletic performance and injury risk.

Fatigue has been reported to be strongly influence EMD, and in certain cases reported to elongate EMD by up to 70% (Zhou et al., 1996). During less strenuous fatigue protocols, fatigue has been shown to elongate EMD in females but not male participants (Minshull et al., 2007). Furthermore, fatigue has actually been shown to positively enhance magnetically evoked EMD in both males and females (21% decrease in EMD). The exact mechanisms for the differential influence of fatigue on voluntary and evoked EMD is not clear, but it was suggested by the authors that the shortened evoked EMD, might indicate a dormant capability for optimal muscle responses during acute stressful exercise and an improved capacity to maintain dynamic joint stability during critical episodes of loading (Minshull et al., 2012). There appears to be no research that has examined the effects of fatigue on explosive strength. Although, it could be expected that fatigue may exert negative influences on RFD, in line with declines in MVF, as previously discussed in this review, the determinants of RFD appear to change throughout the rising force-time curve (Andersen & Aagaard 2006; Tillin et al., 2010), and therefore, fatigue could differentially affect the development of force throughout the time course of an explosive contraction. It is possible that fatigue may exert more pronounced influences on RFD than MVF production, and this may be due to neural and/ or contractile mechanisms. For instance, Type II skeletal muscle fibres have a substantially higher RFD (Brenner et al., 1986; Metzger & Moss, 1990; Harridge et al., 1996), but arguably similar specific tension (isometric peak force/cross-sectional area; (Larsson &

Moss, 1993; Gilliver et al., 2009)) than type I fibres. Therefore, given the lower fatigue resistance of the type II fibres, a greater influence of fatigue on explosive than maximal phases of the evoked contraction could be expected. Furthermore, as previously discussed the MUFFs achieved during explosive force development appears to exceed the MUFF during the plateau phase of contraction which includes MVF (explosive phase, 100-200 Hz vs. plateau phase, 30-50 Hz, Monster & Chan, 1977; Kukulka & Clammann, 1981; Bellemare et al., 1983; Van Cutsem et al. 1998), and therefore, a decline in MUFF with fatigue could be expected to exert a more pronounced effect on explosive than MVF production. It is important to ascertain the influence of fatigue on explosive force capabilities and report the neural and/or contractile mechanisms which may be influential. Fatigue is common during sport and could play a vital role in injury occurrence, and therefore the topic is of considerable interest to applied sport and exercise science as a whole.

2.7.2 Peripheral Contributions to Muscle Fatigue

Fatigue of fast twitch muscle fibres following electrically evoked tetanic contractions is characterised to proceed in three phases. Phase 1 is characterised by a decline of tetanic force by 10–20%, which is accompanied by an increase in tetanic Ca^{2+}; phase 2 is a period of relatively constant tetanic force; and phase 3 is described as a rapid decline of both tetanic force and myoplasmic Ca^{2+}. The following section will briefly discuss the metabolic changes which occur with skeletal muscle and discuss how force is influenced by these changes.

Acute transition from rest to exercise is accompanied by greatly increased demand for energy by the working muscle and the muscle attempts to balance this demand utilising various energy systems. A feature of fast twitch muscle fibres, particularly type IIx is that they can consume adenosine triphosphate (ATP), producing adenosine diphosphate (ADP) and inorganic phosphate (Pi), much faster than they regenerate it. During the early stages of contraction, ATP content is maintained by the breakdown of phosphocreatine (PCr), which causes a rise in free Creatine (Cr) and Pi. Later on, when PCr falls to low levels, ATP begins to fall and a rise in ADP occurs. As ADP rises, adenosine monophosphate (AMP) becomes important and forms ATP through AMP deaminase. Although, it is believed that in muscle fatigue ATP does not fall to low levels (> 60%), the values reflect whole muscle measurements. However, research using muscle biopsy techniques suggest that ATP levels in certain fibres drop considerably more than this. Karatzaferi et al. (2001) reported that when

PCr dropped to 11% of its initial value, ATP dropped to 20% of its initial value, thus it is possible a decline in ATP could exert significant effects on muscle fatigue. However, during fatigue from high intensity exercise, a major challenge in the intracellular milieu of skeletal muscle is not ATP depletion but metabolite accumulation that affects acto-myosin cross-bridge interaction. The metabolic changes which accompany intense muscle contraction include an increase in ADP, Pi, AMP and inosine monophosphate (IMP). An important consideration to the drop in ATP is a rise in moyplasmic magnesium (Mg^+). ATP has a stronger affinity with Mg^+ than ADP, AMP, IMP and thus a decrease in ATP results in parallel increase in Mg^+ which has been suggested to play a role in muscle fatigue. Further pathways to resynthesize ATP include anaerobic glycogenolysis and the aerobic breakdown either of glycogen, glucose, or fat. Anaerobic glycolysis is of central importance in muscle fatigue because it is turned on rapidly during activity, and the net reaction is breakdown of glucose units to lactate ions and protons causing the early acidosis associated with rapid-onset muscle fatigue.

A detailed review of the underlying fatigue mechanisms which contribute to the decline in force goes beyond the scope of the present review. For an excellent review of the topic, readers are recommended to consult Allen et al. (2008a). In short, when considering a fall of 50% in tetanic force of type II fibres, the early fall in force (phase 1), which is usually a 10% reduction, is likely to be caused by a reduction in force due to increased inorganic phosphate (Pi). The remaining 40% was attributed to reduced Ca^{2+} sensitivity of the contractile proteins and reduced sarcoplasmic reticulum (SR) Ca^{2+} release (Allen et al., 2008a). Although, the exact mechanisms responsible are not fully known, it is thought that the reduced Ca^{2+} sensitivity could have contributions both from metabolites such as Pi and from the effects of reactive oxygen species (ROS). Further, it is suggested that the most likely causes for reduced SR Ca^{2+} release appear to be precipitation of Ca^{2+} phosphate in the SR with a contribution from reduced ATP and raised Mg^{2+}. It is important to note that this evidence has arisen largely from experiments on isolated animal muscle preparations.

2.7.3 Neural Contributions to Muscle Fatigue

Central fatigue can be described as a progressive exercise-induced reduction in voluntary activation or neural drive to the muscle (Taylor *et al.* 2006). The ITT has been used widely to report declines in voluntary activation with fatigue. In response to both intermittent and

sustained MVCs there is a decline in voluntary activation. The contribution of central fatigue to the decline in MVF has been studied extensively (for a review see Gandevia, 2001), and found to contribute up to ~20-25% of the decrease in MVF (Taylor *et al.* 2006). A reduction in neural activation with fatigue could be due to either a decline in motor recruitment or MUFF.

A detailed examination of the underlying mechanisms responsible for the reduction in MUFF is beyond the scope of this review, but has been excellently reported elsewhere (Gandevia, 2001). To provide a summary, both spinal and supraspinal mechanisms are thought to be responsible for the decline in force due to 'central' mechanisms. At the spinal level, factors include intrinsic behaviour of the motor neurons, recurrent inhibition, reflex inputs and their pre-synaptic modulation, and other neuromodulatory influences acting upon the motor neurons and or spinal circuitry. At the supraspinal level, the outputs of descending tracts to the motor neurons and that factors which control descending drive are thought to be important (Gandevia, 2001).

2.7.4 Summary of Fatigue and Muscle Performance

In summary, muscle fatigue has been shown to negatively impact on muscle performance and therefore the ability to produce maximal and explosive force. Muscle fatigue is negatively associated with performance of explosive sporting actions and is thought to be influential risk factors for sports related injuries. However, despite a substantial amount of research assessing the influence of fatigue on MVF, there is a paucity of research on fatigue and explosive neuromuscular performance. Contractile and neural mechanisms are responsible for the decline in MVF, but there is however, no documented mechanistic evidence for fatigue and explosive strength. Fatigue could be expected to exert a more pronounced influence on explosive than maximal muscle strength, which could be due to both contractile and neural mechanisms, but this needs to be established. Given the separate associations of both explosive strength characteristics and fatigue with muscle performance and injury risks, it would seem important and relevant to establish the influence of fatigue on explosive strength.

2.8 Main Aims of the Thesis

There has been no comprehensive investigation of the reliability of RFD. Furthermore, the octet could provide an effective technique to examine the MTU capacity for explosive force production, and when considered alongside RFD, could provide an alternative measure of neural drive during the initial stages of contraction. But its reliability needs to be established. Considering the reliability of EMG during explosive force development and contrasting the different normalisation methods needs to be established. Therefore, the first aim of the thesis was to establish the reliability of explosive force/RFD and documenting the reliability of EMG and evoked force responses.

Agonist EMG over 50 ms has been reported to be the primary determinant of voluntary force production (relative force after 50 ms), but its association with voluntary EMD is unknown. As voluntary EMD is considerably longer than evoked EMD, and previous research has reported low relationships between the two variables, agonist EMG could be an important determinant. If EMG is also a strong determinant of voluntary EMD, then this would further highlight the importance of the ability to increase EMG rapidly over the early phases of contraction (initial 50 ms). Therefore, the second aim of the thesis was to establish the relationship of the ability to activate skeletal muscle during the early phase of contraction (EMG 0-50 ms) with the ability for voluntary EMD. It was hypothesised that the ability to activate agonist EMG (0-50 ms) would be an important determinant of voluntary EMD.

It is not clear if the determinants of RFD in single joint situations reflect more complex functional situations. If there is a large BLD for explosive force, and this is due to neural inhibition then the determinants of single joint RFD may not so readily explain more complex situations. There is considerable research required before the determinants of functional sporting actions are fully understood, but assessing the BLD for RFD and documenting the neural contributions to BL single joint RFD would improve our understanding of the neural contributions to RFD, and help begin to bridge the gap between isolated single joint situations and more functional movement. It was hypothesised that there would be a more substantial BLD for explosive force/RFD than MVF, which would be accompanied by a pronounced reduction in agonist activation during explosive force development.

Marked increases in muscle strength during the early phase of a RT program have been observed and these changes appear to be highly specific to the nature of the training task. The specificity of training phenomenon is taken as strong indirect evidence for neural

adaptations; however, there is minimal direct evidence for either neural or morphological mechanisms that might explain this training task specificity. Early adaptations to RT are thought to be primarily explained by neural adaptations such as agonist and antagonist activation, as opposed to peripheral adaptations. Therefore, documenting training specific adaptations over a short term training period may distinguish between the neural and morphological explanations for the task specificity phenomenon.

The role of conventional RT on RFD is equivocal. There is strong evidence that increasing force rapidly from a low or resting force value up to high level of force is effective at training agonist activation during the early phase of contraction. It is possible that conventional RT would not provide this necessary stimulus to train agonist EMG rise and RFD. Therefore, an aim of the thesis was to investigate the influence of conventional RT, with an explosive strength component included (maximal intention to lift the weight as quickly as possible), and document the possible associated neural adaptations. It was hypothesised that conventional RT with maximal intention to lift the weight, despite this gradual lowering would be sufficient to enhance RFD, which would be accompanied by increased agonist activation during the early phases of contraction.

Although, movement is due to a complex cooperation between agonist, antagonist, stabiliser and synergist muscles, there has to date being a relatively isolated approach to investigating the neural contributions to muscle performance and adaptations to training. Stabiliser muscle activation is considered important for the expression of strength and could explain a proportion of the unexplained variability in gains in strength following RT, but has received very little attention. An aim of the thesis was to try to broaden the approach to investigating neural mechanism in muscle performance and consider stabiliser activation alongside agonist and antagonist activation. A further aim was to establish the role of stabiliser activation on isometric explosive force production. It was hypothesised that stabiliser activation would adapt following RT and this may help explain the task specificity phenomenon following isoinertial training. It was further hypothesised that stabiliser activation would not make an independent significant contribution to isolated isometric explosive force production, but may indirectly contribute to performance through a role on inter-muscular coordination.

Fatigue has been reported to be an influential risk factor for sports injuries. Given the presumed role of RFD and EMD on the ability to activate and develop force during the early

phase of the contraction, it would seem relevant to have an understanding of the role of fatigue on EMD and RFD and the associate mechanisms. However, no study has actually examined the influence of fatigue on explosive force production, and there is no mechanistic evidence of how fatigue could comprise explosive force production. Therefore, the final aim of the thesis was to document the influence of fatigue on explosive force production and EMD and document the accompanying neural and contractile mechanisms. It was hypothesised that explosive force production would exhibit a greater decline than MVF with fatigue, and that this could be due to neural and/or contractile mechanisms.

CHAPTER 3

Reliability of Neuromuscular Measurements during Explosive Isometric Contractions, with Special Reference to EMG Normalisation Techniques

Published as:

3.1 Introduction

The ability to rapidly develop muscular force is an important aspect of muscle performance that is fundamental to sports activities such as sprinting, jumping and punching and is also considered important for preventing injuries after mechanical perturbation (Aagaard et al., 2002a; Minshull et al., 2007; Tillin et al., 2010). These explosive movements involve contraction times on the order of 50-250 ms (Haff et al. 1997) which is shorter than the time required for development of maximum voluntary force (MVF, $\geq$ 300 ms, Thorstensson et al., 1976). Therefore, it is important to be able to reliably assess neuromuscular function during explosive contractions.

Explosive muscle strength is typically assessed by measuring either the rate of force development (RFD) or area beneath (force-time integral) the force–time curve during isometric explosive voluntary contractions. Although MVF has been documented widely to have excellent reliability (ICC > 0.95; coefficient of variation [CV] < 4%, Thorstensson et al., 1976; Strass, 1997; Kollmitzer et al., 1999; de Ruiter et al., 2004; Place et al., 2007) the between-session reliability of explosive force production has received little attention. The between-session reliability of RFD in the plantar flexors has been documented, but only in the early phase of the contraction (5- 40% MVF, Clark et al., 2007). Others have noted some reliability data for knee extensor RFD during intervention or comparative studies (Clark et al., 2007; Place et al., 2007; Tillin et al., 2010) but there has not been a comprehensive attempt to assess the reliability of RFD measurements.

Assessment of involuntary RFD in response to evoked contractions can give insight into the intrinsic capacity of the muscle-tendon unit (MTU) for explosive force production without the influence of voluntary control and is therefore thought to reflect muscle morphology and tissue mechanics (Almeida et al., 1994; Harridge et al., 1996; Oda et al., 2007). These intrinsic contractile properties have often been investigated by examining the response to a single electrical impulse and subsequent twitch contractions (Van Cutsem et al., 1998; Rich & Cafarelli, 2000; Andersen & Aagaard, 2006; Pucci et al., 2006). However, research has shown that maximal RFD can only be achieved at high frequencies of stimulation (Buller & Lewis, 1965). Therefore, single twitch contractions may provide less insight into the intrinsic explosive capacity of the MTU than high frequency contractions such as an evoked octet (8 pulses at 300 Hz), which has been found to evoke the maximum capacity for RFD (de Ruiter et al., 2004, 2006). Although, the reliability of supramaximal twitch contractions has been

examined (Clark et al., 2007; Place et al., 2007; Tillin et al., 2011), the reliability of supramaximal evoked octet contractions for determining maximal RFD is unknown.

Volitional neural drive to skeletal muscle is considered a crucial determinant of maximal and explosive muscle performance that is often assessed by measuring the amplitude of the surface electromyographic (EMG) signal (Hakkinen et al., 1995; Van Cutsem et al., 1998; de Ruiter et al., 2004, 2006; Tillin et al., 2010). While the between-session reliability of EMG amplitude has been assessed during maximal, submaximal and sustained isometric contractions (Yang & Winter, 1983; Rainoldi et al. 2001; Mathur et al. 2005; Clarke et al., 2007), its reliability during explosive isometric contractions has not been documented. The absolute EMG amplitude is influenced by a multitude of intrinsic and extrinsic factors that are unrelated to the level of muscle activation (de Luca, 1997), therefore, normalisation of the EMG signal is considered essential for comparisons between participants as well as for repeated measurement sessions with the same individual. The EMG amplitude during a task of interest has typically been normalised to the amplitude obtained from a reference contraction, although there is no general agreement as to the best normalisation method (Perry, 1992).

Isometric maximum voluntary contractions (MVC) are the most widely used (De Luca, 1997) and advocated (Burden, 2010) reference method. There is however, no standard procedure for assessing the EMG during MVCs in order to provide a reliable reference for normalisation. Some authors have used the peak EMG (Bruhn et al., 2006) irrespective of the time it occurs, whereas others have used EMG at MVF (Gruber & Gollhofer, 2004), but there appears to be no evidence as to which is superior. Additionally, there is no consensus on the optimal window length that should be used when processing the amplitude of the EMG signal during MVCs, and a range of window lengths has been reported (100 ms, de Ruiter et al., 2004, 2006; 200 ms, Gruber & Gollhofer, 2007; 500 ms, Place et al., 2007).

Alternatively, de Luca (1997) suggested that sub-maximal contractions at $\leq 80\%$ MVF may provide more stable EMG amplitude than MVCs, and there is evidence that the EMG amplitude during sub-maximal contractions exhibits superior between-session reliability (Yang & Winter, 1983; Rainoldi et al., 1999). Furthermore, the EMG response to an evoked maximal compound muscle action potential (M_{max}) has also been suggested as an alternative normalisation method (Araujo et al., 2000; Gandevia et al., 2001). As the M_{max} response is not confounded by volitional activation, it may provide superior reliability to traditional

normalisation techniques (i.e. to EMG during MVCs). The peak-to-peak amplitude of M_{max} (M_{max} P-P) has been used to normalise EMG during explosive and maximum voluntary contractions (Van Cutsem et al., 1997; Tillin et al., 2010; Tillin et al., 2011). Although, the reliability of EMG normalised to M_{max} P-P has been investigated during MVCs (CV, 12.1-13.4%, ICC, 0.45-0.90, Place et al., 2007) there has been no investigation during explosive contractions. Recent research has also suggested that the cumulative area of the M_{max} (M_{max} Area) may provide a more reliable measurement parameter than M_{max} P-P (Tucker & Turker, 2007), but the reliability of this parameter in either absolute terms, or when used as a normalisation method for volitional EMG has not been assessed.

The aim of this study was to determine the between-session reliability of: (i) knee extensor measurements of voluntary and evoked explosive force production, and (ii) agonist EMG at maximal force and during the initial explosive phase of contraction, with consideration of the most reliable method of EMG normalisation. Specifically, the reliability of absolute EMG from maximal, sub-maximal and explosive contractions was determined, and the influence of EMG window length and selection of EMG epoch during MVCs (at peak EMG vs. EMG at MVF) was considered. Finally, the reliability of using different reference methods for EMG normalisation was compared, including M_{max} P-P, M_{max} Area, and volitional EMG during maximum and sub-maximum contractions.

3.2 Methods

3.2.1 Participants

Thirteen healthy male participants gave written informed consent prior to participating in this study, which had local ethics committee approval (mean ± SD: age, 22 ± 3 yr; height, 1.78 ± 0.04 m; body mass, 70.6 ± 9.2 kg). The participants were physically active, healthy, injury free and had not taken part in any form of lower body resistance exercise in the previous 12 months.

3.2.2 Overview

Each participant attended the laboratory on four separate occasions, once for familiarisation and then for a further three main trials, during which measurements were recorded from the preferred leg. Trials were seven days apart and at a consistent time of day for each participant. The three trials involved the same protocol with measurements of isometric knee extension force and surface EMG of the superficial quadriceps during maximal, sub-maximal and explosive voluntary efforts, as well as evoked twitch and octet contractions.

3.2.3 Measurement Trials

3.2.3.1 Measurements

Participants were firmly secured in the strength testing chair (Parker et al., 1990; Bosjen-Moller et al., 2005) with waist and shoulder straps. The hip and knee angle were fixed at 100° and 85° respectively. An ankle strap was placed 2 cm proximal to the medial malleolus, in series with a calibrated U-shaped aluminium strain gauge (linear response up to 1000 N, Jones & Parker, 1989) that was positioned perpendicular to tibial alignment. The force signal was amplified (x 500), interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK), and sampled at 2000 Hz with a PC utilizing Spike 2 software (CED, Cambridge, UK). Real-time biofeedback of the force response was provided on a computer monitor.

The femoral nerve was stimulated electrically (via a constant current, variable voltage stimulator; DS7AH, Digitimer Ltd., UK) with square wave pulses (0.1 ms in duration) to elicit: (i) single pulse twitch contractions, to facilitate measurement of compound muscle action potentials (*M*-waves); and (ii) octet stimulation (8 pulses at 300 Hz) to determine the muscle's maximal capacity for RFD. The anode (carbon rubber electrode, 7 x 10 cm; Electro-Medical Supplies, Greenham, UK) was taped to the skin over the greater trochanter. The cathode, a custom-adapted stimulation probe 1 cm in diameter (Electro-Medical Supplies, Wantage, UK), which protruded 2 cm perpendicular from the centre of a plastic base (4 x 5 cm), was taped to the skin over the femoral nerve in the femoral triangle. The precise location of the cathode was determined as the position which elicited the greatest twitch response for a particular submaximal current.

Surface EMG was recorded from the rectus femoris (RF), vastus lateralis (VL) and vastus medialis (VM) using a Delsys Bagnoli-4 EMG system (Input impedance, $> 10^{15}\Omega$; common mode rejection ratio, 93 dB, Delsys, Boston, USA). Following preparation of the skin (shaving, light abrading and cleansing with 70% ethanol), double differential electrodes (1 cm inter-electrode distance, DE-3.1, Delsys) were attached over each muscle using adhesive interfaces. To standardise position between sessions and normalise across individuals, the electrodes were positioned in the centre of the muscle belly parallel to the presumed orientation of the muscle fibers at specific lengths along the thigh (from the lateral epicondyle of the femur to the greater trochanter: VM, 20%; VL, 40%; RF, 60%). Skin-electrode impedance was assessed on each occasion and maintained at a consistent level (within 0.5 MΩ) within individuals and at a value < 5 MΩ for all participants. The reference electrode was placed on the patella of the same limb. EMG signals were amplified (x1000; differential amplifier, 20 – 450 Hz) and synchronized with force data by recording at 2000 Hz with the same analogue to digital converter and PC as the force signal. During off-line analysis the signals were band-pass filtered in both directions between 6-500 Hz using a 2nd order Butterworth digital filter.

3.2.3.2 Protocol

Once the participants were firmly secured in the testing chair they performed a series of voluntary isometric contractions of the knee extensors: (i) MVCs to assess maximum voluntary force (MVF) (ii) submaximal contractions at 80%MVF; and (iii) explosive contractions to assess explosive force production. MVCs and explosive contractions were separated, as each has a distinct purpose which requires different instructions and participant attention that can influence the performance outcome (Sahaly et al., 2001).

Therefore, following a warm-up of progressive sub-maximum contractions, participants performed four MVCs, each separated by ≥ 30 s, in which participants were instructed to push 'as hard as possible' for 3 s. Biofeedback was provided by displaying the force trace on a monitor with an on-screen cursor used to mark maximum force. Participants were encouraged to exceed this target on subsequent attempts, and verbal encouragement was provided during and between each maximal contraction. Knee extensor MVF was the greatest force achieved by the participant in any of the MVCs. The EMG signal of the three agonist muscles was assessed with: (i) a 500 ms root mean square (RMS) epoch around MVF (250

ms either side, 'EMG@MVF'); and (ii) the 500 ms epoch that provided the highest RMS value of each muscle irrespective of force 'peak EMG'. RMS EMG values for each muscle were normalised to both M_{max} P-P and M_{max} Area (detailed below). Absolute and normalised EMG values from the three agonist muscles were averaged to give a mean for the whole quadriceps.

The effect of window length on the measured variability between trials of the EMG was examined using repeated measures on single trials. When comparing between random signals as the window length increases mean variability between the signals should decrease according to a power law. However, the EMG signal is only pseudo-random and non-stationary, so variability may increase on longer time scales. A flat 1.5 s segment of the force-time curve was selected for a single trial and divided up into multiple non-overlapping windows. This was done on the same signal for 14 different window lengths (25, 50, 75, 100, 125, 150, 200, 250, 300, 350, 400, 500, 600, 700 ms). A custom MATLAB script (MathWorks Natick, Massachusetts, U.S.A) then computed the coefficient of variation (CV) between windows for groups of the same window length. This meant that the CV was calculated from the same signal with different window lengths as the only variable. This was repeated for each of the MVC attempts with the highest force, for all participants, from each of their three measurement session, and the mean CV for each window length was calculated. A power curve of the form $y = a \times b^x + c$ was fitted to the mean data using a least squares technique in MATLAB.

Participants then performed 3-4 submaximal isometric contractions at 80% MVF. The desired force level was displayed on a computer screen, and participants were instructed to maintain this level of force as steadily as possible for 4-5 s. Mean force and EMG of the agonist muscles during these contractions were assessed with a 500 ms epoch ('EMG@80%MVF') during a stable segment of the force-time curve, where force was ~80% MVF.

Participants then performed 10 explosive voluntary contractions (separated by 20 s). For each contraction, participants were instructed to extend the knee as 'fast' and as hard as possible, with an emphasis on fast, for ~1 s from a relaxed state (Sahaly et al., 2001). Participants were instructed to avoid any countermovement or pre-tension. To determine if countermovement had occurred, the resting force level was displayed on a sensitive scale. In order to provide biofeedback on their explosive performance, the slope of the force time curve (2 ms time constant) was displayed throughout the explosive contractions, and the peak slope of their

best attempt was highlighted with an on-screen cursor. Finally, a visual marker on the screen depicted 80% of MVF during the contractions, and participants were expected to achieve this level of force during each explosive contraction. The three contractions with the highest peak slope and no discernible countermovement or pre-tension (change in force of > 0.5 N in the preceding 100 ms) were used for analysis, and all measurements were averaged across these three contractions. Force was assessed at 50, 100, and 150 ms, from the onset of contraction. RFD was measured as the peak slope (pRFD) and in time windows of 0-50, 50-100 and 100-150 ms from the onset of force. The force-time integral (the area beneath the force-time curve) was also assessed in windows of 0-50, 0-100 and 0-150 ms from the onset of force. Force was reported in absolute terms and normalised to MVF i.e., force/MVF. The RMS EMG was measured in windows of 0-50, 0-100 and 0-150 ms from the onset of EMG activity in the first agonist muscle to be activated. The RMS EMG values for each muscle were normalised to each of the different reference measurements [M_{max} P-P, M_{max} Area (detailed below), EMG@MVF, EMG@80%MVF] before being averaged across the muscles to give a mean value for the quadriceps.

Twitch contractions were elicited at incremental current intensities until a simultaneous plateau in the force and M-wave response was observed. Thereafter, the current was increased by 20%, and three supra-maximal twitches were elicited (separated by 12 s). The average peak-peak amplitude (M_{max} P-P) and total M_{max} area (M_{max} Area) of these three supramaximal M-waves was determined for each muscle and was used for normalisation. The M_{max} area was calculated as the total cumulative area of the M_{max} response from the onset of EMG following the stimulation artefact, to the point at which the EMG signal returned to baseline value (visually identified). The twitch force response was analyzed for peak force (PF), force at 50 ms, pRFD (2 ms time constant), force-time integral (0-50 ms), time to peak force (TPF), and half relaxation time (HRT) and was averaged across the three contractions.

Octet contractions were then elicited at incremental intensities up to the supramaximal current level used for M_{max} measurements. Three supramaximal octet contractions (separated by 12 s) were elicited, and the average of the three was taken for analysis. Analysis included measurement of PF, force at 50 ms, pRFD (2 ms time constant) and force-time integral (0-50 ms). As an additional measure of overall neural efficacy, force production during the voluntary explosive contractions was compared to octet force and force-time integral over 50 ms to assess the participant's voluntary activation capacity and was reported as voluntary percentage of octet performance.

3.2.4 Data Analysis

For the explosive voluntary and electrically-evoked contractions, identification of force and EMG onsets was made manually (visual identification). The same investigator identified signal onsets with a constant y-axis scale of ~ 1 N and 0.1 mV, for force and EMG respectively, and an x-axis scale of 500 ms. A vertical cursor was then placed on the onset and viewed at a higher resolution to determine its exact location (~ 0.5 N and 0.05 mV for force and EMG axes, respectively using an x-axis of 25 ms). Manual identification of signal onsets is considered the "gold standard" method (Allison, 2003; Moretti et al., 2003; Pain & Hibbs 2007; Pulkovski et al., 2008).

To determine the level of reliability of all the aforementioned measurement parameters the intraclass correlation coefficient (ICC) (two-way random effects model, single measure reliability) was employed. Within-participants reliability was calculated using the mean intra-participant coefficient of variation [(SD ÷ Mean) × 100, CV_W]. Both the ICC and CV_W have been widely used to assess reliability, as they both offer different measures of reliability. The CV_W is a measure of within-participant reliability which provides a measure of variability of an individual's value, while the ICC indicates the percentage of the global variance that can be attributed to the variability between participants. ICC values were interpreted as 'excellent' 0.80 – 1, 'good' 0.6 – 0.8 and poor < 0.60 (Bartko, 1966). The between-participant coefficient of variation [(group SD ÷ group mean) × 100, CV_B] was identified for each single session and then averaged across the three sessions, before being used to determine the impact of EMG normalisation on the variability of EMG measurements between participants.

To determine if there was a significant difference between testing days, a one-way repeated measures analysis of variance (ANOVA) was performed for each measurement parameter. If sphericity was violated, then Huynh-Feldt corrected values were used. To determine if differences in CV_W values between measurement parameters were significant, one-way ANOVA with Bonferroni *post-hoc* adjustments or paired *t*-tests were performed. One way ANOVA or paired *t*-tests were performed to examine if the within-participant reliability was different when comparing: i) voluntary explosive force/RFD at different time points/periods (e.g. 50 vs. 100 vs. 150 ms); ii) evoked octet vs. voluntary measures of force and force-time integral over 50 ms; (iii) the twitch vs. octet force responses; iv) voluntary vs. electrically-evoked EMG measures; v) EMG measured across different time windows (0-50 vs. 0-100 vs. 0-150 ms) and vi) absolute EMG vs. different normalisation methods (M_{max} P-P vs. M_{max}

Area vs. EMG@MVF vs. EMG @80% MVF). Data are reported as mean ± SD, and an alpha level of 5% was accepted as statistically significant.

3.3 Results

3.3.1 Force

3.3.1.1 Maximal and Submaximal Voluntary Contractions

The MVF values showed a very high level of reliability across measurement sessions (551 ± 92 vs. 566 ± 86 vs. 567 ± 93 N, CV_W, 3.3%, ICC, 0.95). For the submaximal contractions the assessed 500 ms epoch was very close to the intended contraction intensity (80% MVF) and was a consistent proportion of MVF across the three sessions (79.8 ± 0.5 vs. 80.1 ± 0.4 vs. 79.6 ± 1.3 % MVF, CV_W, 5.0%).

3.3.1.2 Voluntary Explosive Force Production

Force, relative force and force-time integral measurements at all 3 time points were consistent across the three sessions (50, 100 and 150 ms; $P > 0.35$; Table 3.1). For these three parameters the reliability of the very early phase of explosive force development (50 ms) demonstrated poor CV_W values (16.6-18.7%), which were significantly higher than for the CV_W values for 100 (6.4-9.8%, all $P < 0.005$) and 150 ms (5.1-8.4%, all $P < 0.001$). However, the ICC values for these three parameters during the initial 50 ms period were good (0.75-0.80). The RFD between 50-100 ms was the most reliable RFD window. The CV_W values were good (6.8%) and were significantly lower than during 0-50 (16.6%, $P < 0.0005$) and 100-150 ms (10.5%, $P = 0.026$).

Table 3.1 Reliability of voluntary explosive force production. Group data are reported as mean ± SD (N =13) for each of three measurement sessions.

	Session			CV_W	ICC	P
	1	**2**	**3**			
Force (N)						
50 ms	90 ± 45	91 ± 33	83 ± 42	16.6	0.80	0.494
100 ms	301 ± 79	308 ± 67	300 ± 68	6.4	0.91	0.616
150 ms	404 ± 77	418 ± 68	414 ± 81	5.1	0.90	0.352
Relative force						
50 ms (% MVF)	16.5 ± 7.5	16.7 ± 5.7	15.1 ± 6.9	18.0	0.75	0.465
100 ms (% MVF)	55.6 ± 9.6	55.9 ± 7.7	54.7 ± 6.8	7.1	0.69	0.782
150 ms (% MVF)	75.2 ± 7.9	76.2 ± 6.5	75.5 ± 6.9	5.1	0.59	0.847
50 ms (% octet)	41.4 ± 19.1	45.8 ± 16.8	37.2 ± 18.2	18.8	0.80	0.063
RFD (N.s^{-1})						
0-50 ms	1778 ± 891	1829 ± 653	1664 ± 844	16.6	0.80	0.494
50-100 ms	4237 ± 1073	4324 ± 1042	4343 ± 926	6.8	0.90	0.683
100-150 ms	2074 ± 315	2200 ± 369	2277 ± 463	10.5	0.62	0.110
pRFD	6292 ± 1624	6413 ± 1430	6190 ± 1289	7.2	0.90	0.352
Force-time integral (N.s)						
0-50 ms	1.12 ± 0.56	1.16 ± 0.48	1.08 ± 0.58	18.7	0.77	0.731
0-100 ms	11.0 ± 3.1	11.3 ± 2.7	11.0 ± 3.4	9.8	0.82	0.836
0-150 ms	28.1 ± 6.2	29.2 ± 6.1	29.2 ± 6.8	8.4	0.77	0.594
50 ms (% octet)	24.6 ± 12.3	27.1 ± 12.0	22.2 ± 12.0	22.0	0.77	0.175

CV_W, within-participant coefficient of variation; ICC, intraclass correlation coefficient; P, one-way ANOVA p value; N, newtons; MVF, maximum voluntary force; RFD, rate of force development; pRFD, peak rate of force development.

3.3.1.3 Electrically-evoked Explosive Force Production and M_{max}

For the evoked force responses, one participant did not complete the octet measurements in all three sessions, therefore, evoked force responses are reported as N = 12. Evoked octet force responses were consistent across measurement sessions (all, P > 0.182) with very good CV_W values (5.4-7.3%) and good ICC values (0.71-0.83; Table 3.2). The octet CV_W demonstrated significantly lower CV_W values than voluntary measures of explosive force production at 50 ms (force, 5.4 ± 3.8 vs. 16.6 ± 7.4%, P = 0.001; force-time integral, 6.7 ± 4.8 vs. 18.7 ± 9.7%, P = 0.001), but was not different for pRFD (Octet, 7.3 ± 3.7 vs. voluntary, 7.2 ± 3.5%, P = 0.739).

Twitch force responses were less stable; pRFD changed significantly across measurement sessions (P = 0.023), with a tendency for PF (P = 0.072) and force at 50 ms (P = 0.075) to also differ between sessions (Table 3.2). Twitch pRFD was less reliable than octet pRFD (CV_W, 11.3 ± 4.6 vs. 7.3 ± 3.7%, P = 0.017), however twitch and octet measures of peak force, force at 50 ms and force-time integral 0-50 displayed similar reliability (CV_W all, P ≥ 0.41). The time course of the twitch response was stable across measurement sessions with very low CV_W values for TPF and HRT (3.6 & 4.9%, respectively) and excellent ICC values (0.89 & 0.86, respectively).

M_{max} P-P and M_{max} Area differed significantly across measurement sessions (P = 0.025, and P = 0.033, respectively), and CV_W values were moderate to poor (14.1 & 13.7 %, respectively). The ICC (0.95 & 0.93, respectively) and CV_B (58.7 & 54.8 %, respectively) values were high for both measures.

Table 3.2 Reliability of evoked responses to supramaximal twitch and octet stimulation. Group data are reported as mean ± SD (N = 12) for the three measurement sessions.

	Session			CV_W	ICC	P
	1	**2**	**3**			
OCTET						
PF (N)	351 ± 62	350 ± 53	371 ± 53	7.2	0.76	0.149
Force 50 (N)	213 ± 34	213 ± 33	223 ± 31	5.4	0.83	0.240
F-T integral 0-50 (N.s)	4.59 ± 0.85	4.53 ± 0.81	4.69 ± 0.70	6.7	0.77	0.632
pRFD (N.s^{-1})	8534 ± 1616	8356 ± 1170	8922 ± 933	7.3	0.71	0.181
TWITCH						
Force						
PF (N)	109 ± 23	107 ± 18	115 ± 21	7.6	0.83	0.072
Force 50 (N)	88 ± 16	86 ± 14	93 ± 14	8.3	0.76	0.075
F-T Integral 0-50 (N.s)	1.89 ± 0.42	1.84 ± 0.33	1.98 ± 0.39	8.0	0.75	0.225
pRFD (N.s^{-1})	3013 ± 771	2834 ± 654	3228 ± 671	11.3	0.80	0.021
Time course						
HRT (ms)	73 ± 12	73 ± 14	76 ± 13	4.9	0.86	0.175
TPF (ms)	82 ± 11	85 ± 9	83 ± 9	3.6	0.89	0.142
M_{max}						
P-P amplitude (mV)	3.02 ± 2.01	3.33 ± 1.82	3.58 ± 1.97	14.1	0.95	0.025
Area (mV.s)	0.014 ± 0.008	0.016 ± 0.008	0.017 ± 0.010	13.7	0.93	0.033

CV_W, within-participant coefficient of variation; ICC, intraclass correlation coefficient; P, one-way ANOVA p value; PF, peak force; N, newtons; Force50, force at 50 ms; F-T integral 0-50, Area beneath the force-time curve from 0 to 50 ms; pRFD, peak rate of force development; HRT, half relaxation time; TPF, time to peak force; Mmax, maximum compound action potential; P-P, peak to peak; mV, millivolts.

3.3.1.4 Voluntary Activation Capacity (voluntary: octet performance)

The CV_W values for measures of voluntary activation capacity (i.e. voluntary performance compared to the octet) using either force (18.8%) or force-time integral (22.0%) were poor even though the evoked force production from the octet was very reliable. There was also a tendency for the percentage of octet force achieved voluntarily at 50 ms to be significantly different across sessions (P = 0.063). However, the ICCs were good (force, 0.77, force-time integral, 0.80).

3.3.2 EMG

3.3.2.1 EMG Window Length

The variability of EMG (CV_W) measured with different window lengths was plotted in Figure 3.1, and the bin to bin CV data for EMG fit almost perfectly with a power function ($r^2 =$ 0.999, Sum of Squared Errors 0.2297):

$$y = 101.2.x^{-0.577} + 3.451$$

The variability for 25 ms window length was high (CV_W, 19.3 ± 4.2%) and was significantly greater than all other window lengths (P < 0.001). Increasing the window length resulted in a significant decline in EMG variability for all window lengths up to 200 ms (8.2 ± 3.4%), which was the shortest window length in which there was no further significant reduction in EMG variability for any increase in EMG window length (all, P > 0.108).
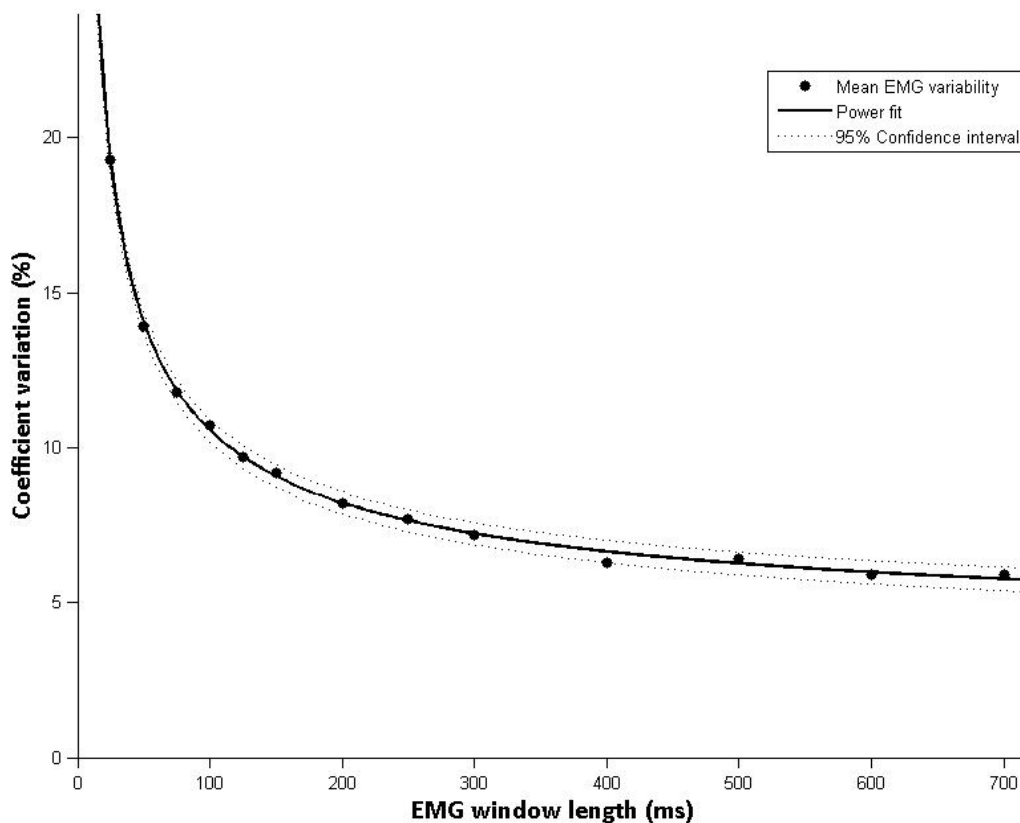
Figure 3.1 Variability of EMG amplitude during MVCs as a function of EMG window length. Data are mean within-contraction coefficient of variation from 37 MVCs. Solid markers represent mean CV values for each respective EMG window length. The solid curve represents the power function ($r^2 = 0.999$), and the dotted lines represent the 95% confidence intervals.

3.3.2.2 Absolute EMG during Reference Measures

Absolute EMG amplitude during the maximum (EMG@MVF, peak EMG) and sub-maximum contractions (EMG@80%MVF) was consistent across measurement sessions (Table 3.3). The reliability of EMG@MVF and peak EMG were very similar, as in fact was EMG@80%MVF (CV_W, 16.6-17.1%; ICC, 0.89-0.91). Furthermore, the reliability of these three reference EMG measures and evoked M_{max} P-P and M_{max} Area responses were similar (P = 0.466).

3.3.2.3 Normalisation of EMG@MVF

Given the similar reliability of EMG@MVF and peak EMG, for simplicity, only the former was normalised to M_{max} parameters. EMG@MVF normalised to M_{max} P-P differed across the three sessions (P = 0.034), but this was not the case when it was normalised to M_{max} area (P = 0.103). The reliability of EMG@MVF normalised to either M_{max} P-P (CV_W, 15.5 ± 9.0%) or M_{max} area (15.5 ± 8.3%) was no better than absolute EMG@MVF (16.6 ± 7.8%, P = 0.699). Normalisation of EMG@MVF did, however, dramatically reduce the between-participant variability (CV_B absolute, 62.5%; normalised to M_{max} P-P, 33.6%; normalised to M_{max} Area, 27.0%).

Table 3.3 Reliability and inter-participant variability of absolute EMG RMS amplitude during volitional reference measures (maximum and submaximum voluntary contractions). During maximum contractions peak EMG and EMG @ MVF were assessed. EMG @ MVF was also normalised to M_{max} parameters. Data are reported as mean ± SD (N = 13) for each of the three measurement sessions.

	Session			CV_W	ICC	P	CV_B
	1	**2**	**3**				
Absolute EMG during volitional reference measures (mV)							
Peak EMG	0.20 ± 0.12	0.24 ± 0.12	0.23 ± 0.15	16.6	0.89	0.092	60.0
EMG@MVF	0.19 ± 0.12	0.21 ± 0.12	0.21 ± 0.14	16.6	0.90	0.503	62.5
EMG@80% MVF	0.12 ± 0.07	0.13 ± 0.06	0.13 ± 0.08	17.1	0.91	0.236	54.5
EMG @ MVF normalised to:							
M_{max} P-P (%)	8.9 ± 3.0	8.7 ± 3.2	7.5 ± 2.3	15.5	0.80	0.034	33.6
M_{max} Area.s^{-1}	17.9 ± 4.4	17.2 ± 5.5	15.6 ± 3.9	15.5	0.69	0.103	27.0

CV_W, within-participant coefficient of variation, ICC, intraclass correlation coefficient; P, one way ANOVA p value; CV_B, between participant coefficient of variation; Absolute EMG, un-normalised root mean squared EMG amplitude; peak EMG, maximum 500ms root mean squared EMG epoch during the maximum voluntary contraction; EMG@MVF, 500ms epoch of EMG around maximum voluntary force; M_{max}, maximum compound action potential; P-P, peak to peak

3.3.2.4 EMG during Explosive Voluntary Contractions

There were tendencies for absolute EMG values during the explosive voluntary efforts to be different across measurement sessions for all three time periods from EMG onset (0-50 ms, P = 0.053; 0-100 ms, P = 0.088; 0-150 ms, P = 0.059). When EMG values were normalised to EMG@MVF, EMG@80%MVF or M_{max} Area there were no changes across the 3 measurement sessions, however normalisation of EMG during the shortest time period (0-50 ms) to M_{max} P-P was not stable across the sessions (P = 0.025). Absolute and normalised methods of expressing EMG amplitude produced similar CV_W values for all 3 time windows, with no differences between any of the methods (P > 0.85). Collapsing individual CV_W values for EMG from all four normalisation methods across the different time windows (0-50 vs. 0-100 vs. 0-150 ms) demonstrated that the averaged CV_W values were significantly higher during 0-50 (19.6 ± 9.3%) than for 0-100 (15.4 ± 6.9%, P = 0.001) or 150 ms (15.7 ± 6.7%, P = 0.001).

Table 3.4 Reliability and inter-participant variability of EMG RMS amplitude during explosive voluntary contractions assessed in 3 time windows from EMG onset (0-50, 0-100 and 0-150 ms). Absolute values are presented and normalised to four reference measures: EMG during maximum (EMG@MVF) and submaximal voluntary (EMG@80% MVF) contractions, and evoked M_{max} peak-to-peak amplitude (M_{max} P-P) and cumulative area (M_{max} Area). Data are reported as mean $\pm$ SD (N = 13) for each of the three measurement sessions.

	Session			CV_W	ICC	P	CV_B
	1	**2**	**3**				
0-50 ms							
Absolute EMG (mV)	0.093 ± 0.084	0.105 ± 0.095	0.091 ± 0.082	17.6	0.97	0.053	101.5
Normalised to:							
EMG@MVF (%)	46.6 ± 24.6	47.5 ± 23.9	44.0 ± 22.5	20.5	0.80	0.690	51.4
EMG@80% MVF (%)	62.8 ± 33.7	65.6 ± 35.9	64.2 ± 33.5	19.1	0.81	0.894	53.5
M_{max} P-P (%)	3.54 ± 1.68	3.74 ± 1.80	2.95 ± 1.61	19.8	0.84	0.025	50.0
M_{max} Area. s^{-1}	7.69 ± 4.40	7.89 ± 4.64	6.45 ± 4.14	21.2	0.85	0.096	60.1
0-100 ms							
Absolute EMG (mV)	0.143 ± 0.090	0.159 ± 0.080	0.154 ± 0.085	14.5	0.95	0.088	56.1
Normalised to :							
EMG@MVF (%)	73.3 ± 22.6	75.7 ± 17.4	77.7 ± 22.9	16.8	0.55	0.740	27.7
EMG@80% MVF (%)	98.7 ± 28.4	$102. \pm 21.4$	114 ± 33.6	16.3	0.58	0.113	26.4
M_{max} P-P (%)	5.86 ± 1.98	6.16 ± 1.56	5.49 ± 1.77	14.6	0.68	0.248	30.5
M_{max} Area. s^{-1}	12.4 ± 4.6	12.4 ± 3.8	11.8 ± 4.7	14.4	0.78	0.703	35.5
0-150 ms							
Absolute EMG (mV)	0.161 ± 0.094	0.186 ± 0.086	0.178 ± 0.102	16.0	0.92	0.059	54.1
Normalised to :							
EMG@MVF (%)	82.3 ± 20.5	87.7 ± 17.7	86.3 ± 20.9	14.1	0.53	0.582	23.1
EMG@80% MVF (%)	111 ± 24.1	118 ± 17.5	126 ± 26.8	13.5	0.55	0.072	19.3
M_{max} P-P (%)	6.71 ± 2.05	7.18 ± 1.70	6.14 ± 1.66	16.0	0.57	0.114	27.1
M_{max} Area. s^{-1}	14.0 ± 4.4	14.4 ± 3.8	13.1 ± 4.3	13.5	0.74	0.365	30.1

CV_W, within-participant coefficient of variation, ICC, intraclass correlation coefficient; P, one way ANOVA p value; CV_B, between participant coefficient of variation; EMG, surface electromyography root mean squared amplitude; Absolute EMG, un-normalised EMG; MVF, maximum voluntary force; M_{max}, maximum compound action potential; P-P, peak to peak amplitude;

3.4 Discussion

The aim of this investigation was to examine the reliability of explosive voluntary and evoked force production and to consider the reliability of absolute and normalised EMG during maximal and explosive voluntary contractions in order to determine the optimal normalisation method. The main findings of the study were that the early phase of voluntary explosive force production was highly variable for an individual but became more consistent from 100 ms onwards. On a group level, explosive voluntary force measurements at all time points were stable and consistent between sessions. The absolute EMG amplitude was highly variable for individuals between measurement sessions for both maximal and explosive voluntary contractions and electrically-evoked measurements. Surprisingly, none of the normalisation techniques improved significantly the within-participant reliability (CV_W) of EMG amplitude measurements for either maximal or explosive contractions. However, all the EMG normalisation techniques reduced the between-participant variability (CV_B) compared to absolute values. Normalised group EMG values were consistent during both maximal and explosive contractions, providing M_{max} P-P was not used as the normalisation method. The octet was a reliable method of examining the maximal evoked explosive performance of the MTU and was more reliable than the twitch for assessing peak RFD. Voluntary activation using explosive voluntary compared to octet force/ force-time integral was not more reliable than normalised EMG amplitude during explosive voluntary contractions.

3.4.1 Reliability of Force Measurements

The between-session reliability of MVF was excellent (CV_W, 3.3%, ICC, 0.95), which further supports previous research that has demonstrated similar findings (Kollmitzer et al., 1999; Thorstensson et al., 1977; Place et al., 2007). The different measures of explosive force production (force, relative force, force-time integral) demonstrated consistent group values across the measurement sessions with similar within-participant (CV_W) reliability. These measures of explosive force production on an individual basis were most variable in the very early phase (e.g. force 50 ms, CV_W 16.6%) but became more reliable at 100 (6.4%) and 150 ms (5.1%). The pattern and magnitude of these reliability values was similar to another recent report (Force: 50 ms, 12.8%; 100 ms, 5.3% and 150 ms, 4.8%, Tillin et al, 2011). Therefore, we conclude that the early phase of explosive force production (50 ms) is variable on an

individual level across measurement sessions, but there is good reliability from 100 ms onwards. When an individual's explosive strength is assessed over the initial 50 ms with the intention of determining longitudinal changes (e.g. following an intervention), the findings should be interpreted with caution.

Evoked responses using the octet were highly reproducible on a group and individual level (CV_W, 5.4-7.3%), thus this measurement offers a reliable method of examining the explosive evoked capacity of the knee extensors. Octet force and force-time integral CV_W values at 50 ms were lower than for the respective voluntary measures, thus evoked explosive force production in the first 50 ms is more reliable than voluntary performance during this early phase of contraction. The CV_W values of the twitch force responses were in line with those previously reported for the knee extensors (Place et al., 2007; Tillin et al., 2011) and lower than those reported for the plantar flexors (Clarke et al., 2007). The time course of the evoked twitch response was very reliable across measurement sessions, but the amplitude was less stable, with tendencies for changes in PF and force after 50 ms. Octet pRFD was more reliable than twitch pRFD on an individual basis (CV_W, 7.3 vs. 11.3%), and it was more stable on a group basis (twitch pRFD differed across measurement sessions). Therefore, the octet provides a more reliable method for examining the intrinsic contractile properties of the MTU and is recommended for future research.

3.4.2 Reliability of EMG during Reference Measures and Normalisation of EMG during MVCs

The window length analysis data fit a declining power function almost perfectly, demonstrating that a longer EMG window length reduces the EMG variability. In this study sample, from 200 ms onwards there was no further significant decline in EMG variability with increasing window length, which is in agreement with a previous report (Vint & Hinrichs, 1999). This indicates that, where possible, a minimum 200 ms window length should be employed during isometric voluntary contractions to maximize the reliability of EMG measurements.

As force and EMG are related, stable and reliable force measurements are required for reliable EMG measurements, and these criteria were satisfied for the volitional maximal and submaximal reference contractions in this study. The absolute measures of group EMG

amplitude during maximum (peak EMG, EMG@MVF) and sub-maximum contractions (EMG@80%MVF) were consistent across measurement sessions. However, unlike MVF, individual absolute EMG values were highly variable between measurement sessions (CV_W 16.6 – 17.1%), which is similar to previous reports (Kollmitzer et al., 1999; Clarke et al., 2007). During maximal contractions the choice of measuring EMG amplitude at MVF or peak EMG had no influence on reliability. Additionally, EMG amplitude during submaximal contractions was no more reliable than during maximal efforts, which is in contrast to early work (Yang & Winter, 1983; Rainoldi et al., 1999) but supports more recent research (Netto et al., 2006; Burnett et al., 2007; Norcross et al., 2010). Therefore, on the basis of measurement reliability, we cannot conclude that any of these reference measures is superior. The group data for the M_{max} parameters (P-P and Area) were not consistent across measurement sessions and demonstrated similarly high individual variability between sessions as absolute volitional EMG measures. The M_{max} P-P ICC value was excellent (0.95) and similar to a previous report (Netto et al., 2006) which is likely a function of the high variability observed between participantss (CV_B, 58.7%). Within-participant reliability of M_{max} P-P was variable (CV_W, 14.1%), which contrasts with one study (CV_W, 7.5%, Clarke et al., 2007) but is similar to another (CV_W, 14.6-18.8, Place et al., 2007). The voluntary EMG signal amplitude depends on the membrane properties of the muscle fibers as well as the timing of the motor unit action potentials. Thus, it reflects both peripheral and central properties of the neuromuscular system. The features of the EMG signal depend on a myriad of non-physiological factors that are unrelated to neural activation (e.g. subcutaneous tissue, skin-electrode impendence, electrode location, motor unit synchronization, signal cancellation, Farina et al., 2004) and changes in some of these factors from session-to-session may explain the reliability findings. It is important to note that although every effort was made to ensure identical EMG repositioning prior to all repeat measurement sessions, in a couple of circumstances the markings from the previous session were not identifiable, and thus the positioning had to be re-measured. These slight changes in electrode location might have influenced the reliability of the absolute EMG signal.

Importantly, normalisation of absolute EMG amplitude at MVF to the evoked reference measures (M_{max} Area and P-P) did not improve the reliability of individual data, which has not previously been documented. Furthermore, group data for EMG@MVF normalised to M_{max} P-P was not consistent across sessions, although normalisation to M_{max} Area was stable between sessions. Place et al. (2007) also found EMG during MVCs normalised to M_{max} P-P

to vary significantly between sessions for one of the superficial quadriceps muscles (RF), but not the other two (VM and VL). However, normalisation to either M_{max} parameter substantially reduced the between-participant variability (~50%, Yang & Winter, 1984). Despite the absence of improved individual reliability values, normalisation of EMG during maximum isometric efforts to M_{max} Area is recommended for evaluating group comparisons or responses, as it substantially reduces between-participant variability.

It is unclear why normalising the absolute EMG signal did not improve the within-participant reliability. Although the issues discussed would be expected to influence the variability in EMG signal amplitude, normalisation of the EMG signal is expected to remove some of these factors (e.g., skin-electrode impedance, electrode placement). Therefore, the fact that normalisation does not reduce the within-participant variability suggests that the M-wave varies in a manner that is not associated with the variability in volitional EMG.

3.4.3 Reliability of EMG during Explosive Contractions

As with the maximal contractions, normalisation of EMG during explosive efforts to a range of reference measures (EMG during volitional maximum and submaximum contractions and evoked M_{max} parameters) did not significantly reduce the within-participant reliability. The CV_W values were lower for EMG over 0-100 and 0-150 ms compared to 0-50 ms, which could be explained by either more reliable neural function or as a consequence of the longer EMG window length. All methods of normalisation significantly reduced the CV_B values to a similar extent (~50%) compared to CV_B for absolute EMG amplitude, while not significantly impacting the within-participant reliability. This reduction in between-participant variation would be expected to increase the effect size and power of statistical comparisons between groups or repeated measures of the same group compared to absolute EMG, making normalised EMG a more sensitive measurement tool. Consequently, in terms of reliability it does not seem to be important which method of normalisation is used, providing the EMG is normalised. The exception however, was M_{max} P-P which resulted in the only significant difference across sessions. Therefore, based on this reliability data, and given the relative simplicity of collecting EMG@MVF (i.e. no electrical stimulation is required), this reference measure may be the normalisation method of choice for EMG recorded during explosive contractions. Although, in the present study we have not considered the validity of different normalisation techniques, when differences in neural activation at MVF might be expected

(e.g. strength training or following injury) then normalisation to M_{max} Area could be more appropriate.

Comparing voluntary and evoked explosive force production during the early phase of contraction has been used as a measure of assessing voluntary activation capacity (de Ruiter et al., 2004, 2006). It was thought that this may offer a more reliable measure of neural activation in the early phase of the contraction than surface EMG. We found that, although the octet was reliable, its use as a reference for voluntary performance did not offer a more reliable measurement method than EMG amplitude over the first 50 ms. Therefore, it appears that the early phase of voluntary activation and force production during explosive contractions is highly variable on an individual level regardless of the measurement methods used. As evoked explosive force was reliable, this likely reflects an inherent variability in neural drive.

In summary, explosive voluntary force production was reliable on a group level, but variable for an individual during the very early phase (50 ms) of contraction. From 100 ms onwards, voluntary explosive force production is reliable across measurement sessions. EMG during both voluntary maximal and explosive contractions was consistent across sessions for the group but was variable for individuals. Surprisingly normalisation of EMG did not improve this within-participant variability, but, as expected, it substantially reduced the variability between participants. The high intra-individual variability of EMG amplitude may limit its use to measuring group as opposed to individual responses to an intervention. For group comparisons, normalisation of EMG amplitude is recommended, specifically to M_{max} Area for EMG recorded during maximum contractions and to EMG@MVF for recordings made during explosive efforts. The evoked octet is recommended as a stable and reliable measurement tool for assessing maximal evoked explosive force capacity. However, use of the octet to calculate voluntary activation capacity during the initial phase of explosive efforts was no more reliable than absolute EMG amplitude.

CHAPTER 4

Neural and Contractile Contributions to Electromechanical Delay

4.1 Introduction

The capacity of the neuromuscular system for explosive force production is considered to be important for the performance of sporting movements such as jumping (Marcora & Miller, 2000; de Ruiter et al., 2006) and sprint running (Tillin et al., 2013), and in providing dynamic joint stability and ligament protection (Shultz & Perrin, 1999). There is evidence that injuries such as an anterior cruciate ligament rupture can occur within 50 ms of ground contact (Krosshaug et al., 2007). Consequently, time allowed for joint re-stabilisation following mechanical perturbation is limited. As such electromechanical delay (EMD, the time delay between the onset of myoelectrical activity and onset of force generation; Zhou et al., 1995) represents an important aspect of neuromuscular reaction time, during which there could be unrestrained development of forces of sufficient magnitude to damage ligamentous tissue in synovial joints (Huston & Wojtys 1996; Mercer et al. 1998; Shultz et al. 2001). Despite the apparent importance of EMD, its exact determinants are not fully understood.

EMD has been suggested to be due to several neuromechanical processes, specifically the time involved in: the propagation of the action potentials along the muscle fibre membrane; excitation contraction-coupling; and the stretching of the series elastic component (SEC) by the contractile component (Cavanagh & Komi, 1979). Using high speed ultrasound, Nordez et al. (2009) demonstrated that the time taken to stretch the SEC accounted for ~50% of EMD (Nordez et al., 2009). Any of these factors could contribute to inter-individual differences in EMD. Some preliminary research into the determinants of EMD has been undertaken, examining the morphological contributions to the variability in EMD and demonstrated EMD to be linked with inter-individual differences in muscle-tendon mechanics [e.g. tendon slack (Muraoka et al., 2004), tendon stiffness (Kubo et al., 2001)] and skeletal muscle composition [e.g. fiber type (Viitasalo & Komi, 1978)]. However, no study has investigated if neural factors may contribute to the variability in EMD. Voluntary EMD of the quadriceps has been shown to be 100% longer (16-25 ms) than electrically evoked EMD (Zhou et al., 1996; Tillin et al., 2010), which suggests that the ability to voluntarily activate skeletal muscle has a contribution on the variability in EMD. Additionally, previous research has considered the potential contribution of single independent variables in isolation, with no study attempting to comprehensively assess the determinants of EMD within the same study.

Assessment of involuntary RFD in response to evoked contractions can give insight into the intrinsic capacity of the MTU for explosive force production without the influence of

voluntary control and is therefore thought to reflect muscle morphology and tissue mechanics (Almeida et al., 1994; Harridge et al., 1996; Oda et al., 2007). Thus, the combined influence of morphological factors to voluntary EMD can be assessed by examining the intrinsic contractile properties in response to an evoked contraction. Assessment of normalised surface EMG amplitude during the early phase of an explosive voluntary contraction could provide information of the importance of the capability to activate skeletal muscle during the very early phase of force development and if examined alongside the intrinsic contractile properties can provide the necessary information concerning the factors which may contribute to the inter-individual EMD. The aim of the study was to determine the neural (agonist neuromuscular activation) and the intrinsic muscle contractile properties (evoked EMD, twitch and octet responses) contributions to EMD.

4.2 Methods

4.2.1 Participants

Twenty-four healthy male participants (mean ± SD: age 22 ± 3 yr; height, 1.79 ± 0.05 m; body mass, 71.8 ± 9.6 kg), with low to moderate activity status, and no history of lower body resistance or power training in the preceding 12 months, volunteered to participate in the study. Participants provided written informed consent prior to their involvement in the study, which complied with the Declaration of Helsinki and was approved by the Ethical Advisory Committee of Loughborough University.

4.2.2 Study Design

Participants attended the laboratory on two separate occasions, once for familiarisation and then for a main trial one week later. The two trials involved the same protocol and were completed at a consistent time of day for each patient. The main session involved the measurement of force and surface EMG during a series of voluntary (maximal and explosive) and electrically-evoked (twitch and octet) contractions of the knee extensors of the preferred limb.

4.2.3 Measurements

Participants were firmly secured into an isometric dynamometer (Bosjen-Moller et al., 2005; Parker et al., 1990) with waist and shoulder straps, with hip and knee angles fixed at 100° and 85° (full extension = 180°), respectively. An ankle strap was placed 2 cm proximal to the medial malleolus, in series with a calibrated U-shaped aluminium strain gauge (linear response up to 1000 N; Jones & Parker, 1989) that was positioned perpendicular to tibial alignment. The force signal was amplified (x 500) interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK) and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). Real-time biofeedback of the force response was provided on a computer monitor.

Surface EMG was recorded from the rectus femoris (RF), vastus lateralis (VL) and vastus medialis (VM) using a Delsys Bagnoli-4 EMG system (Input impedance, > 10^{15} Ω common mode rejection ratio, 93, Delsys, Boston, USA). Following preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol), double differential electrodes (1 cm inter-electrode distance, DE-3.1, Delsys) were attached over each muscle using adhesive interfaces. To standardise position between sessions and normalise across individuals, the electrodes were positioned in the centre of the muscle belly parallel to the presumed orientation of the muscle fibers at specific lengths along the thigh (from the lateral epicondyle of the femur to the greater trochanter: VM, 20%; VL, 40%; RF, 60%). Skin-electrode impedance was assessed to ensure a value < 5 MΩ for all participants. The reference electrode was placed on the patella of the same limb. EMG signals were amplified (x1000; differential amplifier, 20 – 450 Hz) and synchronised with force data by recording at 2000 Hz with the same analogue to digital converter and PC as the force signal. During off-line analysis the signals were band-pass filtered between 6 and 500 Hz using a 4th order zero-lag Butterworth filter prior to analysis.

4.2.4 Protocol

Participants performed a series of warm-up contractions at ~50 and ~75% of MVF followed by three MVCs each lasting 3-s and preceded by ≥30 s rest. Participants were instructed to contract as hard as possible in response to an auditory signal. Biofeedback was provided by displaying the force trace on a monitor with an on-screen cursor used to mark maximum

force. Participants were encouraged to exceed this target on subsequent attempts and strong verbal encouragement was provided during and between each maximal contraction. Knee extensor maximal voluntary force (MVF) was the greatest force achieved by the participant in any of the MVCs or explosive voluntary contractions (see below).

Participants then performed 10 explosive voluntary isometric contractions each separated by 20 s. Participants were instructed to extend their knee as 'fast' and as hard as possible in response to an auditory signal, with an emphasis on fast, for ~1 s from a relaxed state (Sahaly et al., 2001). Participants were instructed to avoid any countermovement or pre-tension. To determine if a countermovement had occurred, the resting force level was displayed on a sensitive scale. In order to provide biofeedback on their explosive performance, the slope of the force time curve (2 ms time constant) was displayed throughout the explosive contractions and the peak slope of their best attempt highlighted with an on-screen cursor. Finally, a visual marker on the screen depicted 80% of MVF during the contractions, and participants were expected to achieve this level of force during each explosive contraction. The three contractions with the highest peak slope and no discernible countermovement or pre-tension (change in force of < 0.5 N in the preceding 100 ms) were used for analysis, and all measurements were averaged across these three contractions. Signal onsets of all voluntary and evoked contractions were visually identified (Allison, 2003; Moretti et al., 2003; Pain & Hibbs 2007; Pulkovski et al., 2008) according to previous research from our laboratory (see Tillin et al., 2010; Buckthorpe et al., 2012). The root mean square (RMS) of the EMG signal for each muscle (RF, VM, VL) was calculated over the initial 50 ms from EMG onset – defined as the onset of the first muscle to be activated. EMG values from the agonist quadriceps (RF, VM, VL) were normalised to the maximal M-wave Area (M_{max} Area, see below) before being averaged across the three muscles to provide an overall value for the quadriceps.

The time difference between the onset of EMG and force was determined for each of the three superficial knee extensors and this defined EMD in this study. The greatest EMD irrespective of the muscle was taken as maximum EMD (EMD_{max}) during each contraction and averaged across the three contractions. Voluntary EMD_{max} was also expressed as a percentage of evoked EMD_{max} recorded during twitch contractions (detailed below: voluntary EMD_{max} / evoked EMD_{max}) to investigate the voluntary neural component of the delay (Tillin et al., 2010).

Using previously published methods (Buckthorpe et al., 2012), supramaximal electrical stimuli were delivered to the femoral nerve to elicit twitch and octet (8 pulses at 300 Hz) contractions. Square wave pulses (0.1 ms in duration) were delivered via a variable voltage stimulator (Model DS7AH, Digitimer, Ltd, Welwyn Garden City, UK). Incremental twitch contractions were elicited until a simultaneous plateau in the force and M-wave response was observed. Thereafter, the current was increased by 20% and three supra-maximal twitches were elicited (separated by 12 s). The average total M_{max} Area of these three supramaximal M-waves was determined for each muscle according to previous published methods and used for normalisation purposes (Buckthorpe et al., 2012). The twitch force response was analysed for peak force (PF), peak RFD (pRFD; 2 ms time constant), time to peak force (TPF), and half relaxation time (HRT) and was averaged across the three contractions. EMD$_{mean}$ and EMD$_{max}$ were calculated in the same manner as for voluntary explosive contractions.

For the evoked octet contractions the current was once again reduced and step wise increments were delivered 15 s apart until the same supramaximal current intensity was achieved (typically 4-5 increments were performed). Thereafter, three maximal octet contractions were elicited (separated by 12 s) and the average of the three taken for analysis. Analysis included measurement of octet PF, F_{50}, and pRFD (2 ms time constant).

4.2.5 Statistical Analysis

Descriptive statistics are presented as mean ± SD. Primary analysis involved performing Pearson's product moment bivariate correlations between voluntary EMD$_{max}$ and evoked EMD$_{max}$, neural (EMG normalised to M_{max} Area) and contractile variables (Twitch, PF, HRT, TPF, pRFD; Octet F_{50}, PF, pRFD). The relationship between evoked EMD$_{max}$ and contractile variables was assessed. Secondary analysis included splitting the sample into two separate groups (N = 12) based upon voluntary EMD$_{max}$ values, with the groups representing the half of the sample with the shortest and longest EMD$_{max}$ values. Independent samples t-test were then computed on all measured variables to identify significant differences between the groups, whilst effect sizes were reported to interpret the magnitude of these changes. Statistical analyses were performed using SPSS version 16 (SPSS inc., Chicago, IL, U.S.A.) and statistical significance was set at $P < 0.05$. Effect sizes (ES) were interpreted according to Cohen's d, where 0.2 is a small effect, 0.5, a moderate effect and > 0.8 a large effect.

4.3 Results

Agonist EMG amplitude was moderately related to voluntary EMD_{max} (r = 0.64, P < 0.01, Figure 4.1). Furthermore, this relationship was similar when considering only the volitional component of the delay (r = 0.61, P < 0.01). There was a significant albeit weak relationship between voluntary and evoked EMD_{max} (r = 0.42, P = 0.037, Figure 4.1). Voluntary EMD_{max} was not related to any of the evoked twitch performance measures (r ≤ 0.20, P ≥ 0.354) or octet PF or F_{50} (r ≤ 0.27, P ≥ 0.202). There was a tendency for voluntary EMD_{max} to be related to octet pRFD (r = 0.397, P = 0.061). Additionally, there was no relationship between evoked EMD_{max} and the twitch and octet contractile properties (twitch, r ≤ 0.217, P ≥ 0.308; Octet, r ≤ 0.40, P ≥ 0.654).

There was a significant difference between the top 12 and bottom 12 participants for voluntary EMD_{max} (15.6 ± 2.2 vs. 22.0 ± 4.5 ms, P < 0.001, Table 4.1). Furthermore, this group demonstrated a significantly lower volitional component of the delay (229 ± 32 vs. 311 ± 60%, P < 0.001, Table 4.1). Agonist EMG amplitude was the only variable which separated the groups, with evoked EMD_{max} and twitch and octet contractile properties being similar between groups (Table 4.1).
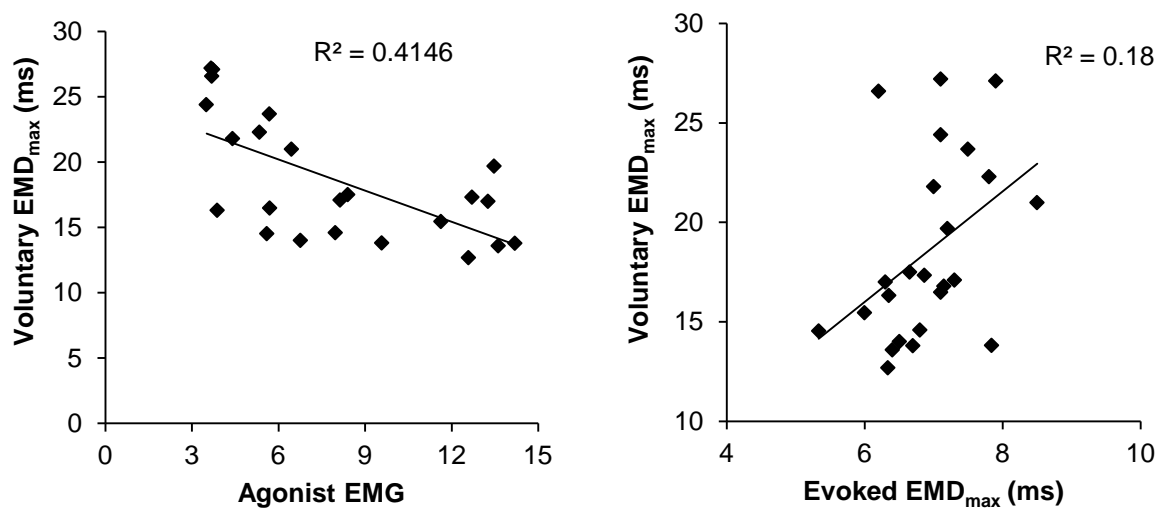
Figure 4.1 Bivariate relationship between voluntary EMD_{max} and A) agonist EMG amplitude and B) evoked EMD_{max}. N = 24.

Table 4.1 Electromechanical delay, neural (EMG normalised to M_{max} Area) and twitch and octet contractile properties for the mean sample, as well as the top and bottom halves of the sample (determined as median voluntary EMD_{max} values). Data are reported as mean ± SD. Mean group N = 24.

Variable	Mean Group	Top 12	Bottom 12	P-value	Effect Size
EMD_{max} (ms)					
Voluntary	18.5 ± 4.6	15.6 ± 2.2	22.0 ± 4.5	**< 0.001**	1.91
Evoked	6.9 ± 0.7	6.8 ± 0.5	7.1 ± 0.9	0.266	0.43
% Voluntary/ Evoked	268 ± 61	229 ± 32	311 ± 60	**< 0.001**	1.78
EMG (RMS/M_{max} Area)	8.0 ± 3.8	11.0 ± 2.7	4.7 ± 1.1	**< 0.001**	3.32
TWITCH					
pRFD (N.s^{-1})	2984 ± 659	3070 ± 534	3049 ± 639	0.659	0.04
PF (N)	110 ± 22	113 ± 20	112 ± 17	0.609	0.05
TPF (ms)	83 ± 10	82 ± 8	81 ± 11	0.641	0.11
HRT (ms)	74 ± 16	73 ± 18	74 ± 16	0.967	0.06
OCTET					
F_{50} (N)	208 ± 33	204 ± 20	217 ± 44	0.280	0.41
pRFD (N.s^{-1})	8203 ± 1417	8026 ± 1119	8693 ± 1619	0.183	0.49
PF (N)	339 ± 62	334 ± 54	349 ± 69	0.294	0.24

EMD_{max}, maximum electromechanical delay; EMG, electromyography; RMS, root mean square; M_{max} Area, cumulative are of evoked maximum compound action potential; N, newtons; pRFD, peak rate of force development; ; PF, peak force; TPT, time to peak force; HRT, half relaxation time; F_{50}, force at 50 ms after onset of force;

4.4 Discussion

The study found that the ability for neuromuscular activation (normalised surface EMG) explained 41% of the inter-individual variability in voluntary EMD_{max}. Electrically evoked EMD_{max} was only weakly related to voluntary EMD_{max} (18%). Furthermore, the intrinsic contractile properties evoked by twitch and octet stimulation did not significantly contribute to either voluntary or evoked EMD_{max}. The sample was split in to two groups and the best 12 participants had a 29% shorter voluntary EMD_{max} than the worst 12 participants. Agonist EMG amplitude was the only variable statistically different between the groups, reporting a very large effect (3.3), with no statistical difference between groups for evoked EMD_{max}, twitch or octet contractile properties.

Voluntary EMD_{max} was 168% longer than evoked EMD_{max} which is similar to other research (Tillin et al., 2010) and suggests a substantial neural contribution to voluntary EMD_{max}. Agonist EMG 0-50 ms explained 41% of the inter-individual variability in voluntary EMD_{max}. Furthermore, the best 12 participants for voluntary EMD_{max} (-29% shorter EMD, ES = 1.9) had significantly superior ability to activate the agonist muscles (+134%, ES = 3.3) over the initial 50 ms of contraction. The large differences in EMG (0-50 ms) between groups suggest that much larger changes in EMG may be necessary to elicit improved EMD performance. Recent cross-sectional studies have confirmed a strong relationship (r^2 = 0.75-0.83) between agonist muscle activation, assessed with normalised EMG and the torque-time integral in the first 40 ms of an explosive contraction, i.e. 40 ms from torque onset (de Ruiter et al. 2004, 2006, 2007). Further work has supported agonist EMG during the initial 50 ms of contraction following force onset as the primary determinants (r^2 = 0.51) of relative explosive force capability after 50 ms. Therefore, the current study provides further compelling evidence as to the importance of neuromuscular activation during the early phase of contraction to explosive neuromuscular performance.

The relationship between neuromuscular activation and the voluntary component of the delay (ratio voluntary/evoked, was not higher than for agonist EMG and voluntary EMD_{max} (r^2 = 0.38 vs. 0.41). This suggests that neural factors other than that of the level of neuromuscular activation contribute to this volitional component. In theory, supra-maximal stimulation of a motor nerve results in the synchronised activation of the whole motor unit pool, and may result in a reversal of the recruitment order of motor units whereby larger, fast contracting motor units are recruited first (Bickel et al. 2011). However, motor unit recruitment during voluntary explosive contractions appears to follow the size principle (Henneman et al., 1965; Van Cutsem et al., 1998). Therefore, even with maximal voluntary activation at onset, it is unlikely that a participant would be able to attain their evoked EMD_{max} values.

An important finding of chapter three of this thesis was that window length had a substantial influence on EMG amplitude variability. The curve between EMG variability and window length almost perfectly fit that of a declining power function, in which EMG amplitude became increasingly more unreliable as window length decreased to short values. Analysis of the research reveals that voluntary EMD_{max} has superior reliability to agonist EMG amplitude for explosive isometric contractions (Tillin et al., 2010; Buckthorpe et al., 2012). Thus, it was decided that rather than matching the EMG window length to that of EMD time (~20 ms), that a more reliable EMG time window would be used. A further consideration is validity in

which a time window needs to reflect the ability to rapidly activate agonist EMG at onset. Consequently, a window length of 50 ms was chosen, as this is more reliable than for 25 ms (Buckthorpe et al., 2012), but reflects this capability of the neuromuscular system for agonist EMG during the very early phase of contraction. Therefore, although it can be argued that the majority of agonist activation assessed within the current study occurs after EMD, and therefore, the exact correlations may not fully reflect the role of agonist EMG as a determinant of EMD, it is thought that the current method provides a more reliable reflection of the importance of the ability to activate the agonist muscles during the early phase of contraction on EMD.

In the present study there was a weak relationship between evoked EMD_{max} and voluntary EMD_{max} ($r^2 = 0.18$), which suggests a significant (P = 0.04) albeit small morphological and mechanical contribution. Other studies did not find significant relationships for voluntary and evoked EMD (Zhou et al., 1995; Minshull et al., 2007). Despite this, there was no relationship between either voluntary or evoked EMD_{max} and the intrinsic twitch or octet contractile properties. There was a strong tendency for octet pRFD to be related ($r^2 = 0.16$, P = 0.06) to voluntary EMD_{max}, which could suggest a minor role of contractile RFD to influence voluntary EMD_{max}, and might contribute the majority of the relationship between evoked and voluntary EMD_{max}. Octet RFD is thought to reflect the muscles maximal contractile RFD (de Ruiter et al., 1999, 2004). As about half of EMD constitutes the time to stretch the SEC (Nordez et al., 2009), a higher RFD would be expected to decrease the time required to stretch the SEC and benefit EMD performance. Surprisingly, the split group comparisons revealed, that there was no difference between groups for evoked EMD_{max}, twitch or octet contractile properties. The low values of evoked EMD_{max} and limited relationship of these measures to voluntary EMD_{max} may, in part, reflect the knee angle utilised during the present investigation. Tendon slack has shown to influence EMD values (Muraoka et al., 2004), but only at shorter muscle tendon lengths. Thus, the long muscle tendon length used within the present study may have shortened the time to stretch the SEC, which may have decreased the contribution peripheral factors (tendon slack) on voluntary and/ or evoked EMD.

In conclusion, the level of neuromuscular activation during the early phase of the contraction was a moderate determinant in voluntary EMD_{max} (41%). There was a significant albeit small relationship between evoked and voluntary EMD_{max} (18%). However, the top 50% of participants for voluntary EMD_{max} reported superior agonist EMG amplitude only (+ 134%),

with no differences between groups for evoked EMD_{max} or twitch and octet contractile properties. Consequently, it appears voluntary EMD_{max} is strongly related to the ability to activate agonist muscles during the very early phase of contraction (0-50 ms).

CHAPTER 5

Bilateral Deficit in Explosive Force Production is not caused by Changes in Agonist Neural Drive

5.1 Introduction

Bilateral deficit (BLD) has been used to describe the phenomenon of a reduction in performance during synchronous bilateral (BL) movements when compared to the sum of identical unilateral (UL) movements. A large body of research concerning BLD has been conducted using isometric and isokinetic tests of maximal voluntary force (MVF) production (for a review see Jakobi & Chilibeck, 2001). BLD and has been reported with deficits of up to ~25% (Koh et al., 1993; Van Dieen et al., 2003; Magnus & Farthing, 2008) and therefore represents a potentially influential factor in the expression of BL muscle strength. However, as consistently highlighted within this thesis, explosive strength is often considered functionally more important than MVF during explosive movements, such as sprinting and jumping or restabilising the body following a loss of balance (Aagaard et al., 2002a; de Ruiter et al., 2004; Tillin et al., 2010, 2013b). There is though, a paucity of research examining BLD in explosive strength with equivocal findings and limited mechanistic evidence. A BLD in peak rate of force development (RFD) has been reported to range between 0–24% (Howard & Enoka, 1991; Koh et al., 1993; Van Dieen et al., 2003; Magnus & Farthing, 2008), with some studies indicating a greater BLD in RFD than MVF (Sahaly et al., 2001; Van Dieen et al., 2003), whereas others have not (Koh et al., 1993; Sahaly et al., 2001).

Despite a large body of research examining BLD, the exact mechanisms explaining the phenomenon are unresolved. The primary explanation for BLD during maximum isometric and isokinetic contractions is reduced neural drive to the agonist muscles. However, the evidence is equivocal, with several studies documenting parallel reductions in force and agonist activation during bilateral tasks (Oda & Moritani, 1995; Van Dieen et al., 2003; Post et al., 2007), whereas others have not (Schantz et al., 1989; Howard & Enoka, 1991; Herbert & Gandevia, 1996; Magnus & Farthing, 2008). In the context of explosive strength, agonist activation has been found to be an important determinant of explosive force production (de Ruiter et al., 2004; Del Balso & Cafarelli, 2007; Tillin et al., 2010). Therefore, explosive force may be more susceptible to any reduction in agonist neural drive during explosive contractions than at MVF, and thus, a more pronounced BLD for explosive than MVF could be expected. However, during the explosive phase of BL vs. UL contractions only one study has assessed agonist, and none have documented antagonist, neuromuscular activation. Van

Dieen et al. (2001) reported no change in agonist activation, despite a 13% decline in peak RFD.

The equivocal evidence for agonist activation contributing to BLD might relate to the sensitivity of EMG measures, which have been questioned for their ability to detect small differences (Jakobi & Cafarelli, 1998). The absolute EMG amplitude is influenced by a multitude of intrinsic and extrinsic factors that are unrelated to the level of muscle activation (de Luca, 1997). Normalisation of the surface EMG amplitude to a maximal compound muscle action potential (M_{max}) is considered a more sensitive measurement tool, but it has not previously been used to investigate the mechanistic basis of any BLD. The assessment of evoked explosive contractions can give insight into the capacity of the muscle-tendon unit (MTU) for explosive force production without the influence of voluntary commands. Identification of BLD in electrically evoked force would indicate BLD mechanism(s) exclusive of voluntary neural drive to the agonist muscles. However, the possibility of a BLD in evoked force production has not been investigated. Furthermore the comparison of volitional to evoked explosive force may also provide an alternative measure of the volitional neural efficacy.

Other potential mechanisms not previously considered that may contribute to a BLD in explosive strength include methodological artefacts associated with the measurement of BLD. For example, a BLD in explosive voluntary force could be due to a lack of synchronisation of agonist activation and force onset from the two limbs. Any offset or delay in the activation and force development from the second limb could compromise combined BL performance and contribute to BLD even if performance of each individual limb in this BL situation were equivalent to UL performance. An additional potential contributory factor arises from the fact that investigators typically utilise a small number of UL and BL contractions, and take the best UL and BL contractions for analysis and comparison (e.g. Jakobi & Cafarelli, 1998; Sahaly et al., 2001; Van Dieen et al., 2003). However, this comparison may involve a statistical bias in favour of UL performance. BL performance relies on the simultaneous performance of two limbs, and statistically it is unlikely that both limbs will produce their highest UL performance during the same BL contraction. This simple measurement artefact could contribute to any apparent BLD irrespective of any physiological effects. Furthermore, as explosive force/RFD is less reliable than MVF (Buckthorpe et al., 2012), this measurement artefact might exert a greater bias on the BLD during explosive contractions. Essentially, whilst combined BL performance (i.e. the best effort of both legs when measured together) is

clearly the actual and criterion measure of BL capability, due to possible measurement artefacts it may under represent the best effort of either leg in the BL situation. Comparison of UL performance to both combined BL performance, and performance of each limb during BL contractions, may highlight the influence measurement artefacts.

The aim of the study was to assess whether a BLD exists in voluntary and evoked explosive force production of the knee extensors, and document the contribution of agonist and antagonist neuromuscular activation, as well as measurement issues to any BLD in voluntary explosive force production. It was hypothesised that there would be a more substantial BLD for explosive force/RFD than MVF. This could be due to a more pronounced reduction in agonist neuromuscular activation and a stronger influence of methodological factors during explosive than maximum voluntary contractions.

5.2. Methods

5.2.1 Participants

Twelve healthy asymptomatic male participants completed the study (mean ± sd: age, 24 ± 4 yr; height, 1.69 ± 0.04 m; body mass, 77.3 ± 6.9 kg). Data from previously published research (Van Dieen et al., 2003) was used to estimate the effect size for estimated BLD of explosive force/RFD. Cautiously, we aimed to detect a standardized effect size of 1.1. This standardized effect size, a statistical power of 80% ($1 – \beta = 0.80$) and $\alpha = 0.05$ were used to determine the necessary sample size of 11 participants. The participants were recreationally active (up to three activity sessions per week), but had not been involved in any systematic physical training during the preceding 12 months. All participants provided written informed consent prior to their involvement in the study, which complied with the Declaration of Helsinki and was approved by the Ethical Advisory Committee of Loughborough University.

5.2.2 Overview

Participants attended the laboratory on two separate occasions, once for familiarisation and then for a main trial one week later. The two trials involved the same protocol and were completed at a consistent time of day. The main session involved the measurement of force and surface EMG during a series of voluntary (maximal and explosive) and electrically-

evoked (twitch and octet) contractions of the knee extensors performed during either UL or BL contractions. In addition UL knee flexor maximum voluntary contractions (MVCs) with each leg were also performed for normalisation of antagonist EMG. To control for the influence of possible order effects, the order of voluntary contractions, involved first UL contraction(s) (either dominant or non-dominant leg, contraction order was randomly assigned), then BL contraction(s), and finally UL contraction(s) with the remaining limb (i.e. UL-BL-UL). Evoked measures began with the same limb that commenced the voluntary contractions followed by UL contractions of the remaining limb, and finally BL contractions (UL-UL-BL). Electrically evoked contractions can cause discomfort, and are not tolerated well by all participants. Therefore, it was decided to elicit single twitch and octet (8 pulses at 300 Hz) contractions unilaterally on both legs first before BL contractions to ensure as many participants completed the evoked measures as possible. In order to assess the BLD of voluntary and evoked contractions, performance during UL contractions were averaged and compared to the genuine BL performance, which involved the simultaneous averaged performance of both limbs obtained from a mutual onset during the same BL contractions (BL_{BL}). Furthermore, the contribution of methodological artefacts (e.g. synchronisation of force onset) was also assessed. This involved comparing UL contractions with single limb performance measured during BL contractions. In practice this was facilitated by the discrete recording (i.e. two independent force transducers) and analysis (i.e. separate force onset) of each limb during BL efforts before averaging across both limbs (BL_{UL}). This allowed for assessment of UL vs. BL_{UL} without the potentially confounding influence of methodological artefacts.

5.2.3 Measurement Trials

5.2.3.1 Measurements

Participants were firmly secured in a custom built strength testing chair with straps across the pelvis and shoulders to minimise extraneous movement. The hip and knee angles were fixed at 100 and 120° (full extension = 180°), respectively. An ankle strap was placed 2 cm proximal to the medial malleolus of each limb in series with two separate S-Beam tension/compression load cells (one for each limb, linear response up to 1500 N, Force Logic UK, Berkshire, UK) positioned perpendicular to tibial movement. The force signal was amplified (x500) and interfaced with an analogue to digital converter (CED micro 1401, CED,

Cambridge, UK) and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). Real-time biofeedback of the force response was provided on a computer monitor. During off-line analysis the force signals were notch filtered at 50 Hz (to remove mains harmonics) and low pass filtered at 500 Hz using a fourth order zero-lag Butterworth digital filter.

The femoral nerve of each leg was electrically stimulated (via two constant current, variable voltage stimulators; DS7AH, Digitimer Ltd., UK) with square wave pulses (0.2 ms in duration) to elicit i) single twitch contractions and ii) octet contractions (8 pulses at 300 Hz) to determine the muscle's maximal capacity for RFD. An anode (carbon rubber electrode, 7 x 10 cm; Electro-Medical Supplies, Greenham, UK) was taped to the skin over the greater trochanter of each limb. A cathode was taped to the skin over the femoral nerve in the femoral triangle of each leg. Both cathodes were identical custom-adapted stimulation probes 1 cm in diameter (Electro-Medical Supplies, Wantage, UK) which protruded 2 cm perpendicular from the centre of a plastic base (4 x 5 cm). The precise location of the each cathode was determined as the position which elicited the greatest twitch response for a particular submaximal current during UL contractions. During BL evoked contractions both stimulators were triggered simultaneously via the Spike 2 software.

Surface EMG was recorded from the superficial quadriceps [rectus femoris (RF), vastus lateralis (VL) and vastus medialis (VM)] and a knee flexor [bicep femoris (BF)] of both legs using two Delsys Bagnoli-4 EMG systems (Delsys, Boston, USA). Following preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol), double differential electrodes (1 cm inter-electrode distance, DE-3.1, Delsys) were attached over each muscle using adhesive interfaces. To normalise the placement across individuals, the electrodes were positioned in the centre of the muscle belly parallel to the presumed orientation of the muscle fibers at specific lengths along the thigh (from the lateral epicondyle of the femur to the greater trochanter: VM, 25%; VL, 50%; RF, 60%; BF, 50%). The reference electrode was placed on the patella of the same limb. EMG signals were amplified (x1000; differential amplifier, 20 – 450 Hz) and synchronised with force data by recording at 2000 Hz with the same analogue to digital converter, PC and software (Spike 2). During off-line analysis the EMG signals were band-pass filtered between 6 and 500 Hz using a 4th order zero-lag Butterworth digital filter.

5.2.3.2 Protocol

Once the participants were firmly secured in the testing chair they performed a warm-up, which consisted of two UL (with each limb) and BL contractions of the knee extensors at 50 and 75% presumed MVF. Participants then performed eight successful explosive voluntary contractions (separated by 20 s rest) of each contraction type (UL-BL-UL, with 2 min between each series, see Figure 5.1). For each contraction participants were instructed to extend their knee(s) as 'fast' and as hard as possible for ~1 s from a relaxed state (Sahaly et al., 2001). Contractions that had any pre-tension or countermovement were discarded and another attempt was made. To determine if a countermovement or pre-tension had occurred, the resting force level was displayed on a sensitive scale. The slope of the force time curve (10 ms time constant) was displayed throughout testing and the peak slope was used to provide visual performance feedback to participants after each contraction. Furthermore, participants were required to exceed 80% MVF during these explosive contractions, specific to that leg(s) which was depicted with a horizontal cursor on the screen. For the BL explosive contractions identical criteria and feedback were used based on the averaged force signal from both load cells.
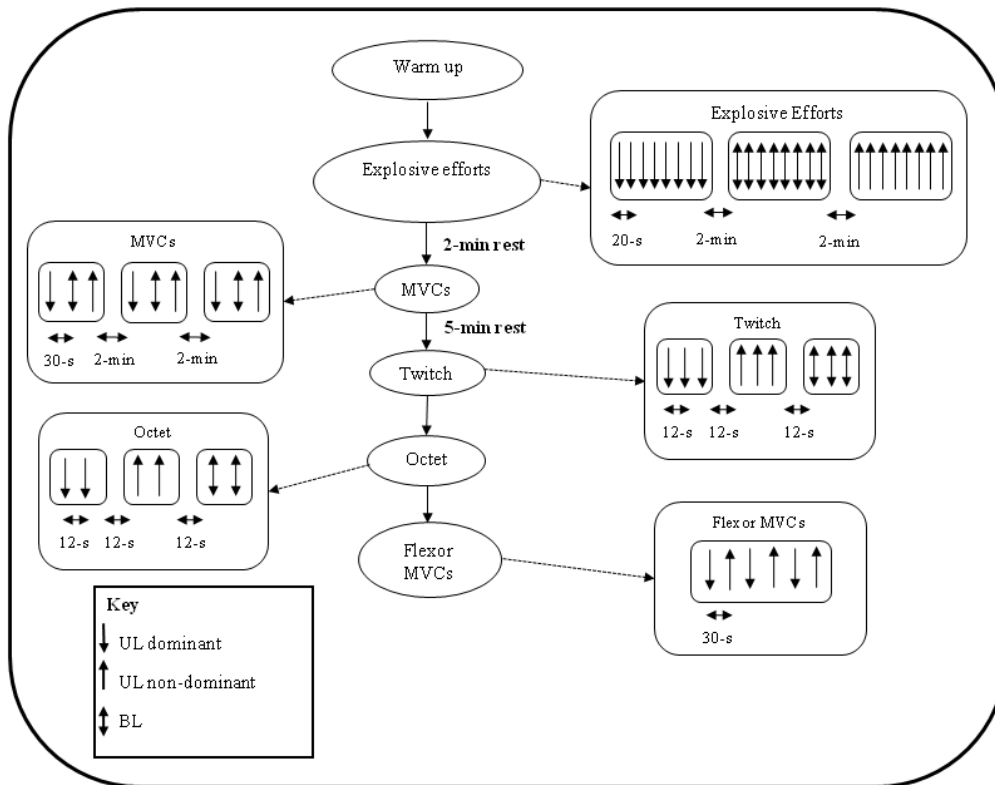
Figure 5.1 Schematic diagram of the protocol.

The three contractions in each condition with the highest peak slope and no discernible countermovement or pre-tension (change in force of <0.5 N in the preceding 100 ms) were used for analysis. We have previously demonstrated that using the best three contractions from a series of explosive voluntary contractions following sufficient familiarisation provides reliable group explosive force and EMG measures (see Chapter 3, Buckthorpe et al., 2012). Force and EMG measurements were taken at specific time points/periods and all measurements were averaged across these three contractions. Signal onsets of all voluntary and evoked contractions were visually identified (Allison, 2003; Moretti et al., 2003; Pain & Hibbs, 2007; Pulkovski et al., 2008) according to previous methods from our laboratory (see Tillin et al. 2010; Buckthorpe et al., 2012). Force was measured at 50, 100, and 150 ms (defined as F_{50}, F_{100}, F_{150}), from the onset of contraction. RFD was measured over three consecutive 50 ms time periods from the onset of force (RFD_{0-50}, RFD_{50-100}, $RFD_{100-150}$). For evaluating purely BL performance (i.e. the average combined ability of the two legs, BL_{BL}) force onset was defined as the deflection of the averaged force signal from baseline. However for assessing UL performance during BL efforts (BL_{UL}) force onsets were specific to that leg.

EMG signal amplitude was quantified as the RMS measured in consecutive windows 0-50, 50-100, and 100-150 ms from the onset of EMG activity in the first agonist muscle to be activated within that limb(s). EMG from each agonist muscle was normalised to the peak-to-peak amplitude of a maximum compound action potential (M_{max} P-P) of that muscle during UL contractions (see below) and averaged across the three superficial quadriceps muscles to give a mean value for the quadriceps. EMG from the BF was normalised to EMG at knee flexor MVF (see below) of that muscle (antagonist EMG). Although, the study was a within session design, the EMG was normalised to reduce the between participant variation (Buckthorpe et al., 2012) which would be expected to increase the effect size and power of statistical comparisons between the conditions. EMG onsets were identified from the first agonist muscle to be activated specific to each leg during UL and BL_{UL} conditions and the first muscle to be activated irrespective of the leg during the BL_{BL} condition. Additionally, the difference between the onsets of force of the two limbs in the BL contractions was identified. The time between the first agonist muscle to be activated and onset of force was determined as the maximum electromechanical delay (EMD_{max}).

Following two minutes rest, participants performed three sets of a single MVC of each type in the specified order (UL-BL-UL) with $\geq$ 30 s between MVCs and 2 min between sets. For each MVC they were instructed to push as hard as possible for 3 s with biofeedback and verbal encouragement provided during and between each maximal contraction. Knee extensor maximal voluntary force (MVF) was the greatest instantaneous force achieved by the participant in any of the MVCs specific to each condition. The root mean square (RMS) of the EMG signal for each muscle (RF, VM, VL and BF) was calculated over a 500 ms epoch surrounding MVF (250 ms either side). Each individual agonist muscle EMG was normalised M_{max} P-P (see below) before averaging across the three muscles to provide a mean value for the quadriceps (agonist EMG). BF EMG was expressed as a percentage of BF EMG at knee flexor MVF (see below).

Five minutes separated the MVCs and evoked measurements. Evoked measures began with the same limb that commenced the voluntary contractions followed by UL contractions of the remaining limb, and finally BL contractions (UL-UL-BL). Twitch contractions were elicited at incremental current intensities until a simultaneous plateau in the force and M-wave response was observed. Thereafter, the current was increased by 20% and three supra-maximal twitches were elicited (separated by 12 s) for each limb during UL contractions. For BL contractions, the current was reduced, and incremental (25, 50, 75% of the supramaximal

current used during UL contractions specific to that limb) evoked contractions were elicited, and three supramaximal BL twitch contractions were recorded. Two participants withdrew from the twitch measurements, therefore twitch responses are reported for N = 10. The mean M_{max} P-P of these three supramaximal M-waves was determined for each muscle and used for normalisation purposes. The twitch force response was assessed at 50 ms after force onset (F_{50}), peak force (PF), and pRFD (10 ms time constant) and averaged across the three contractions for each performance measure.

For the evoked octet contractions the current was once again reduced and step wise increments were delivered 15 s apart until the same supramaximal current intensity was achieved (typically 4-5 increments were performed). Two maximal evoked octet contractions were then elicited. The order of contractions was the same as during evoked twitch contractions (i.e. UL-UL-BL). Three participants withdrew from the octet measurements, therefore octet responses are reported for N = 9. During analysis, the average of the two octet contractions for each contraction type was taken. Analysis included measurement of force at 50 ms (F_{50}), PF and pRFD. As an additional measure of overall neural efficacy, voluntary F_{50} for the three different measurements was reported as a percentage of the equivalent octet F_{50} to assess the participant's voluntary activation capacity over the initial 50 ms of the contraction (Hannah et al., 2012; Tillin et al., 2012a).

5.2.4 Data Analysis

Data are reported as mean ± standard deviation (SD). One-way ANOVA was used to identify significant differences between voluntary performance measures across the three conditions. In the event of significant differences, paired t-tests were performed. For indices measured at two or more time points (EMG, force, RFD during explosive contractions) the effect of test condition (UL vs. BL_{BL} vs. BL_{UL}) was analysed using a two-way repeated measures ANOVA (condition [3] × time [3]). Pairwise comparisons with Bonferroni correction were performed to locate the significant difference between test conditions at specific time points. Effect sizes (ES) were performed to determine the magnitude of the sigfnificant differences and interpreted according to Cohen's d, where 0.2 is a small effect, 0.5, a moderate effect and > 0.8 a large effect. BLD was defined as a difference between the BL_{BL} and UL conditions. Prior to performing the statistical analysis, confirmation of data normality was performed using Shapiro-Wilk test of normality. Statistical analysis was performed using SPSS version

19 and statistical significance was set at P < 0.05. Effect sizes (ES) were interpreted according to Cohen's d, where 0.2 is a small effect, 0.5, a moderate effect and > 0.8 a large effect.

5.3. Results

5.3.1 Voluntary Contractions

There was no difference in MVF (ANOVA, P = 0.551, Table 5.1) or agonist (ANOVA, P = 0.269, Table 5.1) or antagonist (ANOVA, P = 0.987, Table 5.1) EMG at MVF between the three measurement conditions.

There was a significant difference between conditions for force (ANOVA, P = 0.022) and RFD (ANOVA, P = 0.022) during the explosive voluntary contractions. Pairwise comparisons revealed F_{50} was similar for all three conditions (P > 0.90, Table 5.2). However, there was a BLD in F_{100} with BL_{BL} values 11.2% lower than UL (P = 0.007, ES = 1.19), and with a tendency for BL_{UL} to also be lower than UL (P = 0.067, ES = 0.90). There was a tendency for a BLD in F_{150} with BL_{BL} lower than UL (P = 0.059, ES = 0.83), but there was no difference in F_{150} between BL_{UL} and UL (P = 0.116, ES = 0.55, Figure 5.2). RFD_{50-100} was 14.9% lower for BL_{BL} (P = 0.004, ES = 1.30) and 12.5% lower for BL_{UL} (P = 0.022, ES = 1.19) compared to UL (Figure 5.2), with no differences in RFD_{0-50} or $RFD_{100-150}$ between conditions (P > 0.90). Additionally, there were no significant differences in RFD between BL_{UL} and BL_{BL} (All, P > 0.90).

Table 5.1 Force and EMG during maximum voluntary contractions performed unilaterally (UL) and bilaterally (BL_{BL}, averaged simultaneous performance of both limbs; BL_{UL}, single leg performance during BL contractions). Data are reported as mean ± SD (N = 12).

	UL	BL_{BL}	BL_{UL}
MVF (N)	736 ± 83	739 ± 92	744 ± 89
Agonist EMG (%M_{max})	8.2 ± 2.0	8.6 ± 2.5	8.3 ± 2.3
Antagonist EMG (%EMG_{max})	8.4 ± 6.8	8.5 ± 5.1	8.9 ± 6.4

MVF, Maximum voluntary force; N, Newton; M_{max}, peak to peak amplitude of maximum compound action potential; EMG_{max}, maximum RMS EMG obtained during knee flexor maximum voluntary contraction

Table 5.2 Force during explosive voluntary contractions during unilateral (UL) and bilateral contractions (BL$_{BL}$, averaged simultaneous performance of both limbs; BL$_{UL}$, single leg performance during BL contractions). Data are reported as mean ± SD (N = 12).

Force (N)	UL	BL$_{BL}$	BL$_{UL}$
50 ms	168 ± 45	159 ± 46	165 ± 57
100 ms	442 ± 42	392 ± 37[**]	404 ± 56
150 ms	580 ± 63	528 ± 51	543 ± 72

*N, Newton; ** denotes significant difference compared to UL (P < 0.01).*
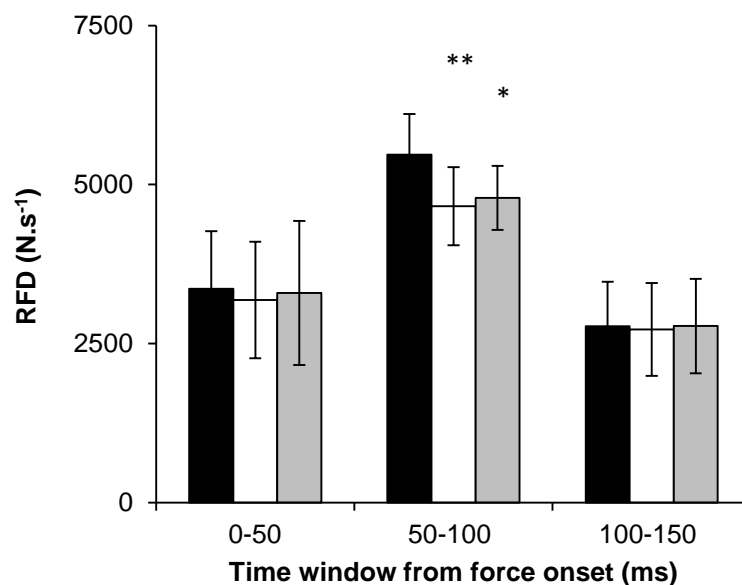
Figure 5.2 Rate of force development (RFD) during explosive unilateral (UL, black bars) and bilateral contractions (BL$_{BL}$, white bars, averaged simultaneous performance of both limbs; BL$_{UL}$, grey bars, single leg performance during BL contractions) explosive contractions of the knee extensors. Data are reported as mean ± SD (N = 12). A significant difference between conditions is denoted by * P < 0.05 vs. UL, ** P < 0.01 vs. UL.

There were no differences in agonist (two-way ANOVA, P = 0.233, Figure 5.3A) or antagonist (two-way ANOVA, P = 0.873, Figure 5.3B) EMG amplitude between the three measurement conditions during the explosive contractions. Additionally, neural efficacy, the percentage of evoked octet F$_{50}$ achieved voluntarily was also similar for the three measurement conditions (UL, 55.5 ± 17.3; BL$_{BL}$, 58.4 ± 18.7; BL$_{UL}$, 61.3 ± 20.6%, ANOVA, P = 0.212).
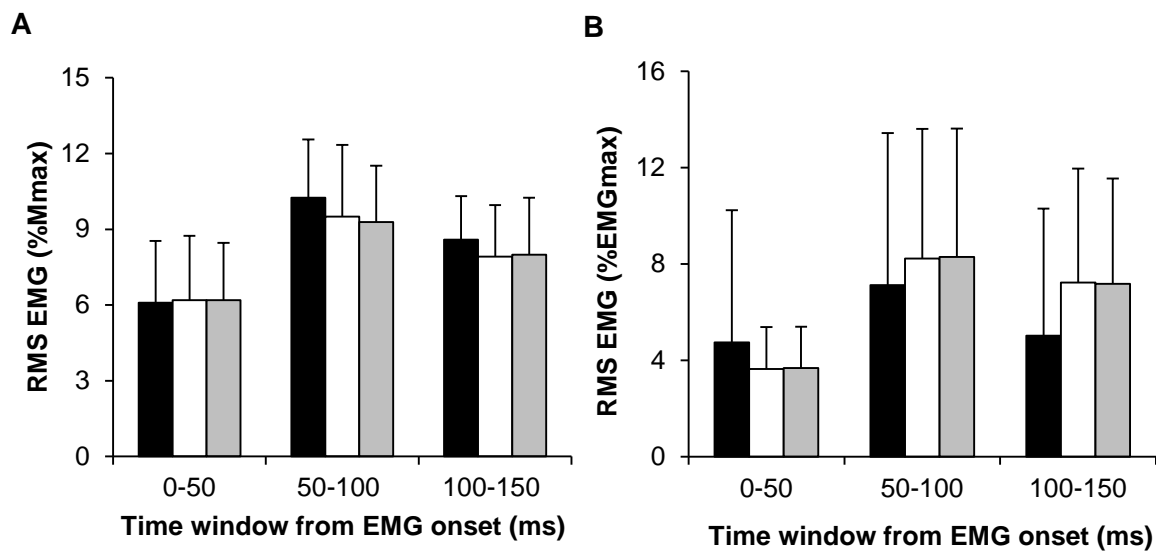
Figure 5.3 A, Agonist EMG normalised to M_{max} and B, Antagonist EMG normalised to EMG at knee flexor MVF during unilateral (UL, black bars) and bilateral contractions (BL_{BL}, white bars, averaged simultaneous performance of both limbs; BL_{UL}, grey bars, single leg performance during BL contractions) explosive voluntary contractions. Data are reported as mean (SD) (N = 12).

The time difference in force onset between the two limbs during the BL explosive contractions was 3.2 ± 1.7 ms. There was no difference in EMD_{max} between UL and BL contractions (UL, 18.5 ± 3.6 vs. BL_{UL}, 18.4 ± 4.1 ms, Paired t-test, P = 0.942).

5.3.2 Electrically-evoked Contractions

Twitch F_{50} and PF were lower for both BL_{UL} and BL_{BL} compared to UL (7.8-9.1%, $P \leq 0.002$, ES = 0.38-0.44), with no difference for twitch pRFD between measurement conditions (Table 5.3). Additionally, there was no difference in M_{max} P-P between measurement conditions (UL, 3.0 ± 1.1 vs. BL_{BL}, 2.8 ± 1.0 mV, Paired t-test, P = 0.138). Octet F_{50} was lower for both BL_{UL} (6.0%, ES = 0.58) and BL_{BL} (6.3%, ES = 0.61) than UL (Both, P < 0.001, Table 5.3), but there were no differences for octet PF or pRFD (Table 5.3). There were also no differences between BL_{UL} and BL_{BL} for either twitch or octet measure ($P \geq 0.187$).

Table 5.3 Force parameters during evoked twitch and octet contractions during unilateral (UL) and contractions (BL$_{BL}$, white bars, averaged simultaneous performance of both limbs; BL$_{UL}$, grey bars, single leg performance during BL contractions). Data are reported as mean ± SD (Octet, N = 9; Twitch, N=10).

	Condition:			P-value
	UL	BL$_{BL}$	BL$_{UL}$	
Octet				
F$_{50}$ (N)	300 ± 30	281 ± 32	282 ± 32	< 0.001
PF (N)	480 ± 52	477 ± 55	480 ± 54	0.585
pRFD (N.s^{-1})	13511 ± 2785	13278 ± 2433	14164 ± 2892	0.243
Twitch				
F$_{50}$ (N)	115 ± 24	106 ± 24	105 ± 23	< 0.001
PF (N)	134 ± 27	122 ± 26	123 ± 24	< 0.001
pRFD (N.s^{-1})	3920 ± 1210	3709 ± 1227	3754 ± 1153	0.290

UL, unilateral; BL, bilateral; F, force; N, newton; PF, peak force; pRFD, peak rate of force development; F$_{50}$, force at 50 ms after force onset. P-value, One-way analysis of variance significance value.

5.4 Discussion

This study investigated BLD in voluntary and electrically-evoked explosive contractions of the knee extensors and considered the contribution of agonist neuromuscular activation and measurement issues to any BLD. We observed a BLD in voluntary explosive force/RFD but not MVF. The BLD in explosive force occurred at 100 ms only and reflected a BLD specific to RFD$_{50-100}$. BLD measurement issues made only a minor contribution to the observed BLD and thus these results support an underlying physiological mechanism explaining BLD. However, the fact that we observed a BLD in evoked force production and no change in EMG during explosive voluntary efforts suggests the BLD was not solely attributable to reduced agonist or antagonist neural drive.

The finding of no BLD in MVF is consistent with numerous reports (e.g. Schantz et al., 1989; Hakkinen et al., 1996; Jakobi & Cafarelli, 1998), but in contrast to an equal number that have shown a BLD in knee extensor MVF (e.g. Howard & Enoka, 1991; Van Dieen et al., 2003; Kuruganti et al., 2010). As there was no BLD in MVF, it is unsurprising that there was no difference in agonist or antagonist activation, evoked peak force measures with high force values, or influence of methodological factors. This is in accordance with previous findings of no BLD or mechanistic differences between BL and UL MVCs (Jakobi & Cafarelli, 1998)

Despite no BLD for MVF, a BLD was observed in explosive force of 11.2% during these single joint voluntary contractions. The BLD was specific to F_{100}, but there was a tendency for a BLD in F_{150}. Furthermore, there was a 14.9% BLD for RFD_{50-100}, with no BLD for RFD_{0-50} or $RFD_{100-150}$. This is the first study to investigate the possibility of a BLD in explosive strength by analysing force/RFD throughout the rising force-time curve. Previously, only pRFD had been assessed in this context, and reported to range from 0-20% (Koh et al., 1993; Jakobi & Cafarelli, 1998; Van Dieen et al., 2003). The mechanisms for the observed BLD in explosive force could have been due to measurement issues in the comparison of UL and BL performance, neuromuscular activation of agonist, antagonist muscles that were assessed in this study, or even activation of stabiliser muscles that we did not assess.

The assessment of single limb performance during BL contractions allowed for the delineation of measurement artefacts that may have contributed to any observed BLD. Although, the BL_{UL} measure reported only a tendency for a difference to UL for F_{100}, there was a difference for RFD_{50-100}, confirming a BLD due to a physiological effect exclusive of measurement issues. There were also no differences in explosive or maximal force/RFD between the two BL measures, indicating measurement artefacts played only a minor role in the observed BLD. Surprisingly, the onset of force discrepancy between the two limbs during BL contractions was relatively small (3.2 ms), which suggests that neuromuscular system is capable of near simultaneous activation of the knee extensor muscles of both legs during BL actions.

The current study found no differences in agonist EMG between UL and BL explosive contractions. This is despite the widely suggested mechanism for BLD being a reduction in neural drive to the agonist muscles. Our findings support previous research demonstrating a BLD in RFD in the absence of a change in agonist EMG (Van Dieen et al., 2003). It is important to note that the sensitivity of EMG for assessing BLD has been questioned (Jakobi & Cafarelli, 1998). However, in the present study we normalised the EMG amplitude to M_{max}, which would be expected to increase the effect size and power of statistical comparisons between the conditions. Additionally, we averaged across three quadricep muscles and across the best three contractions during the explosive efforts. These methods would be expected to improve the reliability and sensitivity of the EMG measurements. Furthermore, we also measured neural efficacy, which assesses agonist neuromuscular activation during the initial phase of the contractions (50 ms), and provided further evidence that agonist activation was not different during the early phase of UL and BL explosive contractions. Our study suggests

that the observed BLD in RFD was not attributable to agonist activation, and indicates a role for an alternative mechanism.

Agonist and antagonist activation contribute simultaneously to net joint torque and thus the level of co-activation could account for any BLD. This is the first study to assess if antagonist activation influenced the BLD during explosive force production, and found that the observed BLD in RFD was not attributable to antagonist activation. A possible remaining explanation concerns stabiliser activation.

BL evoked contractions were utilised within the present study to help establish if the BLD was influenced by a physiological mechanism(s) exclusive of voluntary neural drive to the agonist muscles. Interestingly, there was a BLD in evoked force production, which occurred in both twitch and octet F_{50} (8.7 and 6.3%, respectively), and twitch PF (9.0%) and was of a similar magnitude to the observed declines in explosive voluntary force/RFD (8.6 – 14.9%). This is the first study to investigate a potential BLD in evoked force production and provides further support that the BLD in voluntary explosive force production was due to mechanisms other than agonist neural drive. A possible explanation for the BLD in both evoked and voluntary force is a difference in postural stability/ stabiliser activation requirements during UL and BL actions. Stabiliser activation was not measured within the present study, but is thought to be important for optimal force expression (Folland & Williams, 2007a). For instance, Nozaki et al. (2007) demonstrated that even during a relatively simple task such as an isometric knee extension used within the current study, that there was a large variation, both between and within-participants in the ability to stabilise the adjacent joint torque through effective inter-muscular coordination. The greater postural requirement for BL than UL strength tasks has been proposed as the mechanism accounting for the BLD (Herbert & Gandevia, 1996). In support of this suggestion, the BLD has been observed to be higher in an action requiring greater activation of postural stabilising muscles (leg press versus hand grip, Magnus & Farthing, 2008). In the current study insufficient stabilisation during BL explosive contractions may have afforded greater movement of adjacent joints, particularly the hips, increasing biological compliance and reducing explosive force production. Whilst the BLD in evoked explosive force we have observed might appear to contradict this possibility (as only the agonists are activated by the stimulation), there is undoubtedly stabiliser activation in anticipation of, and/or in response to, the stimulation, and this could be similarly less effective in the BL compared to UL situation. The similarity of MVF across BL and UL contractions might also argue against a role of stabiliser activation in the BLD we have

observed, however during these longer contractions force production is unlikely to be influenced by compliance and hence stabilisation. Future research should consider the role of stabiliser muscle activation on the BLD.

The observed 15% deficit in RFD, despite no influence of BL actions on MVF has important implications for sport and exercise training science and suggests specific training to offset this deficit should be performed in order to maximise the performance of BL explosive sporting tasks. The observed deficit could be explained by reduced inter-muscular coordination (lower stabiliser activation) during BL efforts and suggests that specific practice of coordinated explosive BL tasks and improved core/joint stability could be expected to improve the expression of BL explosive sporting tasks through reducing this RFD BLD.

In summary, there was a BLD in explosive but not MVF of the knee extensors, which was specific to RFD_{50-100}. Measurement issues played only a minor role on the observed BLD. The novel finding of a BLD in evoked force production and no change in agonist EMG during explosive voluntary efforts suggest the BLD in voluntary explosive force was not attributable to reduced agonist neural drive, but was explained by an alternative mechanism.

Chapter 6

Inter-muscular Coordination and Explosive Isometric Force Production in the Elbow Flexors

6.1 Introduction

As alluded to throughout the PhD, explosive strength is considered an important characteristic of muscle performance (Marcora & Miller, 2000; Paasuke et al., 2001; de Ruiter et al., 2006) as well as for the stabilisation of the musculo-skeletal system following mechanical perturbation (Fleming et al., 1991; Izquierdo et al., 1999; Chang et al., 2005; Pijnappels et al., 2008), and thus the prevention of falls and injury. Therefore, it is of considerable importance to have a detailed understanding of the determinants of explosive strength.

Numerous factors have been demonstrated to be associated with isometric explosive force capabilities such as isometric maximum voluntary force production (MVF; Andersen & Aagaard, 2006), morphological factors such as fibre type composition (Harridge et al., 1996; Viitasalo et al., 1978) and muscle-tendon unit (MTU) stiffness (Bosjen-Moller et al., 2006), as well as neural factors such as the level of agonist neuromuscular activation (de Ruiter et al., 2004; Del Balso & Cafarelli, 2007; Tillin et al., 2010). However, no study has considered the relative importance of the level of stabiliser neuromuscular activation on explosive force production. The effective stabilisation of joints is thought to be important for optimal force production (Sale, 1993; Folland & Williams, 2007a). A key finding from chapter 5 was that the BLD in explosive force observed was not explained by the level of agonist or antagonist activation and may possibly be explained by differences in stabiliser activation requirements between tasks. However, no study has actually investigated if stabiliser activation may influence explosive isometric strength.

The contribution of neural activation of particular types of muscle groups (agonists, antagonist, stabilisers) to joint performance is typically quantified by examining either the relationship between muscle activation of particular muscle groups and force output of the respective joint (i.e., de Ruiter et al., 2004, 2010; Del Balso & Cafelli, 2007) or by documenting the change in muscle activation and strength following particular interventions such as resistance training (i.e., Aagaard et al., 2002a; Tillin et al., 2011, 2012). The contribution of muscle activation to joint performance has to date only been examined by considering the respective muscle groups in isolation, with no consideration of the possible inter-relations between agonist, antagonist and stabiliser muscle activation during explosive actions. Muscles require sufficient stability at the origin to appropriately contract and apply force to contribute to torque production at the required joint. Mono-articular muscles such as

the brachialis fixate to bony processes and thus have sufficient base of support. However, bi-articular muscles such as the biceps brachii originate from the adjacent joint and therefore would be expected to require sufficient adjacent joint stability to provide a solid base in order to effectively apply force at the tendon insertion (i.e., act at the elbow as an agonist). It is possible therefore, that poor adjacent joint stability (i.e., low stabiliser muscle activity), may compromise activation of the bi-articular agonist muscle group and consequently, indirectly impact on the force capabilities about the primary joint. It is important to examine the relationship between agonist activation and stabiliser activation during explosive isometric efforts to help fully elucidate a possible role of stabiliser activation on isometric explosive strength.

The aim of this study was to investigate the relationship between the level of stabiliser activation and explosive isometric force production and then consider the inter-relationships between agonist, antagonist and stabiliser activation across the rising force-time curve. It was hypothesised that the level of stabiliser activation would be related to the level of explosive force production. It was further hypothesised that a relationship would also exist between the level of stabiliser activation and activation of the bi-articular agonists and once this relationship was accounted for, there would be no independent contribution of stabiliser activation to the explained variance in explosive force capabilities.

6.2 Methods

6.2.1 Participants

Thirty-six male participants (age, 23 ± 2 yr; height, 1.77 ± 0.08 m; mass, 73.7 ± 9.9 kg) completed the study. The participants were physically active, healthy, injury free and had not taken part in any form of strenuous upper body exercise for the previous 12 months prior to the study. All participants provided written informed consent prior to their involvement in the study, which was approved by the Loughborough University ethical advisory committee.

6.2.2 Overview

Participants attended the laboratory on two occasions, once for familiarisation and then for a main trial one week later. The two trials involved the same protocol and were completed at a consistent time of day for each patient. The measurement sessions involved the assessment of force and surface EMG during maximal and explosive isometric contractions of the elbow flexors. EMG was recorded over the agonist, antagonist and stabiliser muscles throughout these volitional contractions. EMG obtained during explosive isometric efforts was normalised to the maximal EMG obtained from each muscle during reference contraction whilst acting as an agonist maximum isometric elbow flexion (agonists); maximum isometric elbow extension (antagonist); and maximum isometric bench press (stabilisers).

6.2.3 Measurements

Participants sat upright (hip joint angle of 90°) in a custom built strength testing chair and were strapped at the hip and chest to the seat and back of the chair to prevent movement of the body (Figure 6.1). The elbow and shoulder joints were flexed to 60 and 90°, respectively (0° being full elbow extension), with the upper arm placed on a horizontal board, and externally rotated with the elbow position maintained by blocks anterior and lateral to the joint. The forearm was supinated and the wrist was attached to an S-Beam tension-compression load cell (Applied Measurements Ltd, Berkshire, UK) positioned perpendicular to the forearm during elbow flexion/extension. The force signal was amplified ($\times$ 500) interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK) and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). During off-line analysis the force signal was low-pass filtered (500 Hz cut-off) with a fourth order zero-lag Butterworth digital filter. Real-time biofeedback of the force response was provided on a computer monitor.

Figure 6.1 The isometric strength testing apparatus used to measure elbow flexion/extension force.

EMG was recorded using two Delsys Bangnoli-4 EMG systems (Delsys, Boston, USA) from two agonist muscles (short and long heads of the biceps brachii, BBS and BBL), the antagonist (triceps brachii lateral head, TB) and two stabilisers (anterior deltoid, AD; clavicular head of the pectoralis major, PM). Following preparation of the skin (shaving, abrading and cleansing with 70% ethanol), the same investigator attached double differential surface electrodes (1 cm inter-electrode distance, DE-3.1, Delsys) to the skin over each of the muscles using adhesive interfaces. The electrodes were positioned at specific measured sites along the arm, in the centre of the muscle belly and parallel to the presumed orientation of the muscle fibers. BBS and BBL electrodes were positioned distally 75% of the distance between the coracoid process and medial epicondyle of the humerus (Lee et al., 2010). AD, PM and TB were positioned according to SENIAM guidelines. The EMG reference electrodes were placed on the contralateral clavicle. EMG signals were amplified ($\times$ 100, differential amplifier 20-450 Hz) and sampled at 2000 Hz with the same analogue to differential converter and PC as the force signal. During off-line analysis the EMG signals were band-pass filtered in both directions (6-500 Hz) using a 4th order zero-lag Butterworth digital filter.

6.2.4 Protocol

Participants began the trial by first completing a warm up of submaximal isometric voluntary contractions. They then performed three elbow flexion isometric maximum voluntary contractions (iMVCs) (separated by 1 min) followed by three elbow extension iMVCs, in

which participants were instructed to pull or push as hard as possible for ~3 seconds. Biofeedback and verbal encouragement were provided during and between each iMVC. Elbow flexor isometric maximal voluntary force (iMVF) was the greatest instantaneous force achieved by the participant in any of the iMVCs. EMG from all muscles recorded during explosive elbow flexion was normalised to the maximal EMG (EMG_{max}) achieved during reference tasks when each muscle group was acting as an agonist. Biceps Brachii EMG_{max} was recorded during the elbow flexor MVCs and assessed as the RMS of a 500 ms epoch around MVF (250 ms either side; BB EMG_{max}). EMG_{max} of the TB (TB EMG_{max}) was assessed as a 500 ms epoch at elbow extension iMVF (250 ms either side). Participants then performed 10 isometric explosive voluntary contractions (separated by 20 s). For each contraction participants were instructed to flex their arm as 'fast' and as hard as possible for ~1 s from a relaxed state (Sahaly et al., 2001) and achieve at least 80% iMVF. Participants were instructed to avoid any countermovement or pre-tension. To determine if countermovement had occurred, the resting force level was displayed on a sensitive scale. The slope of the force time curve (10 ms time constant) was displayed throughout and the peak slope was used to provide visual feedback to participants after each contraction. The three contractions with the highest peak slope, no discernible countermovement or pre-tension (change in force of < 0.5 N in the preceding 100 ms), and with a peak force of at least 80% iMVF were used for analysis, and all measurements were averaged across these three contractions. Force was assessed at 50, 100, and 150 ms, from the onset of contraction and reported in absolute terms and normalised to iMVF. The RMS EMG was measured in windows of 0-50, 0-100 and 0-150 ms from the onset of EMG activity in the first agonist muscle to be activated. Agonist BBS and BBL RMS EMG were normalised to EMG_{max}. Stabiliser AD and PM RMS EMG were normalised to the EMG_{max} during the isometric maximum bench press (see below) and then averaged to give a mean value. Antagonist EMG was normalised to the TB EMG_{max} (antagonist EMG).

Participants performed isometric bench press contractions to determine maximal EMG of the stabilising muscles (AD and PM EMG_{max}). Participants lay supine on an inclined bench (head up ~ 15° to the horizontal) with their shoulders aligned vertically below a fixed immovable bar and their knees bent and feet positioned on the end of the bench, which was positioned on a portable force plate (Quattro Jump, Type 9290 AD, Kistler, Switzerland). The height of the bar was adjusted so that when participants grasped the bar their upper arm was horizontal, whilst their forearms were vertical (i.e. shoulders abducted at 90° and elbows flexed at 90°).

Participants performed three iMVCs of 3 s duration with $\geq$ 30 s rest between efforts. Verbal encouragement was provided throughout the efforts and the recorded force during the effort was provided as feedback to the participants following each attempt. AD and PM EMG_{max} was recorded over the highest 500 ms epoch during the series of bench press iMVCs and used for normalisation of stabiliser EMG during elbow flexor strength tasks.

6.2.5 Data Analysis

For the explosive voluntary contractions, identification of force and EMG onsets were made manually (visual identification) as this is considered the "gold standard" method (Allison, 2003; Moretti et al., 2003; Pain & Hibbs, 2007, Pulkovski et al., 2008). The same investigator identified signal onsets with a constant y-axis scale of ~ 2 N and 100 mV, for force and EMG respectively, and an x-axis scale of 500 ms. A vertical cursor was then placed on the onset and viewed at a higher resolution to determine its exact location (~ 0.5 N and 50 mV for force and EMG axes respectively using an x-axis of 25 ms).

Descriptive statistics are presented as mean ± standard deviation (SD). Preliminary analysis involved calculating Pearson's product moment bivariate correlations between explosive voluntary force and individual neural predictor variables (agonist, antagonist, stabiliser EMG) and between the individual predictor variables themselves. In the event of significant relationships between stabiliser activation and force parameters, stabiliser activation was entered into a stepwise multiple linear regression analysis alongside other neural factors to establish if it exerted an independent contribution once it's possible association with other muscle groups was considered. Statistical analyses were performed using SPSS version 19 (SPSS inc., Chicago, IL, USA) and statistical significance was set at $P < 0.05$.

6.3 Results

6.3.1 Neural Contributions to Explosive Force Production

iMVF was 239 ± 42 N. Descriptive statistics for absolute and relative explosive force and EMG of the agonist, antagonists and stabilisers are displayed in Table 6.1. The level of neuromuscular activation of the stabilisers throughout the initial 150 ms was 28.9-41.6 % EMG_{max} (Table 6.1). Stabiliser EMG during the early phase of the contraction (0-50 ms) was

significantly related to both absolute and relative explosive force at 50 ms ($r = 0.35$-0.44, $P \leq 0.033$). There was no relationship between stabiliser activation and force production over longer time periods from EMG/force onset ($r \leq 0.230$, $P \geq 0.170$).

Agonist EMG was related to absolute force at 50 ($r = 0.60$, $P < 0.001$) and 100 ms ($r = 0.39$, $P = 0.016$). Furthermore, agonist EMG over the respective time period was related to relative explosive force throughout the initial 150 ms of contraction ($r = 0.37$-0.50, $P \leq 0.025$). There was no relationship between antagonist EMG and absolute or relative explosive force at any time point during the explosive efforts ($-0.07 \geq r \leq 0.102$, $P \geq 0.548$).

Table 6.1 Explosive force and normalised EMG of the agonists, antagonists and stabilisers during explosive isometric contractions of the elbow flexors. Data are displayed as mean ± SD (N = 36).

Variable	Time Point after force onset (ms)		
	50	100	150
Absolute Force (N)	60.2 ± 20.3	156.3 ± 29.5	190.9 ± 33.1
Relative Force (%MVF)	25.5 ± 8.9	65.7 ± 8.1	80.2 ± 6.2
Agonist EMG (%EMG_{max})	59.2 ± 32.0	82.0 ± 28.7	90.1 ± 26.2
Antagonist EMG (%EMG_{max})	1.6 ± 1.7	3.3 ± 2.3	4.0 ± 2.5
Stabiliser EMG (%EMG_{max})	28.9 ± 19.5	39.7 ± 18.4	41.6 ± 16.9

N, Newtons; MVF, maximum voluntary force; EMG, electromyography

6.3.2 Inter-relations of EMG Amplitude during Explosive Efforts

Agonist and stabiliser EMG were significantly related to one another throughout all time periods during the initial 150 ms of explosive force production ($r = 0.51$-0.70, $P \leq 0.001$). Additionally, there was a negative relationship between stabiliser EMG_{0-150} and antagonist EMG_{0-150} ($r = -0.33$, $P = 0.047$). There was no relationship between agonist and antagonist EMG for either time period ($-0.29 \leq r \leq -0.14$, $P \geq 0.084$).

Multiple regression analysis confirmed that the level of stabiliser EMG did not further contribute to the explained variance of either absolute or relative force at 50 ms, or any other time point, once agonist EMG was accounted for ($P \geq 0.493$). The explained variance of

agonist EMG for absolute and relative explosive force at 50 ms was 0.36 and 0.25 respectively.

6.4. Discussion

The primary purpose of the present study was to establish the relationship of stabiliser muscle activation on explosive isometric force production of the elbow flexors and report the inter-relationship of muscular activation from agonist, antagonist and stabiliser muscles during explosive isometric contractions. Stabiliser neuromuscular activation was significantly related to absolute and relative explosive force production over the initial phase (50 ms) of the contraction. Furthermore, Stabiliser EMG was also strongly associated with the level of agonist EMG amplitude at all time points after force onset. When the relationship between agonist and stabiliser EMG was accounted for (through multiple linear regression analysis) there was no independent contribution of stabiliser activation to the explained variance in either absolute or relative explosive force production. Additionally, although the level of antagonist activation was not associated with either absolute or relative explosive strength, or with the level of agonist activation, there was a small negative relationship with the level of stabiliser neuromuscular activation.

6.4.1 Neural Contributions to Explosive Force Production

A key finding of the study was the association of stabiliser EMG with early phase (50 ms) absolute and relative explosive force production, suggesting it was an important neural component in explaining the variability in explosive strength between participants. No previous research has attempted to document the relationship between stabiliser activation and explosive force capabilities. Similarly to previous research a significant relationship was observed between the level of agonist neuromuscular activation and explosive force during the early phase (50 ms) of the contraction (de Ruiter et al., 2004; Del Balso & Cafarelli, 2007; Hannah et al., 2012). This observed relationship was lower than previously reported by some of these studies (de Ruiter et al., 2004, 2007) but similar to others (Hannah et al., 2012; Folland et al., 2013). Although to a lesser extent, agonist EMG was also related to explosive force at 100 ms ($r^2 = 0.15$), but there was no relationship with force at 150 ms. Furthermore,

there was a significant relationship between agonist EMG and relative explosive force at all time points (r^2 = 0.14 – 0.25). Finally, there was no association between the level of antagonist EMG amplitude and either absolute or relative explosive force production. It is important to note that the current study only assessed the biceps brachii (short and long head) and single antagonist (triceps brachii lateral head) and therefore the muscles do not reflect the entire elbow flexor/extensors muscle group. The brachialis is a mono-articular elbow flexor muscle and is thought to be the main agonist of the elbow flexor muscle group, and therefore the relationships observed within the current study unlikely fully reflect the importance of agonist activation on elbow flexor explosive force production.

6.4.2 Inter-relations of Muscle Activation during Explosive Force Production

This is the first study to assess the inter-relations of neuromuscular activation of the agonist, antagonists and stabilisers during explosive contractions. A key finding of the study was the strong relationship observed between agonist and stabiliser EMG during the early phase of the contraction (r^2 = 0.49). This relationship decreased as the time from onset increased, but there was still a strong relationship over 100 and 150 ms (r^2 = 0.26 – 0.30). This finding may highlight the importance of effective joint stability for optimal agonist activation during the early phase of explosive isometric contractions. Once this relationship was accounted for (stepwise multiple linear regression analysis) there was no independent contribution of stabiliser EMG to the explained variance in early phase absolute and relative explosive force production. This suggests that the stabiliser muscle activation capability is not a direct determinant of isometric explosive force production, but instead may exert an indirect influence on explosive force production through its association with the capability to activate the agonist muscles during the early phase of the rising force time-curve. It is possible that stabiliser activation serves as a foundation for bi-articular agonist activation in that the bi-articular bicep brachii require a base of support in which to appropriately act as an agonist at the elbow joint. The relationship observed in the present study could suggest that either a) the level of stabiliser neuromuscular activation is inter-linked with agonist neuromuscular activation, in that those with an enhanced capability to activate the agonists during the rising force-time curve require higher stabiliser activation or b) actually the level of agonist output during explosive force development is constrained by the ability to sufficiently stabilise the shoulder joint (stabiliser activation). From the current study design it is not possible to

delineate which of these scenarios is valid. Further research is needed to consider how the level of stabiliser activation may influence the expression of agonist activation.

It is important to note that the present study only assessed two of the stabilisers at the shoulder joint (pectoralis major and anterior deltoid), which may not fully reflect activation of all the stabilisers and therefore stability of the joint. Thus, further research replicating the study with a larger pool of stabiliser muscles may be required to fully elucidate these findings. However, the strong association observed in the presence of only a small number of stabilisers, performed utilising an isometric model, highlights a potentially influential role of stabiliser activation on muscle performance. Future research should begin to explore the role of the stabiliser muscle system on muscle performance during more functionally relevant tasks.

The observed negative relationship between stabiliser activation and antagonist activation over 150 ms may suggest a superior muscle coordination strategy. Joint torque is a consequence of both agonist activation and antagonist activation, and therefore a lower level of antagonist activation would allow for a greater net joint torque. Stabiliser activation has been shown to increase following RT, with concomitant declines in antagonist activation during sub-maximal contractions (Cacchio et al., 2008), that has been suggested to be as a result of altered motor control strategy, re-organised to optimise force production. Therefore, optimal stabiliser activity could be a trained strategy indicative of enhanced neural control, designed to optimally enhance force capabilities.

In summary, the study demonstrates an important role of stabiliser activation on explosive strength through its association with agonist activation. Once agonist activation was accounted for, stabiliser activation no longer independently influenced absolute or relative explosive force production. Furthermore, the study reporting a negative relationship between stabiliser activation and antagonist activation, potentially indicative of superior muscle control strategies. The current study provides stimulating findings which need to be further replicated and applied to more functionally relevant situations.

CHAPTER 7

Task-specific neural adaptations to isoinertial resistance training

7.1 Introduction

Marked increases in muscle strength during the early phase of a RT program have been observed (Abe et al., 2000; Pucci et al., 2006; Del Balso & Cafarelli, 2007) and these changes appear to be highly specific to the nature of the training task. For example, conventional dynamic isoinertial RT has repeatedly been found to produce disproportionately greater increases in isoinertial lifting strength (one repetition maximum [1RM]) than isometric strength (Thorstensson et al., 1976, Rutherford & Jones, 1986). This specificity of training phenomenon is often taken as evidence for neural adaptations to RT (Folland & Williams, 2007a). However at present there is minimal direct evidence for either neural or morphological mechanisms that might explain this training task specificity. Furthermore, as functional tasks generally require isoinertial strength, establishing the mechanisms that lead to a greater improvement in isoinertial versus isometric strength is important from both a sport and rehabilitation perspective.

Early adaptations to RT are thought to be primarily explained by neural adaptations, e.g. adaptations in agonist, antagonist and stabiliser muscle activation (Folland & Williams 2007a), with a greater contribution from morphological adaptations, such as selective hypertrophy and/or architectural changes, as training duration progresses (Narici et al., 1996). Therefore, documenting training specific adaptations over a short term training period may distinguish between the neural and morphological explanations for the task specificity phenomenon. Although numerous studies have reported an increase in absolute agonist EMG amplitude during isometric tasks following RT (Hakkinen et al., 1983, 1998; Kubo et al., 2006; Tillin et al., 2011) only one investigation has found an increase in agonist EMG during an isoinertial task (Hakkinen et al., 1998). Moreover, the comparative changes in agonist activation during isometric and isoinertial tasks, or how these changes may explain the strength gains that occur, have not been elucidated. Furthermore, the evidence for RT-induced changes in antagonist muscle co-activation is equivocal (Hakkinen et al., 1998; Pucci et al., 2006; de Boer et al, 2007; Tillin et al., 2011). Whilst an increase in antagonist co-activation might attenuate gains in strength, for some joints this may be a necessary adaptation to maintain joint integrity in response to increased loading post-training (Tillin et al., 2011).

In addition to agonist and antagonist activation, changes in stabiliser muscle activation could also influence strength gains and contribute to task specificity, but has not previously been

investigated in any strength tasks. Learning to effectively stabilise the adjacent joints through increased stabiliser activation may facilitate an increase in strength during the early phase of RT. Additionally, isoinertial lifting tasks involve more degrees of freedom within the musculo-skeletal system than isometric strength tasks, and therefore may require a greater degree of stabilisation. Although the influence of RT on stabiliser EMG during submaximal contractions has been investigated (Cacchio et al., 2008), the contribution of adaptations in stabiliser activation to the changes in maximum isoinertial or isometric strength after RT has not been examined.

Explosive strength is also considered an important characteristic of muscle performance (de Ruiter et al., 2006; Tillin et al., 2013), as well as for the stabilisation of the musculoskeletal system following mechanical perturbation (Fleming et al., 1991; Izquierdo et al., 1999; Chang et al., 2005; Pijnappels et al., 2008), thus reducing the risk of falls and injury. The efficacy of RT for improving explosive isometric strength is controversial, with some reports finding an improvement (Hakkinen et al., 1998; Aagaard et al., 2002a) and others no change (Andersen et al. 2010; Tillin et al., 2011). Moreover, it is not known whether just three weeks of conventional RT will influence explosive strength, and if so, whether this effect is due to specific neural adaptations.

The aim of the present study was to assess the task specific adaptations in isometric (maximum and explosive) and isoinertial strength following three weeks of isoinertial RT, and to document the concurrent neural changes in agonist, antagonist and stabiliser muscle activation. It was hypothesised that conventional isoinertial RT would induce greater increases in isoinertial, than isometric strength, and that this would be concomitant with greater changes in agonist and stabiliser muscle activation during the isoinertial task.

7.2 Methods

7.2.1 Participants

Forty-five male participants (age, 23 ± 3 yr; height, 1.77 ± 0.08 m; mass, 73.7 ± 9.9 kg) completed the study. The participants were physically active, healthy, injury free and had not taken part in any form of strenuous upper body exercise for the previous 12 months prior to the study. All Participants provided written informed consent prior to their involvement in the

study, which complied with the Declaration of Helsinki and was approved by the Ethical Advisory Committee of Loughborough University.

7.2.2 Overview

Participants were tested twice pre (pre-1, pre-2) and once post three weeks of isoinertial elbow flexor RT. Pre-1 and Pre-2 training measurements were separated by seven days, with pre-2 measurements recorded 3-7 days prior to the commencement of training. Post-training measurements were collected 3-5 days after the last training session. The measurement sessions were all performed at a consistent time of day for each participant. The training involved participants performing elbow flexion exercises (unilateral and bilateral preacher curls) three times per week (Monday, Wednesday, Friday) for three weeks. Although participants trained both arms, measurements were only recorded from the dominant arm. During each measurement session participants performed a series of elbow flexion contractions to determine maximum isoinertial lifting strength (1RM), and isometric maximum and explosive strength. Surface EMG was recorded during pre-2 and post measurement sessions from the agonist, antagonist and stabiliser muscles throughout these volitional contractions as well as during reference tasks/measurements that were used for normalisation of EMG: a supramaximal evoked compound action potential (M_{max}) of the biceps brachii (agonists); maximum isometric elbow extension (antagonist); and maximum isometric bench press (stabilisers). Muscle thickness of the short-head of the biceps brachii was also measured at rest using ultrasound during both the pre-2 and post- measurement sessions. The effects of training were determined by comparing pre-2 to post-training measurements.

7.2.3 Training

Each training session involved two similar elbow flexion exercises. Firstly, unilateral elbow flexion curls were performed with a dumbbell on a modified preacher bench (Body Solid, Forest Park, IL, USA; Figure 7.1A with participants performing alternate sets with the dominant and then the non-dominant arms. Secondly, participants performed bilateral elbow flexion exercises using a weights machine (Pro Club Line Bicep Curl; Body Solid, Forest Park, IL, USA). Both exercises were performed with a load of 8-10RM. Two sets of each

exercise were performed, with two minutes rest between exercises involving the same muscle group. The training load was increased when participants could complete 10 repetitions on both sets. Participants were instructed to lower the weight in a controlled manner over 2-3 s during the eccentric phase, and then lift the weight as quickly as possible during the concentric phase, in order to maximise the rate of force development. They were also directed to move through a full range of motion (from ~15° to ~140°; 0° = full extension) throughout both exercises.

7.2.4 Measurement Trials

7.2.4.1 Measurements

Participants sat upright (hip joint angle of 90°) in a custom built strength testing chair and were strapped at the hip and chest to the seat and back of the chair to prevent movement of the body (Figure 7.1A). The elbow and shoulder joints were flexed to 60 and 90°, respectively (0° being full elbow extension), with the upper arm placed on a horizontal board, and externally rotated with the elbow position maintained by blocks anterior and lateral to the joint. The forearm was supinated and the wrist was attached to an S-Beam tension-compression load cell (Applied Measurements Ltd, Berkshire, UK) positioned perpendicular to the forearm during elbow flexion/extension. The force signal was amplified ($\times$ 500) interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK) and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). During off-line analysis the force signal was low-pass filtered (500 Hz) with a fourth order zero-lag Butterworth digital filter. Real-time biofeedback of the force response was provided on a computer monitor.
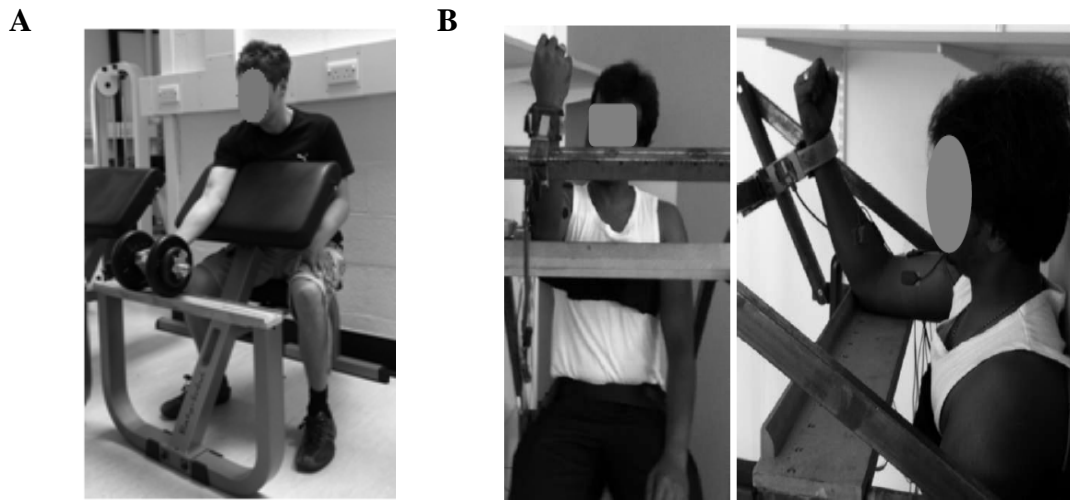
Figure 7.1 A, The modified preacher curl bench used for both training and testing of the elbow flexors and B, The isometric strength testing apparatus used to measure elbow flexion/ extension force.

Surface EMG was recorded using two Delsys Bangnoli-4 EMG systems (Delsys, Boston, USA) from two agonist muscles (short and long heads of the biceps brachii, BBS and BBL), the antagonist (triceps brachii lateral head, TB) and two stabilisers (anterior deltoid, AD; clavicular head of the pectoralis major, PM). Following preparation of the skin (shaving, abrading and cleansing with 70% ethanol), the same investigator attached double differential surface electrodes (1 cm inter-electrode distance, DE-3.1, Delsys) to the skin over each of the muscles using adhesive interfaces. The electrodes were positioned at specific measured sites along the arm, in the centre of the muscle belly and parallel to the presumed orientation of the muscle fibers. BBS and BBL electrodes were positioned distally 75% of the distance between the coracoid process and medial epicondyle of the humerus (Lee et al., 2010). AD, PM and TB were positioned according to SENIAM guidelines. The EMG reference electrodes were placed on the contralateral clavicle. EMG signals were amplified ($\times$ 100, differential amplifier 20-450 Hz) and sampled at 2000 Hz with the same analogue to differential converter and PC as the force signal. During off-line analysis the EMG signals were band-pass filtered in both directions (6-500 Hz) using a 4th order zero-lag Butterworth digital filter.

The musculocutaneous nerve was electrically stimulated (via a constant current, variable voltage stimulator; DS7AH, Digitimer Ltd., UK) with square wave pulses (0.2 ms duration) to elicit twitch contractions, and facilitate measurement of compound muscle action potentials (*M*-waves) with EMG. The self-adhesive anode (5 x 5 cm; Verity Medical, Andover, UK) was attached to the skin over the triceps brachii. The cathode (1 cm diameter,

Electro Medical Supplies, Wantage, UK) was held in place over the nerve between both biceps brachii heads at ~25% of the distance between the coracoid process and medial epicondyle of the humerus (Lee et al., 2010). However, the precise location was determined as the position that evoked the greatest response for a particular electrical current (10-30mA).

7.2.4.2 Protocol

1RM was assessed with a series of incremental dumbbell lifts according to an adapted protocol (Baecke, 1982). Isoinertial strength was measured with the same modified preacher bench used during training (Figure 7.1A). Briefly, the preacher bench was modified with a horizontal rack at the end of the motion that provided a consistent starting point pre and post training for the isoinertial lifts. The height of the bench was adjusted according to the length of each participant's arm, so that it was underneath the axilla, with the participant leaning forward on to the bench such that the shoulder was flexed at ~75º. This position ensured the elbow was fully extended when each participant's hand gripped the dumbbell on the rack. Participants were instructed to lift the dumbbell through the full range of motion, from full elbow extension to full flexion. The non-lifting hand rested on the knee of the same side, both feet remained flat on the floor and the knees were flexed at 90°. Participants warmed up by performing 10 reps at 40% of their previous 1RM (or estimated 1RM for pre-1). After 1 min rest, participants performed 3 repetitions at 80% of their previous 1-RM. Thereafter they performed a series of single lifts interspersed with 1 min rest intervals, firstly at the previous 1-RM, and then at increments of +0.5 kg if the preceding lift was successful. The 1-RM was defined as the highest load lifted on that occasion, and was generally determined within 3-5 attempts, although more attempts were completed if necessary. EMG amplitude of all the muscles during the 1-RM lift was assessed for a 500 ms epoch that gave the highest agonist RMS EMG during the concentric phase of the lift. BBS and BBL RMS EMG were normalised to M_{max} peak-to-peak amplitude (see below) and then averaged to give a mean value for the biceps brachii (agonist EMG); AD and PM RMS EMG were normalised to the maximum value attained by each muscle during the isometric maximum bench press (see below) and then averaged to give a mean value for the stabiliser muscles (stabiliser EMG); and antagonist EMG was normalised to the TB EMG at elbow extension iMVF (antagonist EMG; see below).

Participants were transferred to the isometric elbow flexion/extension apparatus (Fig. 7.1B) and completed a warm up of submaximal isometric voluntary contractions. They then performed three elbow flexion isometric maximum voluntary contractions (iMVCs) (separated by 1 min) followed by three elbow extension iMVCs, in which participants were instructed to pull or push as hard as possible for ~3 seconds. Biofeedback and verbal encouragement were provided during and between each iMVC. Elbow flexor isometric maximal voluntary force (iMVF) was the greatest force achieved by the participant in any of the iMVCs. The amplitude of the EMG signal was assessed as the RMS of a 500 ms epoch around peak force (250 ms either side) for each muscle. EMG was normalised in the same manner as for the 1RM efforts. Maximal EMG of the TB (EMG_{max}) was assessed as a 500 ms epoch at elbow extension iMVF (250 ms either side) and used for normalisation of antagonist EMG during elbow flexion tasks.

Participants then performed 10 isometric explosive voluntary contractions (separated by 20 s). For each contraction participants were instructed to flex their arm as 'fast' and as hard as possible for ~1 s from a relaxed state (Sahaly et al., 2001) and achieve at least 80% iMVF. Participants were instructed to avoid any countermovement or pre-tension. To determine if countermovement had occurred, the resting force level was displayed on a sensitive scale. The slope of the force time curve (10 ms time constant) was displayed throughout and the peak slope was used to provide visual feedback to participants after each contraction. The three contractions with the highest peak slope, no discernible countermovement or pre-tension (change in force of < 0.5 N in the preceding 100 ms), and with a peak force of at least 80% iMVF were used for analysis, and all measurements were averaged across these three contractions. Force was assessed at 50, 100, and 150 ms, from the onset of contraction and reported in absolute terms and normalised to iMVF. Peak rate of force development (pRFD) was measured as the maximum slope (10 ms time constant), and the time at which it occurred was also recorded. The force-time integral (the area beneath the force-time curve) was assessed in windows of 0-50, 0-100 and 0-150 ms from the onset of force. The RMS EMG was measured in windows of 0-50, 0-100 and 0-150 ms from the onset of EMG activity in the first agonist muscle to be activated, and normalised in the same manner as during the maximal strength tasks. The time between the first agonist muscle to be activated and onset of force was determined as the maximum electromechanical delay (EMD_{max}).

For the explosive voluntary contractions, identification of force and EMG onsets were made manually (visual identification) as this is considered the "gold standard" method (Allison,

2003, Moretti et al., 2003). The same investigator identified signal onsets with a constant y-axis scale of ~ 2 N and 100 mV, for force and EMG respectively, and an x-axis scale of 500 ms. A vertical cursor was then placed on the onset and viewed at a higher resolution to determine its exact location (~ 0.5 N and 50 mV for force and EMG axes respectively using an x-axis of 25 ms).

Single twitch contractions were elicited by stimulation of the musculocutaneous nerve at incremental intensities until there was a plateau in the *M*-wave response of both heads of the bicep brachii. Thereafter, the current was increased by 20% and three supra-maximal twitches were elicited (separated by 12 s). The average *M*-wave peak to peak amplitude (M_{max}) of these three supramaximal *M*-waves was determined for each muscle and used for normalisation of agonist EMG during elbow flexor strength tasks. Six participants were uncomfortable with the stimulation and thus EMG normalised to M_{max} is reported for N = 39.

Participants performed isometric bench press contractions to determine maximal EMG of the stabilising muscles (AD and PM). Participants lay supine on an inclined bench (head up ~15° to the horizontal) with their shoulders aligned vertically below a fixed immovable bar and their knees bent and feet positioned on the end of the bench, which was positioned on a portable force plate (Quattro Jump, Type 9290 AD, Kistler, Switzerland). The height of the bar was adjusted so that when participants grasped the bar their upper arm was horizontal, whilst their forearms were vertical (i.e. shoulders abducted at 90° and elbows flexed at 90°). Participants performed three iMVCs of 3 s duration with ≥ 30 s rest between efforts. Verbal encouragement was provided throughout the efforts and the recorded force during the effort was provided as feedback to the participants following each attempt. Maximal EMG of the AD and PM muscles was recorded over the highest 500 ms epoch during the series of bench press iMVCs (EMG_{max}) and used for normalisation of stabiliser EMG during elbow flexor strength tasks.

In-vivo muscle thickness was examined prior to the start of each measurement session, using an ultrasound scanner (SSA-370A Power Vision 6000, Toshiba Corporation, Otawara-Shi, Japan) with a 6 cm (8 MHz) linear array transducer. Scans of the BBS were obtained from the dominant arm, whilst participants lay supine with the elbow fully extended and the shoulder abducted at 90°. Strips of surgical tape (50 mm long, 2 mm wide; 3M, Neuss, Germany) were placed at 50 mm intervals along the length of the upper arm from the cubital crease to the shoulder and acted as markers with which muscle length could be determined. Muscle

thickness was then assessed at 25, 50 and 75% BBS muscle length. The values at the three sites were averaged to give a mean BBS muscle thickness value. Reliability analysis was performed with N = 8 who performed measures in duplicate. For this, the surgical strips were removed, and the testing procedure was repeated. Due to technical reasons, nine of the participants were unable to complete both pre and post muscle thickness measurements. Therefore, muscle thickness data are reported for N = 36.

7.2.5 Statistical Analysis

Data are reported as mean ± standard error of the mean (SEM). Significant differences between pre-2 and post-training absolute measures (iMVF, 1RM and EMG during the respective contractions, muscle thickness, pRFD and time to pRFD during the explosive contractions) were determined using paired t-tests. Effect sizes were reported to interpret the magnitude of these differences according to Cohen's d, where 0.2 is a small effect, 0.5, a moderate effect and > 0.8 a large effect. Relative changes in 1RM and iMVF were calculated as mean ± SEM of individual percentage changes, and compared with a paired t-test. Two-way repeated measures ANOVA were performed to contrast the changes in EMG variables for iMVF and 1RM with training (task: MVF vs. 1RM; training, pre vs. post).

Time-series data during the explosive contractions (force and EMG) were assessed with two-way repeated measures ANOVA (training [pre vs. post] vs. epoch [0-50, 0-100, 0-150 ms]). Post-hoc pair-wise comparisons (Bonferroni corrected paired t-test) were used to determine if there were pre- vs. post-training differences at specific time points. Statistical analysis was completed using SPSS version 17, and the significance level was set at $P < 0.05$. Reliability analysis for strength data was performed using pre-1 and pre- 2 measurements in which 15 participants were chosen at random from the data sets of 1RM, iMVF and explosive force production. Significance testing to assess the consistency of the mean values (as above) was determined and the within-participant coefficient of variation (CV) and intra class coefficient (ICC) used to further determine reliability.

7.3 Results

7.3.1 Reliability

There were no significant changes in iMVF (paired t-test, P = 0.473), 1RM (paired t-test, P = 0.827) or explosive force production (ANOVA, P = 0.127) from pre-1 to pre-2 measurement sessions. Furthermore, these strength measures demonstrated moderate to excellent reliability (iMVF, CV 3.4%, ICC 0.97; 1RM, CV 3.5%, ICC 0.98; Explosive force, 50 ms, CV 14.6%, ICC 0.85; 100 ms, CV 4.1%, ICC 0.97; 150 ms, CV 3.5%, ICC 0.97). Additionally, muscle thickness measurements taken in duplicate before training were very consistent (paired t-test, P = 0.819) and showed high levels of reliability (CV 2.5%, ICC 0.94). There were no changes in EMG amplitude recorded during the reference tasks between the pre-2 and post-training measurement sessions: M_{max} amplitude (pre, 11.6 ± 1.3 vs. post, 12.2 ± 1.3 mV, paired t-test, P = 0.280); TB EMG_{max} during elbow extension (pre, 0.27 ± 0.03 vs. post 0.25 ± 0.03 mV; paired t-test, P = 0.130); AD and PM EMG_{max} during bench press (AD pre, 0.65 ± 0.04 vs. post, 0.62 ± 0.05; PM, pre, 0.50 ± 0.13 vs. post, 0.35 ± 0.04 mV; both, paired t-test, P ≥ 0.148).

7.3.2 Maximum Isometric Strength and 1RM

Elbow flexor iMVF increased from 240 ± 7 to 258 ± 8 N (paired t-test, P < 0.001, ES = 0.33) and 1RM lifting strength increased from 10.9 ± 0.4 to 12.6 ± 0.4 kg (paired t-test, P < 0.001, ES = 0.57) after three weeks of RT, with a greater percentage increase of 1RM than iMVF (17.0 ± 2.0 vs. 7.4 ± 1.4% , paired t-test, P < 0.001, Figure 7.2).
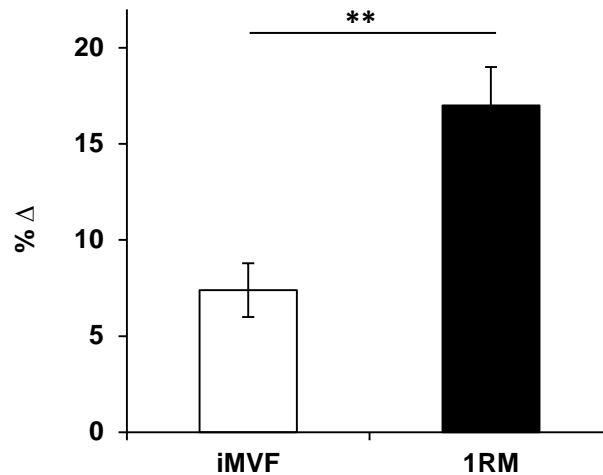
Figure 7.2 Percentage change in iMVF and 1RM following three weeks of resistance training. (Mean ± SEM, N = 45). ** indicates a significantly greater change in 1RM than isometric MVF (paired t-test, P < 0.001).

There was a 15.7% increase in agonist EMG amplitude during the 1RM (paired t-test, P < 0.001, ES = 0.44), with no change in agonist EMG at iMVF showed no changes after the RT (paired t-test, P ≥ 0.541; Figure 7.3A). The increase in agonist EMG amplitude was greater for the 1RM than MVF (ANOVA, task x training P = 0.005, respectively). Agonist EMG amplitude was also 32.6% higher during the 1RM than iMVF pre-training and 57.3% higher post-training (both, paired t-test, P < 0.001).

Whilst, antagonist EMG during 1RM increased by 26.2% (paired t-test, P = 0.032, ES = 0.35) and co-activation at iMVF was unchanged (paired t-test, P = 0.701, Figure 7.3B), there was no difference between the changes in 1RM and iMVF (ANOVA, task x training, P = 0.143). Antagonist EMG was 52.9% higher during the 1RM than at iMVF pre-training and 84.9% higher post-training (both, paired t-test, P ≤ 0.001).

Stabiliser EMG also increased during the 1RM (43.2%, paired t-test, P < 0.001, ES = 0.59, Figure 7.3C) and this reflected an increase for both the AD and PM (AD, pre, 56.0 ± 6.7 vs. post, 82.0 ± 6.5% EMG_{max}; PM, pre, 56.0 ± 5.3 vs. 80.7 ± 8.8% EMG_{max}, both, paired t-test, P ≤ 0.002). Similarly, at iMVF overall stabiliser EMG (+53.1%, paired, t-test, P < 0.001, ES = 0.84) and that of each of the stabiliser muscles was elevated post-training (AD, pre, 26.7 ± 2.2 vs. post, 44.7 ± 3.9; PM, pre 41.5 ± 3.8 vs. post, 60.8 ± 4.6% EMG_{max}, both, paired t-test, P < 0.001). The training-induced changes in stabiliser EMG were similar for 1RM and MVF

(ANOVA, task x training, P $\geq$ 0.297). Stabiliser EMG was 65.4% higher during the 1RM than at iMVF pre-training and 50.6% higher post-training (both, paired t-test, P $\leq$ 0.001).
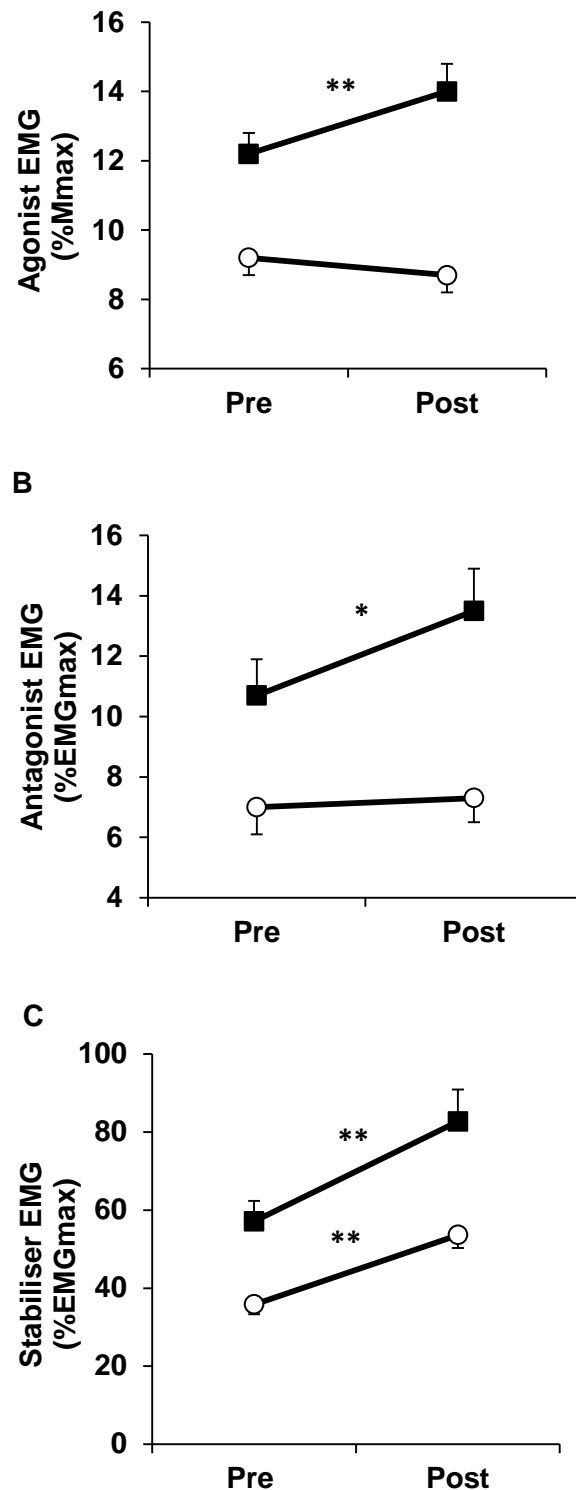
Figure 7.3 Agonist (A), antagonist (B) and stabiliser (C) muscle EMG amplitude measured at isometric maximum voluntary force (open circles) and during the 1RM (filled squares). Data are reported as mean $\pm$ SEM (N= 45, [N = 39 for agonist EMG normalised to M_{max}]). A training effect is denoted by * (P < 0.05), ** (P < 0.001).

7.3.3 Explosive Isometric Contractions

During the explosive contractions there was no training effect on absolute force production at any time point (ANOVA, training, P = 0.595, Figure 7.4A), peak RFD (pre, 3224 ± 108.4 vs. post, 3210 ± 124.3 N.s^{-1}; paired t-test, P = 0.853) or the force-time integral produced over any of the time periods (ANOVA, training, P = 0.495). However, there was a training effect for explosive force production normalised to MVF (ANOVA, training, P = 0.008), with a significant decrease in % MVF achieved at 50 ms (-16.5%, ES = 0.47, Bonferroni, P = 0.003), but no change at 100 (ES = 0.29, Bonferroni, P = 0.123), or 150 ms (Bonferroni, P = 0.735; Figure 7.4B). Similarly, there was a decrease in relative peak RFD (pre, 13.5 ± 0.3 vs. post, 12.6 ± 0.4 MVF.s^{-1}, paired t-test, P = 0.005, ES = 0.39), and an increase in the time to reach peak RFD (pre, 57.2 ± 2.3 vs. post, 63.6 ± 2.3 ms, paired t-test, P = 0.004, ES = 0.45). Voluntary EMD$_{max}$ remained unchanged after training (pre, 25.1 ± 0.8 vs. post, 24.5 ± 0.9 ms, paired t-test, P = 0.417).
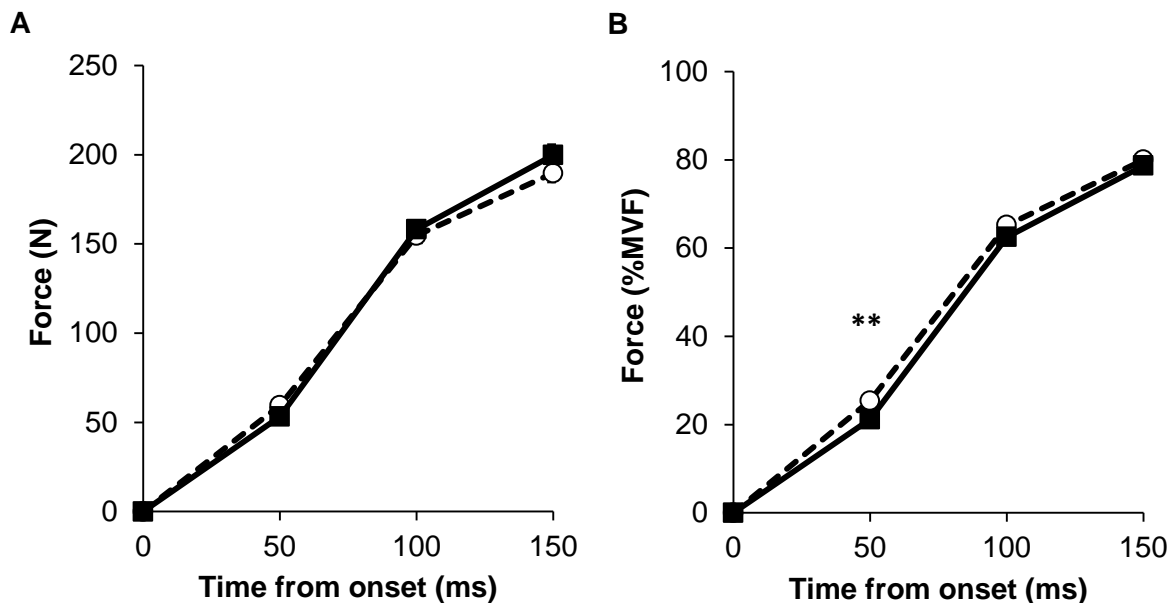
Figure 7.4 Absolute (A) and relative (B) force (relative to maximum voluntary force; MVF) during explosive isometric contractions of the elbow flexors pre (bold line, filled squares) and post (dashed line, open circles) training. Data are mean ± SEM for the group (N = 45). A training effect is denoted by ** (P < 0.01).

The training did not influence the EMG amplitude during the explosive contractions for either the agonist (ANOVA, training, P = 0.133, Figure 7.5A) or antagonist muscles (ANOVA, training, P = 0.682; Figure 7.5B), but there was an increase in stabiliser muscle

activation (ANOVA, training, P < 0.001, Figure 7.5C) with significant increases over 0-50 (45.3%, Bonferroni, P = 0.003, ES = 0.47) and 0-150 ms (26.2%, Bonferroni, P < 0.001, ES = 0.60), but not 0-100 ms (Bonferroni, P = 0.201, ES = 0.28).
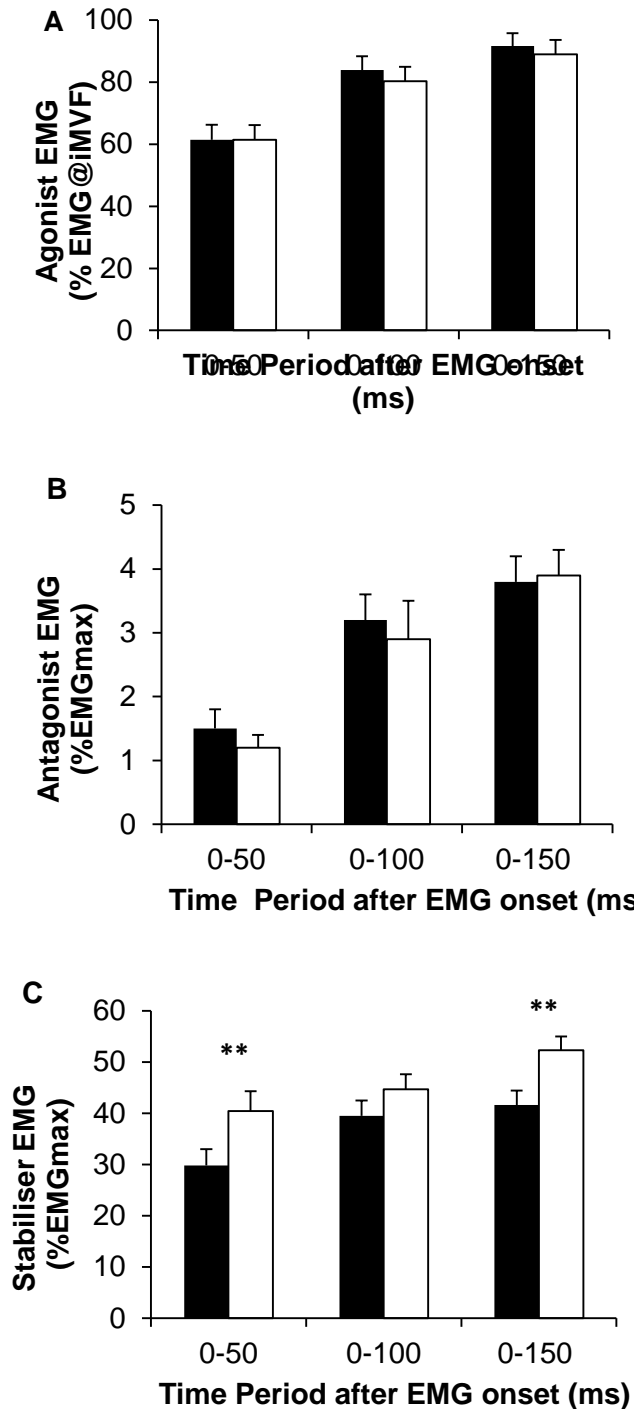
Figure 7.5 Agonist (A), antagonist (B) and stabiliser (C) muscle EMG amplitude during explosive isometric contractions of the elbow flexors pre (black) and post (white) training. Date are reported as mean ± SEM for the group (N = 45). A training effect is denoted by * (P ≤ 0.05), ** (P ≤ 0.001).

7.3.4 Muscle Thickness

Thickness of the biceps brachii short-head increased from 15.0 ± 0.5 to 15.7 ± 0.6 mm (paired t-test, P = 0.003, ES = 0.21) following training, representing a change of 5.3 ± 1.4 %.

7.4 Discussion

This study investigated the changes in elbow flexion strength tasks (isoinertial, and isometric maximum and explosive strength), and evaluated the adaptations in agonist, antagonist and stabiliser neuromuscular activation that may contribute to improved strength following 3-wk RT. It was hypothesised that conventional isoinertial RT would induce greater increases in isoinertial, than isometric strength, and that this would be concomitant with greater changes in agonist and stabiliser muscle activation during the isoinertial task. After training there was more than a two-fold greater increase in training task specific isoinertial than isometric strength (17 vs. 7%) that appeared to be due to task specific neural adaptations during the 1RM. Specifically, task-specific adaptations in agonist EMG during the 1RM (+16%) with no change in agonist EMG at iMVF. A novel finding of this study was that training increased stabiliser muscle activation during all elbow flexion strength tasks, but with no task specific training effects. After training there was no change in absolute explosive force production, but there was a decrease in relative early phase explosive force production.

This study included a large cohort of participants (N = 45) and demonstrated good to excellent reliability of the strength and muscle thickness measurements. Furthermore, there was no change in any of the EMG reference measures following training. Normalisation of agonist, antagonist and stabiliser EMG during the elbow flexion contractions to these reference measurements would be expected to reduce measurement variability and enhance the statistical power of the experiment (Buckthorpe et al. 2012). These methodological strengths of the study provide confidence that the changes in performance and underlying physiological mechanisms are adaptations as a result of the short-term training.

We observed a 17% increase in the 1RM following RT which is equivalent to the changes observed in another upper body RT study [chest press 1RM 17% increase after 4 weeks, (Abe et al., 2000)]. This increase in 1RM was more than two-fold greater than the 7% increase in iMVF and this differential response is similar in magnitude to previous longer duration

isoinertial RT studies [18 vs. 40% increase after 9 weeks, (Folland et al., 2002); 16 vs. 40% after 12 weeks (Hubal et al., 2005)]. This is the first study to provide strong evidence for neural mechanisms explaining this tasks specificity phenomenon. The task specific increase in agonist activation during the 1RM (no change in agonist EMG at iMVF) provides a ready explanation for the greater increase in isoinertial vs. isometric strength. Previous research has reported an increase in absolute agonist EMG during the 1RM after 12 weeks of RT [knee extensors, (Hakkinen et al., 1998)], but within the present study we also used the more robust method of normalised EMG (to M_{max} +16%) and found strong evidence that increased agonist activation contributes to isoinertial strength gains after RT. Considering isometric strength, evidence for changes in normalised agonist EMG (to M_{max}) at iMVF following RT is equivocal (Van Cutsem et al., 1998; Pucci et al., 2006; Tillin et al., 2011). The conflicting findings may relate to the muscle group investigated, as Behm and colleagues (2002) found agonist activation at iMVF to vary between muscle groups for untrained participants. Using the interpolated twitch technique (ITT), activation of the elbow flexors even in untrained participants has been reported to be very high (> 98%, (Allen et al., 1998)), which may explain why training did not increase agonist activation at iMVF in the current study.

Absolute agonist EMG during the 1RM was higher than at iMVF (pre, + 33%; post +57%), which might indicate a higher level of muscle activation during the 1RM or simply reflect methodological differences between these measurements. As discussed above, elbow flexor activation at iMVF assessed with the ITT has been reported to be very high/maximal. Therefore, if the ITT and EMG are valid measures of voluntary activation, it does not seem plausible for EMG during the 1RM to be over 50% higher than EMG at iMVF. On the other hand, the validity of the ITT for providing a quantifiable measurement of agonist activation at iMVF is controversial (e.g. de Hann et al., 2009). Elbow flexor iMVF was recorded at 60° elbow flexion, whereas peak EMG during the 1RM typically occurred at a more extended joint angle, i.e. ~10-50°. There was also a subtle difference in shoulder flexion angle between the two tasks (1RM 75° vs. iMVF 90°). Joint angle has been found to influence the amplitude of volitional EMG (Kasprisin & Grabiner, 2000) and thus angle specific differences in both shoulder and elbow angles could explain the task specific discrepancy in EMG amplitude we have found. Despite the issues surrounding the quantification of voluntary muscle activation, the present results suggest that the greater gains in isoinertial lifting strength were explained by task specific adaptations in agonist neuromuscular activation.

The consistency of antagonist co-activation values pre and post training at iMVF is in accordance with two previous studies (Hakkinen et al., 1998; Pucci et al., 2006), but conflicts with studies that have reported both increased (Simoneau et al., 2006; de Boer et al., 2007) and decreased (Hakkinen et al., 1998; Tillin et al., 2011) co-activation following RT. Tillin et al. (2011) recently found increased co-activation at iMVF following four weeks of RT, but a reduction in antagonist activation expressed as a ratio of agonist activation, across a range of contraction intensities up to iMVF post-training. It may be that in the present study the increase in iMVF was not large enough to require an increase in co-activation to maintain joint integrity, or that the elbow joint is sufficiently stable at iMVF not to require increased co-activation as strength increases. There was however a large increase in co-activation during the 1RM following RT (26%), which may have attenuated the overall gains in isoinertial strength, and might reflect a greater need to stabilise the elbow joint during the 1RM (Cochrane et al., 2006, Tillin et al., 2011). There was no change in antagonist EMG during the explosive contractions, which may be due to the consistent force and agonist EMG of these contractions after training.

Stabiliser muscle activation increased by a similar extent during both the 1RM and at iMVF (39 vs. 53%, respectively) and is a novel finding, which likely contributed to changes in strength during both tasks. The effective stabilisation of joints is thought to be important for optimal force production (Folland & Williams, 2007a), and the adaptation in stabiliser activation that was observed may help to explain the commonly observed discrepancy between muscle size and iMVF changes following RT (e.g. Narici et al., 1996). It was hypothesised that the greater increase in isoinertial, than isometric strength, would be concomitant with greater changes in stabiliser muscle activation during the isoinertial task. Whilst there was no task specific training effect on stabiliser activation, it is possible that increased stabiliser activation was of greater consequence during the 1RM that for iMVF. During isoinertial elbow flexion more movement is available at the shoulder than is the case during the isometric measurements where force production is constrained to the elbow joint by the apparatus and strapping. Therefore the elbow flexion 1RM may be more responsive to synergistic contributions from shoulder joint muscles or more effective stabilisation of the shoulder during this task. Thus the 1RM could be more responsive to enhanced stabiliser activation after training. Furthermore, increased stabiliser activation and shoulder joint stabilisation during the 1RM after RT may have facilitated an increase in agonist activation, particularly for the bi-articular bicep brachii. Thus the 1RM could be more responsive to

enhanced stabiliser activation after training. It would be interesting for future work to consider the response of a wider range of stabiliser muscles after training. The 5% increase in muscle thickness we observed indicates the occurrence of hypertrophy after only three weeks of RT, and is in agreement with an earlier study that also found hypertrophy after only three weeks of training [knee extensors, + 3.5-5.2%, (Seynnes et al., 2007)]. Therefore, this study supports the notion that skeletal muscle hypertrophy can occur during the initial 3 weeks, or 9 sessions, of training, and might be expected to have made a greater relative contribution to the observed gains in isometric than isoinertial strength.

There was no change in absolute force and a decrease in early phase relative force production (50 ms) during the explosive voluntary contractions following RT, which further questions the efficacy of RT for enhancing explosive strength (Andersen et al., 2010, Tillin et al., 2011). There is some evidence that including an explosive strength component to RT (i.e., intending to lift the weight as quickly as possible) is sufficient to enhance early phase explosive strength and agonist neural drive (Behm & Sale, 1993; Van Cutsem et al., 1998; Del Balso & Cafarelli, 2007). However, during conventional isoinertial RT, the continuous, cyclic nature of the repetitions, with gradual controlled lowering (eccentric) immediately followed by lifting (concentric), may involve high levels of activation and force throughout each set. Therefore, even if attempting to perform the concentric phase of the lift as quickly as possible (as was the case in the present study) there may be no transition from low to high levels of activation/force that is required to enhance explosive force production. Alternatively, it is possible that adaptations in agonist neural drive and explosive force production with the training task may have been specific to the early concentric phase of the lift, i.e. at more extended joint angles than the angle used to measure isometric strength. Future investigation into angle-specific changes in explosive strength and associated mechanisms following isoinertial maximum and/or explosive strength training would have strong implications for athletic training.

The large increase in stabiliser activation during the explosive contractions in the absence of any changes in absolute force further supports the findings of chapter 6, that the level of stabiliser activation does not exert a direct influence on explosive force production. Further research is required on the influence of stabiliser activation on explosive strength during isoinertial strength tasks.

In summary, task specific neural adaptations, particularly increased agonist activation during the 1RM, appeared to explain the greater increase in isoinertial than isometric strength. Increased antagonist co-activation during the 1RM was the likely result of an increased load lifted and may be a protective mechanism to maintain joint integrity. Changes in iMVF were thought to be explained by increased muscle size and stabiliser muscle activation rather than changes in agonist or antagonist muscle activation. Despite participants attempting to lift the weight as quickly as possible, three weeks of RT resulted in no change in absolute explosive force production and a decrease in relative early phase explosive force production, which questions the efficacy of conventional isoinertial RT for enhancing explosive strength.

CHAPTER 8

Central fatigue contributes to the greater reductions in explosive than maximum strength with high intensity fatigue

8.1 Introduction

Repeated high force contractions of skeletal muscle causes a decline in the force generating capacity, referred to as muscle fatigue, that negatively influences the performance of explosive sporting actions (Mohr et al., 2003; Krustrup et al., 2006) and is thought to be an important risk factor for sports injuries (Hawkins et al., 2001). Much of the research investigating the influence of fatigue on the functional capacity of the neuromuscular system has focused on the decline in maximum voluntary force (MVF). However, the ability to develop force rapidly, termed explosive strength, is considered functionally more important than MVF during explosive movements, such as sprinting, jumping or restabilising the body following a loss of balance (de Ruiter et al., 1999; Aagaard et al., 2002a; Tillin et al., 2010). Therefore, an understanding of how fatigue affects explosive strength would seem important in understanding its influence on athletic performance and injury risk. There is however, a paucity of research investigating the influence of fatigue on voluntary explosive strength with no documented mechanistic evidence. Furthermore as the determinants of explosive force production appear to change throughout the rising force-time curve (Andersen & Aagaard, 2006; Tillin et al., 2010) fatigue may differentially affect the development of force throughout the time course of an explosive contraction.

The underlying mechanisms of muscle fatigue can be broadly separated into central and peripheral components. Peripheral fatigue is defined as the loss of force caused by processes occurring distal to the neuromuscular junction, and is thought to be the main contributor to muscle fatigue during high-intensity exercise (for a review see Allen et al., 2008). The influence of fatigue on the contractile properties of the muscle tendon unit (MTU) has typically been quantified with evoked twitch contractions. However, a muscles' maximal capacity for rate of force development (RFD) can only be achieved at high stimulation frequencies (Buller & Lewis, 1965; de Ruiter et al., 1999) such as an evoked octet (typically 8 pulses at 300 Hz) in human skeletal muscle (de Ruiter et al., 1999, 2007). The influence of fatigue on the response to an evoked octet, and thus the intrinsic contractile capacity for explosive force production and underlying peripheral fatigue mechanisms, has not been investigated. Type II skeletal muscle fibres have a substantially higher RFD (Brenner et al. 1986; Metzger & Moss, 1990; Harridge et al., 1996), but arguably similar specific tension (isometric peak force/cross-sectional area; (Larsson & Moss, 1993; Gilliver et al., 2009)) to

type I fibres. Given the lower fatigue resistance of the type II fibres, a greater influence of fatigue on explosive than maximal phases of the evoked contraction could be expected.

Central fatigue can be described as a progressive exercise-induced reduction in voluntary activation or neural drive to the muscle (Taylor et al., 2006). The contribution of central fatigue to the decline in MVF has been studied extensively (for a review see Gandevia, 2001), and found to contribute up to ~20-25% of the decrease in MVF (Taylor et al., 2006). However, the contribution of central factors to the decline in explosive force with fatigue is unknown. A reduction in neural activation could be due to a decline in motor recruitment or motor unit firing frequency (MUFF). As the MUFFs during the explosive (rising) phase of force production appears to exceed the MUFF during the plateau phase of contraction which includes MVF (explosive phase, 100-200 Hz vs. plateau phase, 30-50 Hz, Monster & Chan, 1977; Kukulka & Clammann, 1981; Van Cutsem et al., 1998), a decline in MUFF with fatigue could be expected to exert a more pronounced effect on explosive than maximum force production. Differential changes in neuromuscular activation during explosive and MVF production can be examined via measurement of surface EMG amplitude (normalised to a maximum compound action potential, M_{max} to control for any changes in neuromuscular junction and sarcolemma excitation). Furthermore, reporting voluntary force achieved after 50 ms as a percentage of octet force after 50 ms may give an additional index of the voluntary ability to utilise the available contractile capacity (de Ruiter et al., 2004; Buckthorpe et al., 2012), and hence volitional neural drive to the muscle during the early phase of an explosive contraction.

The aim of this study was to carefully document the influence of a fatiguing exercise protocol on the development of explosive force throughout the rising force-time curve, and contrast these changes with the influence of fatigue on MVF. The neural (EMG, voluntary/octet force) and contractile (evoked twitch and octet) contributions to impaired performance were assessed to determine the mechanisms for any functional changes. It was hypothesised that explosive force production would exhibit a greater decline than MVF with fatigue, and that this might be due to neural and/or contractile mechanisms.

8.2 Method

8.2.1 Participants

Eleven healthy male participants (mean ± SD: age, 24 ± 4 yr; height, 1.69 ± 0.03 m; body mass, 77.1 ± 6.8 kg) completed the study. This sample size is in accordance with previous studies on neuromuscular fatigue using similar measurement techniques (Matkowski *et al.* 2011; Maffiuletti *et al*. 2013; Minshull & James, 2013). The participants provided written informed consent prior to their participation in this study that was approved by the Ethical Advisory Committee at Loughborough University to the standards set by the Declaration of Helsinki. The participants were recreationally active (up to three activity sessions per week), but not involved in any systematic physical training during the preceding 12 months.

8.2.2 Overview

Each participant attended the laboratory on two separate occasions, once for familiarisation and then for a main trial one week later. The main session involved isometric measurements of force and surface EMG from the dominant limb during a series of explosive maximum voluntary contractions (explosive MVCs) and evoked (twitch and octet) contractions of the knee extensors during a fatiguing protocol. Limb dominance was assessed according to de Ruiter et al. (2010). The fatigue protocol comprised 10 sets of five 3-s explosive MVCs. These volitional contractions were used to assess voluntary force production and induce fatigue. Force and EMG measurements were obtained from the explosive (rising) and maximal (plateau) phases of the explosive MVCs. The EMG measures were used to quantify changes in neural drive with fatigue. In between each set of volitional contractions twitch and octet contractions were evoked via electrical stimulation to document the intrinsic contractile changes that occurred with fatigue. Familiarisation involved participants practicing the explosive MVCs, experiencing the evoked twitch and octet contractions and performing a single set of the fatigue protocol.

8.2.3 Measurements

Participants were firmly secured in a strength testing chair with straps across the pelvis and shoulders to minimise extraneous movement. The hip and knee angles were fixed at 100 and 120° (full extension = 180°), respectively. An ankle strap was placed 2 cm proximal to the medial malleolus in series with an S-Beam tension/compression load cell (linear response up to 1500 N, Force Logic UK, Berkshire, UK) positioned perpendicular to tibial alignment. The force signal was amplified (x500) and interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK) and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). Real-time biofeedback of the force response was provided on a computer monitor. During off-line analysis the force signals were notch filtered at 50 Hz (to remove mains harmonics) and low pass filtered at 500 Hz using a fourth order zero-lag Butterworth digital filter.

The femoral nerve was electrically stimulated (via a constant current, variable voltage stimulator; DS7AH, Digitimer Ltd., UK) with square wave pulses (0.2 ms in duration) to elicit i) single twitch contractions and ii) octet contractions (8 pulses at 300 Hz) to determine the MTUs maximal capacity for explosive force production. The anode (carbon rubber electrode, 7 x 10 cm; Electro-Medical Supplies, Greenham, UK) was taped to the skin over the greater trochanter. The cathode, a custom-adapted stimulation probe 1 cm in diameter (Electro-Medical Supplies, Wantage, UK) which protruded 2 cm perpendicular from the centre of a plastic base (4 x 5 cm), was taped to the skin over the femoral nerve in the femoral triangle. The precise location of the cathode was determined as the position which elicited the greatest twitch response for a particular submaximal current.

Surface EMG was recorded from the superficial quadriceps [rectus femoris (RF), vastus lateralis (VL) and vastus medialis (VM)] of the participants' dominant limb using two Delsys Bagnoli-4 EMG systems (Delsys, Boston, USA). To improve the reliability of the EMG signal two EMG electrodes were placed over the surface of each of the superficial quadriceps. Following preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol), double differential electrodes (1 cm inter-electrode distance, DE-3.1, Delsys) were attached to the skin using adhesive interfaces. To normalise the placement across individuals, the medial ($_M$) and lateral ($_L$) EMG electrodes on each muscle were positioned at specific distances along the thigh (VM_M, 20%, VM_L, 30%; VL_M, 45%; VL_L, 55%; RF_M, 55%; RF_L, 65% from the lateral epicondyle of the femur to the greater trochanter), with 1 cm of medio-lateral separation (0.5 cm either side of mid-muscle belly), and in parallel to the presumed orientation of the muscle fibers. The reference electrode was placed on the patella of the same

limb. EMG signals were amplified (x1000; differential amplifier, 20 – 450 Hz) and synchronised with force data by recording at 2000 Hz with the same analogue to digital converter and PC as the force signal. During off-line analysis the EMG signals were band-pass filtered between 6 and 500 Hz using a 4th order zero-lag Butterworth digital filter.

8.2.4 Protocol

Following preparation of the skin for EMG placement, participant's performed a standardised warm up on a cycle ergometer (10 minutes at 1.33 W.kg^{-1} body mass, Monark Ergomedic 874E). Following this the skin was re-cleaned and EMG electrodes attached. Once the participants were firmly secured in the testing chair they performed a series of submaximal voluntary contractions of the knee extensors. Participants then performed 2-3 short explosive contractions to re-familiarise themselves with the explosive MVCs prior to the main protocol. Next a series of incremental twitch contractions were elicited until there was a simultaneous plateau in the force and M-wave response of the agonists. A supramaximal current of ≥125 % of this level was used to evoke twitch and octet contractions during the fatigue protocol. Participants were then briefly re-familiarised with the octet contractions prior to the commencement of the fatigue protocol.

The fatigue protocol comprised 10 sets of five explosive MVCs, each lasting 3-s separated by 2-s rest (see Figure 8.1). This protocol is similar to previously adopted using voluntary maximal contractions (Zhou et al., 1996). In response to an audio and visual signal participants were instructed to push as fast and hard as possible and maintain this throughout the contraction for 3-s (Aagaard et al., 2002a). Participants were provided with strong verbal encouragement and instructed to avoid countermovement or pre-tension. Following each explosive MVC they were instructed to relax quickly in order to return to the resting baseline force. To provide participants with feedback on the occurrence of any countermovement, the resting force level was displayed on a sensitive scale. The slope of the force time curve (10 ms time constant) and the maximal force achieved were displayed throughout to provide feedback on the explosive and maximal phases of each contraction. Five seconds separated each set of volitional contractions and during this time twitch (2-s after voluntary contractions) and octet (1-s after twitch contraction) contractions were evoked. The total

duration of the fatigue protocol was five minutes, including ~150 s of maximal voluntary effort.

Five, 3-s maximal explosive isometric contractions separated by 2-s rest

Electrically-evoked twitch contraction

Electrically-evoked octet contraction
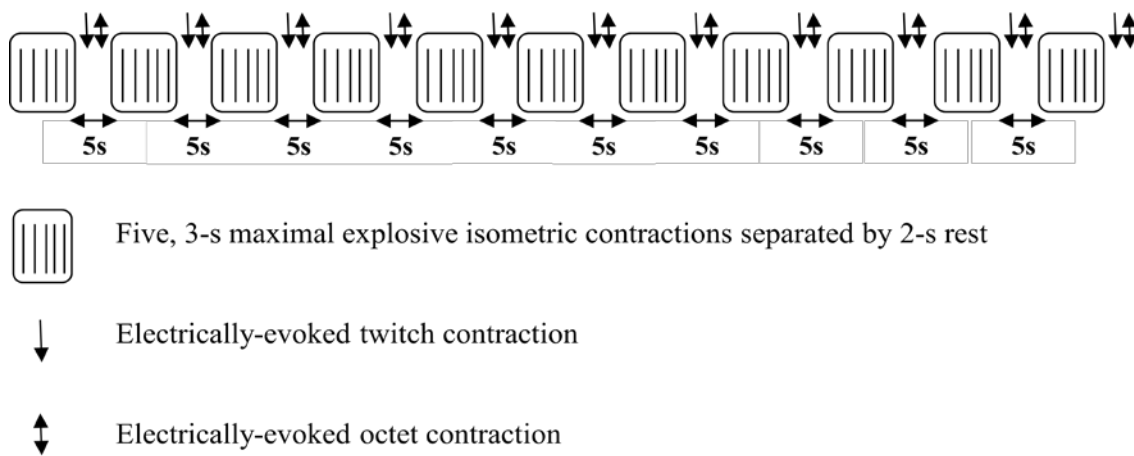
Figure 8.1 The exercise protocol used to induce fatigue and assess changes in neuromuscular function.
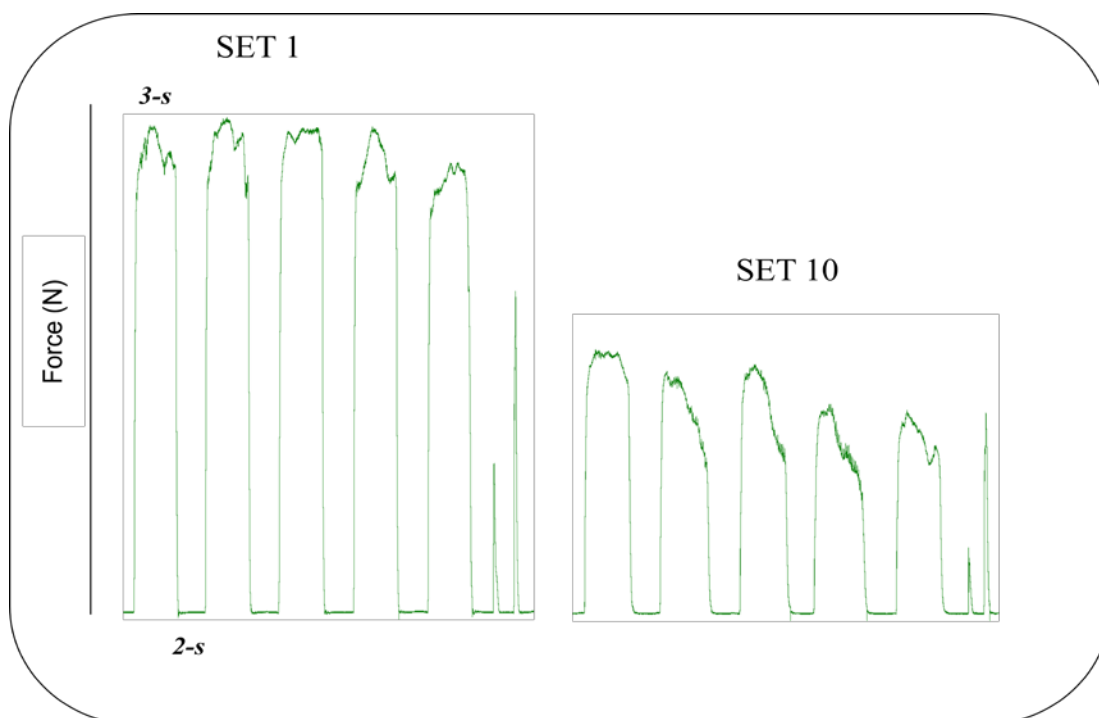
Figure 8.2 Presentation of actual force data from set 1 and set 10 of a single participant (to scale). The data shows the 5 MVCs, followed by a twitch and octet contraction.

8.2.5 Data Analysis

The three contractions in each set with the highest peak slope and no discernible countermovement or pre-tension (change in force of < 0.5 N in the preceding 100 ms) were used for analysis of explosive force measurements, which were averaged across these three contractions. Explosive force was measured at 50, 100, and 150 ms (defined as F_{50}, F_{100}, F_{150}), from force onset. Rate of force development (RFD, change in force divided by change in time) was quantified for consecutive 50 ms time periods (0-50, 50-100 and 100-150 ms). Peak RFD (pRFD) was measured as the maximum slope (10 ms time constant). Both force and RFD were measured in absolute terms (N and N.s^{-1}, respectively) and normalised to MVF (assessed within the same set of contractions, %MVF and MVF.s^{-1}, respectively). The three contractions within each set with the highest instantaneous force were averaged to determine knee extensor MVF.

The amplitude of the EMG signal was assessed as the root mean square (RMS) for each recording site (RF_M, RF_L, VM_M, VM_L, VL_M, and VL_L) during explosive (0-50, 0-100 and 0-150 ms from the onset of EMG activity, denoted as EMG_{0-50}, EMG_{0-100}, EMG_{0-150}) and maximal (500 ms epoch around MVF, 250 ms either side, EMG_{MVF}) aspects of the force time curve. EMG onset was defined as the onset of the first agonist muscle/site to be activated. Furthermore, volitional EMG amplitude values from each recording site (RF_M, RF_L, VM_M, VM_L, VL_M, VL_L) were expressed in relation to their respective maximal M-wave area (M_{max} Area; see below) in response to the evoked twitch contraction following that specific set of volitional contractions, and averaged across the six sites to provide a value for the quadriceps. The median frequency of each agonist EMG recording was calculated during the initial 150 ms after signal onset during the explosive phase of contraction (MF_{0-150}) and for the same 500 ms epoch associated with MVF (MF_{MVF}). Measurements were obtained at a frequency resolution of 7.8 Hz and averaged across sites to provide a mean knee extensor value. The time between the earliest EMG onset (agonist muscle activation) and the onset of force was determined as the maximum electromechanical delay (EMD_{max}).

Signal onsets of all voluntary and evoked contractions were visually identified (Allison, 2003; Moretti et al., 2003; Pain & Hibbs, 2007, Pulkovski et al., 2008). Details of this method and its reliability have previously been published (Tillin et al., 2010). Briefly, force and EMG recordings were initially viewed with consistent y-axis scales of 1 N and 0.1 mV, and an x-

axis scale of 500 ms. These scales provided sufficient resolution for the accurate discrimination of signal onset, which was defined as the last peak or trough before the signal deflected away from baseline noise. A vertical cursor was then placed on the onset and viewed at a higher resolution to determine its exact location (0.5 N and 0.05 mV for force and EMG axes, respectively using an x-axis of 25 ms).

Each twitch force response was analysed for pRFD (10 ms time constant), peak force (PF), time to achieve pRFD and PF after force onset, and half relaxation time (HRT). M_{max} Area was recorded as previously in our laboratory (Buckthorpe et al., 2012). Analysis of octet force included pRFD, F_{50}, PF, and time to achieve PF and pRFD. As an additional measure of overall neural efficacy (Neural Efficacy 0-50), voluntary F_{50} was compared to octet F_{50} to assess the participant's voluntary activation capacity over the initial 50 ms of the contraction (Hannah et al., 2012; Tillin et al., 2012) and was reported as voluntary percentage of octet performance. Both twitch and octet force and RFD were measured in absolute terms (N and $N.s^{-1}$, respectively) and normalised to their respective maximal values (i.e., %octet PF and octet $PF.s^{-1}$, respectively).

Data are reported as group mean ± standard deviation (SD). Primary analysis compared set 1 and 10. Absolute measures from these sets (iMVF and EMG at MVF, MF, pRFD and time to pRFD for the voluntary explosive, evoked twitch and octet contractions) were compared for significant differences using paired t-tests. Effect sizes (ES) were reported to quanify the magnitude of differences between measures and interpreted according to Cohen's d, where 0.2 is a small effect, 0.5, a moderate effect and > 0.8 a large effect. Time-series data during the explosive contractions (force and EMG) were assessed with two-way repeated measures ANOVA (exercise set (1, 10) x measurement time point/period (e.g. 50, 100, 150 ms)). If sphericity was violated then Huynh-Feldt corrected values were used. Significant main effects of exercise set were further investigated with post-hoc pair wise comparisons (Bonferroni corrected paired t-test) for specific time points/periods. Secondary data analysis included relative changes which were calculated as mean ± SD of individual percentage changes. In order to quantify and contrast voluntary force changes throughout the 10 sets of the exercise protocol an exponential of the form a·exp(b·x)+c was fitted to the relative change data for MVF and for F_{50}, F_{100}, F_{150}, pooled from all participants (N = 11), using the custom fitting tool in Matlab (The MathWorks Inc., Natick, MA, USA). Significant differences between the exponential curves for MVF and measures of explosive force (F_{50}, F_{100}, F_{150}) were assessed using the extra-sum-of-squares F-test as described in Motulsky and

Christopoulos (2004). One-way ANOVA was used to compare relative changes in voluntary and involuntary measures of pRFD (voluntary, twitch and octet) as well as different measures of neural drive (Neural Efficacy 0-50, EMG_{0-50}, EMG_{0-100}, EMG_{0-150}, EMG_{MVF}), with Bonferroni post-hoc comparison where required. Statistical analysis was performed using SPSS version 19 and statistical significance was set at $P < 0.05$.

8.3 Results

8.3.1 Voluntary Force

MVF declined throughout the exercise (see Figure 8.3) and was 42% lower by set 10 (paired t-test, $P < 0.001$, ES = 2.64, vs. Set 1). Explosive force during the initial 150 ms of contraction declined by 47-56% by set 10 (ES = 1.25-3.93, Figure 8.4A). Likewise absolute RFD declined by 39-56% across the three consecutive 50 ms time periods (ES = 1.25-2.49, Figure 8.4C). pRFD also declined (set 1, 12100 ± 2859 vs. set 10, 5322 ± 1745 $N.s^{-1}$; 56%, paired t-test, $P < 0.001$, ES = 2.37), but the time to voluntary pRFD remained unchanged (set 1, 54.8 ± 17.6 ms vs. set 10, 62.7 ± 17.1 ms, paired t-test, $P = 0.180$, ES = 0.45).

There was a reduction in normalised explosive force (to MVF) at 50 (29%, ES = 0.61), 100 (17%, ES = 0.85) and 150 ms (11%, ES = 1.07), with reductions in normalised RFD during the initial 50 ms (29%, $P = 0.038$, ES = 0.61) but no change for RFD_{50-100} or $RFD_{100-150}$ ($P \geq 0.178$, ES = 0.24-0.51, Figure 8.4D). Normalised pRFD was 26% lower at set 10 (set 1, 13.6 ± 3.5 vs. set 10, 10.0 ± 2.9 $MVF.s^{-1}$, paired t-test, $P = 0.002$, ES = 1.03). Explosive force measurements (F_{50}, F_{100} and F_{150}) all had a greater exponential decay, or a lower plateau than MVF (all, F-test, $P < 0.001$, Table 8.1).
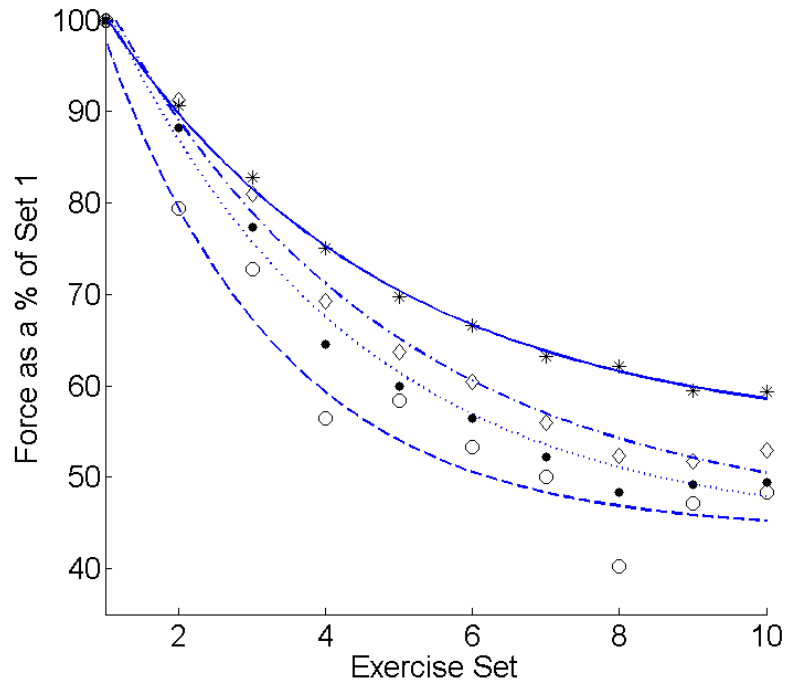
Figure 8.3 The decline in MVF (solid line, stars), and explosive force at 50 (dashed line, open circles), 100 (dotted line, black circles) and 150 ms (dashed and dotted line, open triangles) after force onset over the course of the fatigue protocol. Data are reported as mean individual percentage changes in relation to set 1. (N = 11). Force changes with set/time are fitted with an exponential of the form: $a \cdot \exp(b \cdot x) + c$.

Table 8.1 Mean half-life and goodness of fit (r^2) evaluated from the exponential fits to the group force response data across the 10 sets of contractions. The values are presented for force at 50, 100 and 150 ms and for MVF.

	Equation	**Half life (sets)**	**r^2**
F_{50}	$= 55.7 \cdot \exp(-0.394 \cdot x) + 43.8$	1.76	0.963
F_{100}	$= 57.8 \cdot \exp(-0.307 \cdot x) + 43.7$	2.26	0.991
F_{150}	$= 57.0 \cdot \exp(-0.263 \cdot x) + 45.0$	2.64	0.989
MVF	$= 46.6 \cdot \exp(-0.262 \cdot x) + 54.2$	2.65	0.997

F_{50}, force at 50 ms; F_{100}, force at 100 ms; F_{150}, force at 150 ms; MVF, maximum voluntary force; exp, exponential.
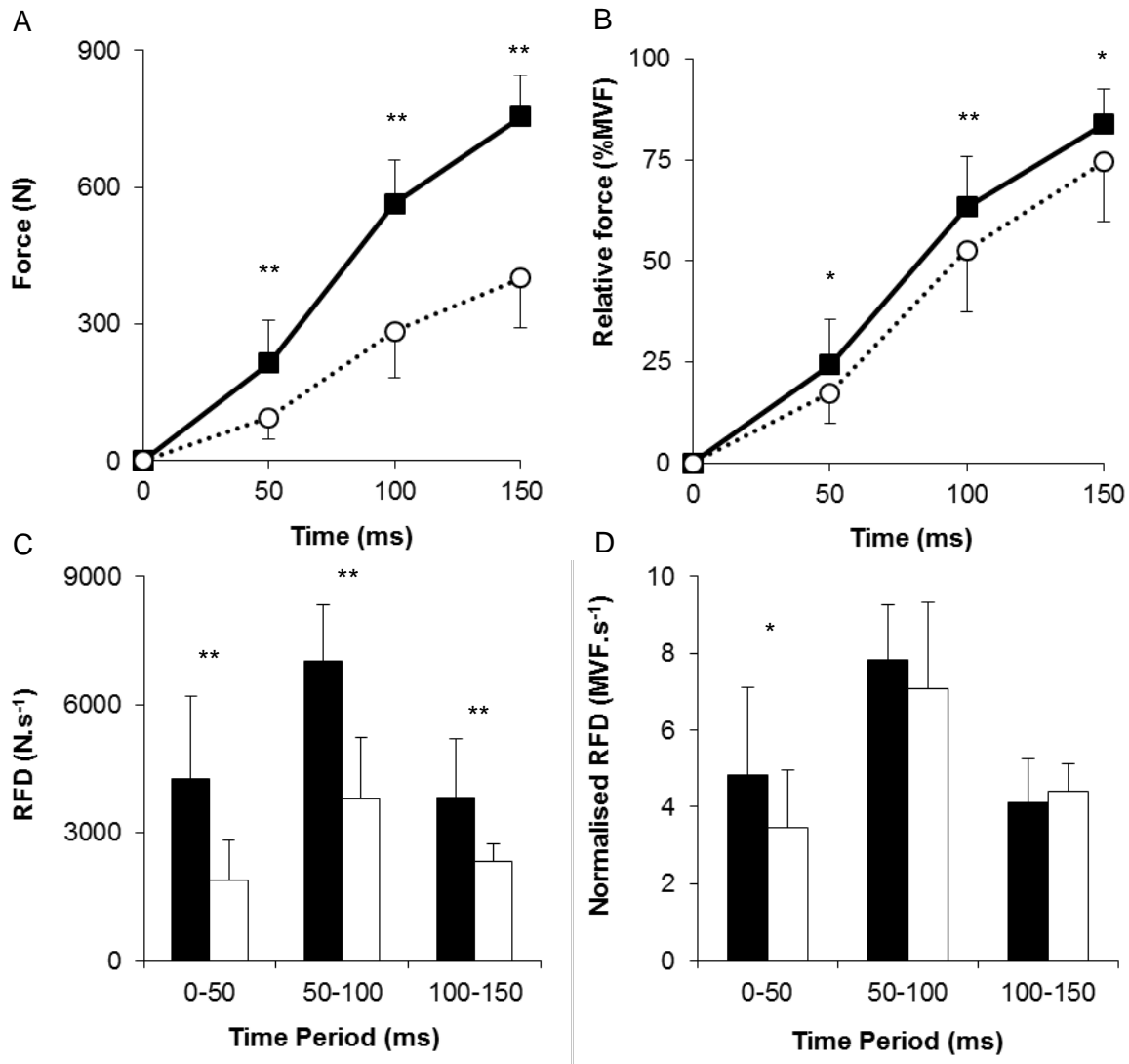
Figure 8.4. Absolute and normalised (to MVF) force (A and B) and rate of force development (C and D) at set 1 (bold line, filled squares/black bars) and set 10 (dashed line, open circles/white bars) during the initial 150 ms of isometric explosive MVCs of the knee extensors. Data are mean ± SD for the group (N = 11). Significant Bonferroni post-hoc comparisons are denoted by * (P < 0.05), ** (P < 0.01).

8.3.2 Intrinsic contractile properties

There was no change in M_{max} Area throughout the fatigue protocol (Table 8.2). At the end of the fatigue protocol (set 10) octet PF, F_{50} and pRFD were all lower than set 1 (23-28%, ES = 0.63-0.73, Table 8.2). There was no change in normalised octet pRFD (Table 8.2), and actually an increase in normalised octet F_{50} (7.2%, ES = 0.78). Twitch PF and pRFD both declined with no change in the time taken to reach PF or pRFD (Table 8.2). However, there was a decrease in normalised twitch pRFD (-20.8%, paired t-test, P = 0.001, ES = 1.53).

Differential changes were observed between voluntary and evoked twitch and octet pRFD (ANOVA, P < 0.001), specifically, voluntary (-55.4 ± 13.8%) and twitch (-65.6 ± 18.3%) pRFD both declined more substantially than octet pRFD (Octet, -26.7 ± 20.8%, both, Bonferroni, P < 0.01).

Table 8.2 Evoked force and time characteristics in response to twitch and octet stimulation before (set 1) and after (set 10) fatigue. Group data are reported as mean ± SD (N =11).

	Set 1	Set 10	P value	Effect size
OCTET				
PF (N)	595 ± 230	427 ± 180	**< 0.001**	0.73
pRFD (N.s^{-1})	16701 ± 6256	12291 ± 5971	**0.004**	0.70
Normalised pRFD (PF.s^{-1})	28.4 ± 3.3	28.6 ± 3.6	0.804	0.06
F_{50} (N)	394 ± 145	303 ± 122	**< 0.001**	0.63
Normalised F_{50} (%PF)	66.9 ± 5.8	71.4 ± 4.9	**0.003**	0.78
TWITCH				
Force				
PF (N)	301 ± 55	124 ± 47	**< 0.001**	3.21
pRFD (N)	11645 ± 2271	3981 ± 2316	**< 0.001**	3.37
Normalised pRFD (PF.s^{-1})	39.0 ± 5.3	30.9 ± 5.2	**0.001**	1.53
EMG				
M_{max} Area (mV.s)	0.013 ± 0.005	0.014 ± 0.005	0.319	0.20

PF, peak force; N, newtons; pRFD, peak rate of force development; HRT, half relaxation time; M_{max} Area, Area of evoked maximum compound action potential; F50, force at 50 ms after force onset; %Δ, percentage change from set 1; P value; paired t-test significance value set 1 versus set 10. Bold indicates significance level < 0.05.

8.3.3 Neuromuscular Activation

Neural Efficacy 0-50 declined by set 10 (set 1, 47.7 ± 20.7 vs. set 10, 29.5 ± 15.4 %, P = 0.002, ES = 0.88). Furthermore, explosive agonist EMG (all time periods, 20-30%, Bonferroni, P < 0.001, ES = 0.53-0.93, Figure 8.5A) and EMG$_{MVF}$ (15%, t-test, P < 0.001, ES = 0.57, Figure 8.5A) also declined by set 10. Although, the relative decline in measures of early phase neuromuscular activation appeared to be greater than EMG$_{MVF}$ (Neural Efficacy 0-50, 34.1 ± 22.5 and EMG$_{0-50}$, 28.1 ± 29.0 vs. EMG$_{MVF}$, 14.6 ± 11.9%, Figure 5.5B), there

was in fact no significant difference in the relative decline between different measures of neural drive (ANOVA, P = 0.250). By set 10 MF_{MVF} had decreased by 10.0% (108.9 ± 19.4 to 98.0 ± 18.5 Hz, t-test, P < 0.001, ES = 0.56) but MF_{0-150} showed only a tendency to decline (105.3 ± 16.5 to 99.0 ± 14.7 Hz, t-test, P = 0.073, ES = 0.38).
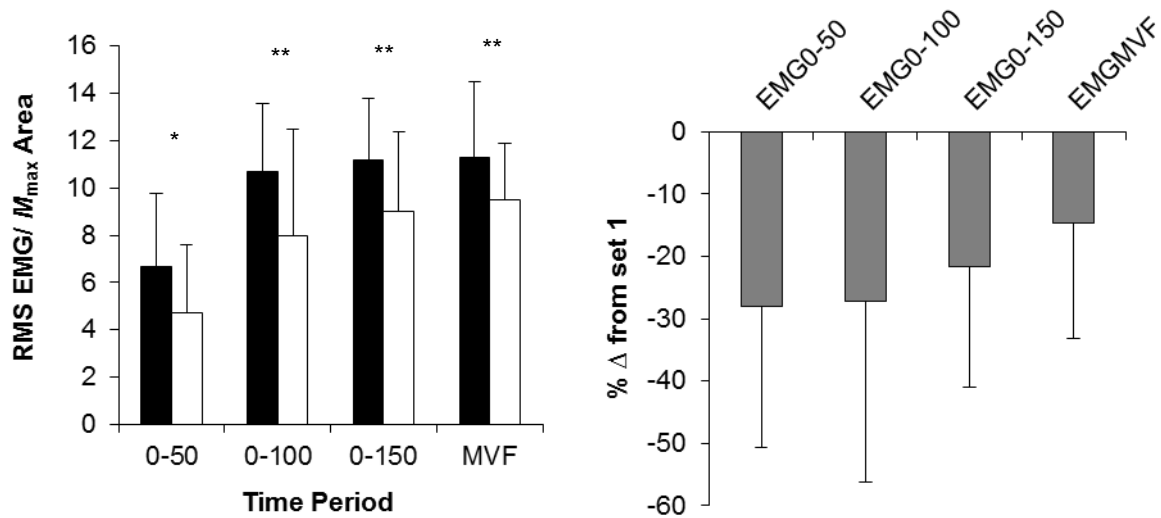
Figure 8.5. A) Agonist normalised EMG during set 1 (black) and set 10 (white) of isometric explosive MVCs of the knee extensors; B) Percentage change in different measures of neuromuscular activation (EMG; Neural Efficacy) at set 10 compared to set 1. EMG amplitude was measured as the root mean square of the EMG signal normalised to maximal M-wave area (M_{max} Area.s-1) during the explosive phase of contraction (0-50, 0-100, 0-150) and at MVF. Neural Efficacy (NE 0-50) was defined as voluntary force as a percentage of octet force at 50 ms. Data for reported as mean ± SD for group data (A) and individual percentage changes from set 1 (B). An effect of time is denoted by ** (P ≤ 0.001).

8.3.4. Electromechanical Delay

Voluntary EMD_{max} was not different at the end of the protocol (set 1, 20.8 ± 3.0 vs. set 10, 20.2 ± 5.2 ms, paired t-test, P = 0.775), but there was a 51% elongation of evoked EMD_{max} by set 10 (set 1, 6.5 ± 1.5 vs. set 10, 8.8 ± 1.4 ms, paired t-test, P < 0.001, ES = 1.53).

8.4 Discussion

The current study is the first to assess the influence of fatigue on explosive force production throughout the rising force-time curve. MVF and explosive voluntary force declined substantially (42%, 47 to 57% respectively), confirming that the protocol was sufficient to produce marked impairments in voluntary force. There was a reduced ability to express the available force generating capacity explosively, particularly during the early phase of contraction (normalised RFD_{0-50}, 29%) and this resulted in a lower normalised explosive force (to MVF) throughout the first 150 ms of contraction. The mechanistic measurements revealed evidence for neural fatigue during both the explosive and maximal phases of contraction. In addition there was demonstrable contractile fatigue with reduced explosive and peak octet (23-28%) and twitch force responses (59-66%). Although, normalised pRFD of the octet remained unchanged, it was reduced for the twitch after fatigue (21%).

Explosive force declined more rapidly and markedly than MVF throughout the fatigue protocol as evidenced by a shorter half-life or lower plateau for all explosive force curves versus MVF (Figure 8.2). Therefore, as hypothesised there was a more pronounced influence of fatigue on explosive than MVF throughout a range of fatigue levels. Additionally, fatigue caused lower normalised voluntary explosive force throughout the initial 150 ms of contraction (11-29%) and thus compromised the ability to express the available force generating capacity explosively. This was due to a lower normalised RFD during the initial 50 ms (-29%) as the normalised RFD during later time periods was unaffected by fatigue. Explosive force production is considered functionally more relevant than MVF during explosive dynamic movements, such as sprinting and jumping (de Ruiter et al., 2006; Tillin et al., 2013). Furthermore, it has been suggested that sports related injuries such as anterior cruciate ligament ruptures occur within 50 ms after ground contact (Krosshaug et al., 2007). Therefore, the fatigue induced reduction in explosive force capabilities that we have observed, particularly during the first 50 ms of contraction, would be expected to contribute to the increased incidence of injury with fatigue in team sports (Hawkins et al., 2001) and the decline in performance of dynamic explosive sporting actions with high intensity fatigue (e.g. Mohr et al., 2003; Krustrup et al., 2006).

This is the first study to assess and report a decline in neural activation during rapid force development with fatigue. The reduction in NE_{0-50} (34%) suggests there was a substantial neural contribution to fatigue. Furthermore, the greater relative reduction in voluntary pRFD

(55%) than octet pRFD (27%) and greater relative reduction in MVF (42%) than octet PF (28%) also suggests central fatigue occurred. In support of this, EMG was shown to decline by 20-28% during explosive force development and by 15% during MVF production. Interestingly, despite the decline in neuromuscular activation during the initial 50 ms (EMG_{0-50}, 28%; NE_{0-50}, 34%) being two-fold more than at MVF (EMG_{MVF}, 15%), the difference between changes in neuromuscular activation during the early phase of explosive force development and at MVF were not statistically different. It is possible that despite taking great care with the measurements of EMG (e.g. recording from 6 sites on the quadriceps muscle and using M_{max} Area normalisation) that this lacked the sensitivity to contrast activation changes during the different phases of contraction. For example, the inter-individual variability in the changes in EMG amplitude with fatigue were very high (EMG_{0-50}, relative changes, range, +16% to -83%), thus, potentially limiting our ability to contrast the changes in EMG during different phases of contraction. Therefore, the non-significant two-fold greater decline in neural drive during the initial 50 ms of contraction may have contributed to the 29% decline in normalised RFD_{0-50}.

Octet RFD is thought to reveal the MTUs maximal capacity for explosive force production (de Ruiter et al., 2004), and therefore a 23-28% decline in octet responses (F_{50}, pRFD) clearly demonstrated a marked reduction in the intrinsic contractile capacity for explosive force production. Evoked twitch responses were also included within the study to provide further information on the contractile mechanisms of fatigue. The substantial decrease in twitch force responses (59-66%) reinforced the impairment of contractile function with fatigue. In a recent review, Allen and colleagues (2008) suggested that the underlying mechanisms of contractile fatigue are largely attributed to reduced Ca^{2+} sensitivity of the contractile proteins and reduced sarcoplasmic reticulum (SR) Ca^{2+} release. These mechanistic changes would be expected to exert a more pronounced influence at low than high frequencies of stimulation (see Balog, 2010), and thus likely explains the observed discrepancies between twitch and octet force responses. Normalised octet pRFD (to octet PF) was unchanged throughout the protocol, indicating no disproportionate effect of contractile fatigue on explosive force production. Thereby, it appears that the disproportionate drop in volitional explosive than MVF, typified by the reduction in normalised RFD_{0-50}, was not due to contractile fatigue per se.

Within the current study neuromuscular activation during the early phase of explosive contraction (0-50 ms) measured using either the neural efficacy technique (30-40% activation) or surface EMG (~50% EMG_{MVCF}) was low and likely indicates on average a sub-maximalr level of intracellular Ca^{2+} saturation during this period. Early phase explosive voluntary force production (0-50 ms) has been found to be related to the evoked twitch response (peak twitch RFD, Andersen & Aagaard, 2006; twitch force at 50 ms, Folland *et al*. 2013), and may reflect a common response to sub-maximal activation and incomplete Ca^{2+} saturation (Andersen & Aagaard, 2006). Therefore, the decline in early phase normalised voluntary explosive force/RFD with fatigue in this study could in part be due to a more pronounced decline in the contractile response to sub-maximal activation (as shown by the substantial decrease in the evoked twitch response), than is the case for maximal activation.

To conclude, it was found that explosive force declined more rapidly and in a more pronounced manner than MVF. Both neural and contractile fatigue mechanisms appeared to contribute to the decline in absolute explosive force and MVF. The early phase of explosive force development (0-50 ms) was particularly susceptible to fatigue, and the reduction in normalised explosive force compromised force development throughout the rising force-time curve. The decline in normalised explosive voluntary force in the early phase of contraction was likely due to the greater impairment of explosive neural drive, and/or the more marked reduction in the explosive contractile response to sub-maximal activation (reflected by an evoked twitch).

CHAPTER 9

General Discussion

The aim of the thesis was to further understand the contribution of neural factors such as the level of agonist, antagonist and stabilisr muscle activation to maximal muscle performance, with a specific focus on explosive neuromuscular performance, and document how these change with RT and high intensity fatigue. The aim of the thesis was achieved using six experimental investigations and the main findings of these investigations are summarised below:

- Chapter 3 reported that the within-participant between session reliability of explosive force/RFD over 50 ms was highly variable, but reliable for time points/periods greater than 50 ms. On a group level, explosive voluntary force measurements at all time points were stable and consistent between sessions. The absolute EMG amplitude was highly variable for individuals between measurement sessions for both maximal and explosive voluntary contractions, and this was not improved by normalisation of the signal to any reference technique used. Electrically-evoked measurements were typically reliable for individuals and the group between sessions.

- Chapter 4 reported that the ability for agonist neuromuscular activation during the early phase of contraction explained 41% of variability in voluntary EMD_{max}, whilst evoked and voluntary EMD_{max} had only a small relationship with one another (18%).

- Chapter 5 reported a BLD in explosive but not MVF production. The BLD in explosive force occurred at 100 ms only and reflected a BLD specific to RFD during the 50-100 ms time window. The deficit was thought to be due an underlying physiological mechanism, as methodological factors accounted for little of this deficit. The BLD was not solely attributable to reduced agonist or antagonist neural drive, and could have been explained by the level of stabiliser activation during explosive force production.

- Chapter 6 revealed that stabiliser neuromuscular activation was not an independent determinant of explosive strength for any time point, but did have a strong indirect association with explosive strength, through its high shared variance with agonist activation during the early phase of contraction (0-50 ms).

- Chapter 7 reported a more than two-fold greater increase in training-task specific isoinertial than isometric strength, which appeared to be due to task specific increase in agonist activation. A key finding was the increased stabiliser activation during all elbow flexion tasks post training. Isoinertial RT even with maximal intention to lift

the weight as quickly as possible was not sufficient to elicit adaptations in explosive strength, despite a large increase in stabiliser activation during explosive force production.

- Chapter 8 reported that fatigue exerted a more rapid and pronounced influence on explosive than maximal isometric strength. This decline was most pronounced during the very early phase (0-50 ms) which may have implications for injury risk. Fatigue had no effect on voluntary EMD_{max}, but substantially elongated evoked EMD_{max}. Neural and contractile mechanisms were thought responsible for the reduction in explosive strength with fatigue.

9.1 Implications for Assessment of Neuromuscular Function

The low within-participant reliability of early phase explosive force and EMG amplitude throughout the rising force time curve and at MVF limits the ability to assess inter-individual changes in following an intervention. Poor reliability for force was only observable at 50 ms, and actually force at 100 and 150 ms had very good reliability. Chapter three shaped the analytical techniques of the latter chapters and suggests that we need to be cautious with EMG and early phase explosive force as measurement techniques, and thus, when interpreting individual changes between measurement sessions. As a main focus of the PhD was specifically early phase explosive strength, an individual response was not adopted throughout the PhD. If the focus of research is specifically on early phase explosive strength or EMG, then group as opposed to individual responses are recommended. This high inter-individual variability of EMG and early phase explosive strength would undoubtedly limit the applicability of these techniques within an applied setting, in which applied scientists are typically interested in individual adaptations to training and rehabilitation or identifying individual strength or activation weaknesses. Normalisation of the EMG signal did not improve the EMG reliability but did reduce the between participant variability, and therefore should still be utilised where possible to enhance the statistical power of a research study. An approach to try to enhance the reliability of EMG within this thesis was to use duplicate EMG electrodes on each muscle where possible. Although, I did not directly assess if this approach enhances the reliability or validity of EMG, it could be expected to enhance its use for assessment of neuromuscular activation. However, despite recording from six sites on the quadriceps muscle and normalising EMG to M_{max} within chapter five, there were still concerns as to the ability of the method to contrast activation changes during the different

phases of contraction. Neural Efficacy was shown not to be a more reliable measurement technique than EMG, and may in part relate to a high variability in agonist activation during the early phase of contraction, which would be high regardless of the measurement technique. The much higher variability in early phase explosive force (0-50 ms) than later phase explosive force (100, 150 ms), could be due largely to a much stonger influence of agonist activation on early than late phase force production.

An approach adopted to improve our understanding of RFD within this thesis was the use of separate time windows during the rising force-time curve. Tillin et al. (2012) suggested this method to be superior and use of this analysis technique undoubtedly contributed to a superior insight into the understanding of RFD and associated mechanisms throughout this thesis. The use of separate windows allowed for more detailed reporting of how the interventions differentially affected RFD throughout the rising force-time curve. For instance, fatigue was shown to exert a reduction in relative RFD during the initial time period only (0-50 ms), which resulted in the declines in explosive force at 50, 100 and 150 ms, whilst the use of BL actions in chapter 5 negatively affected relative RFD for 50-100 ms only. Continued use of separate windows across the force-time curve during explosive contractions is recommended to other scientists conducting scientific research on explosive neuromuscular function.

The relative importance of stabiliser activation found within the current thesis suggests that the stabiliser muscle system should receive further investigation in future research studies when the focus is to understand the neural contributions to expression of maximal muscle performance. This research is the first to assess stabiliser activation during explosive contractions as well as documenting how stabiliser EMG changes following RT during maximal strength tasks. Implications from this thesis, such as the inter-relationship between stabiliser and agonist activation during explosive force production suggest that the level of stabiliser muscle activation should be measured alongside that of agonist activation when assessing the human neuromuscular systems control strategies.

9.2 Neural Contributions to Maximal Muscle Performance

9.2.1 Maximal Strength

A key contribution of this thesis was the findings of task specific adaptations in muscle strength following short term (three weeks) RT. For years, training specificity has been suggested to provide strong indirect evidence for neural adaptations to RT, and thus a contributor to the expression of muscle strength (Sale, 1988; Folland & Williams, 2007a). However, no study until now had actually demonstrated this phenomenon to be due to neural or morphological mechanisms. Early adaptations to RT are thought to be primarily explained by neural adaptations, e.g. enhanced agonist, antagonist and stabiliser muscle activation (Folland & Williams 2007a), with a greater contribution from morphological adaptations, such as selective hypertrophy and/or architectural changes, as training duration progresses (Narici et al., 1996). This task specificity phenomenon was accompanied by greater increase in agonist activation during the isoinertial strength task than at iMVF. Although, stabiliser activation increased during the training task this was not a task-specific adaptation. Thus, strong support now exists to suggest that training specific changes following RT, are likely explained by task specific increases in agonist activation.

An unanswered question from chapter six was 'how the changes in stabiliser activation may have i) contributed to changes in isometric and isoinertial strength and ii) contributed to the task specific changes in strength observed?' During isometric strength tasks, the requirement for stabiliser activation is limited as the joint is constrained in place to limit extraneous movement from adjacent joints and simplify the task to allow for a more precise examination of the joint at hand. Thus, increased stabiliser activation might be expected to contribute minimally to this type of task. Cacchio et al. (2008) reported a greater cross over training effect from constrained training to unconstrained path chest press strength than vice versa, and suggested that the motor pattern improvements from isoinertial type training cannot be utilised during constrained path strength analysis. On the other hand given the greater number of degrees of freedom and requirement to voluntarily stabilise joint complexes during the isoinertial task, it is possible this increased stabiliser activation, may have facilitated increased strength, thereby contributing to the observed training specificity.

A further question arising from chapter seven was a) 'does an increase in agonist activation and resultant increase in strength place an increase demand on the stabiliser muscles, due to a

greater load being lifted, or b) that the enhanced joint stability, through elevated stabiliser activation actually facilitated an increased level of agonist activation, and thereby increased isoinertial strength?'. The agonist muscles assessed with chapter six and seven were that of the biceps brachii, which are bi-articular muscles originating at the shoulder and inserting into the elbow. Optimal muscle activation requires a sufficient base of support, thereby in the context of these muscles, a sufficient level of shoulder stability. Chapter six reported an association between the level of agonist and stabiliser muscle activation which confirmed they were inter-related, and although MVF was not specifically measured, there was a relationship over the initial 150 ms of contraction. It is possible that poor shoulder stability, i.e. low stabiliser muscle activation prior training could have acted as a constraint to the level of bi-articular agonist activation. There was limited increase in maximal and no change in absolute explosive force during isometric elbow flexion tasks following training, despite a large increase in stabiliser acitvation, which would suggest that stabiliser activation does not constrain the level of agonist activation during these tasks. However, important considerations of this study were that these measurements were made isometrically and therefore, it is possible that enhanced inter-muscular coordination (increased stabiliser activation) could not effectively be utilised during these tasks (Cacchio et al., 2008). There was increased strength and stabiliser and agonist activation during the isoinertial strength tasks and therefore, further research is needed to fully understand the role of stabiliser activation on muscle performance, but particularly during isoinertial strength tasks.

Finally, a key finding from chapter 7 was that isoinertial agonist EMG during the 1RM was substantially higher that agonist EMG at iMVF. Activation of the elbow flexors, as assessed via the ITT, during an isometric MVC is thought to be very high/ maximal (98-100%, Allen et al., 1998; Behm et al., 2002). The validity of the ITT has been questioned (see de Hann et al., 2009), but such a substantial increase in EMG does seem contradictory to the current research. It is important to assess the level of agonist EMG during isoinertial and isometric maximal tasks, as well as further research the validity of various methods of assessing voluntary activation during muscle contractions.

9.2.2 Electromechanical Delay

A novel finding of the thesis was the association reported between early phase agonist activation capability and voluntary EMD_{max} (41%) in chapter 4, which further emphasises the role of agonist activation on explosive neuromuscular performance. Evoked EMD_{max} explained only a small proportion of the variability in voluntary EMD_{max} (18%). Other studies have failed to report an association between evoked and voluntary EMD (Zhou et al., 1996; Minshull et al., 2007), but these studies did not assess EMD_{max}, but instead EMD of the individual constituent muscles within the quadricep muscles. Together it appears that evoked EMD capabilities do not exert a strong influence on the variability in voluntary EMD performance. Despite a decline of ~30% of agonist activation during the initial 50 ms and 50% elongation in evoked EMD_{max}, voluntary EMD_{max} was unchanged with fatigue in chapter 8. The split group analysis performed in chapter 4 did reveal that although agonist EMG was the only variable which differed between the groups, there was a large difference in EMG compared to voluntary EMD_{max} between groups (EMG, 134% vs. EMD, 29% difference), which may suggest very large changes in agonist EMG are required to elicit changes in voluntary EMD_{max}.

9.2.3 Explosive Strength

9.2.3.1 Agonist Activation

There is now considerable evidence available on the importance of agonist EMG on early phase explosive isometric force production. For instance, numerous studies have investigated the relationship between agonist EMG and force during explosive force production over the initial 40-50 ms after contraction onset, and have reported explained variance ranging from 33 up to 75% (de Ruiter et al., 2004; Del Balso & Cafarelli, 2007; Klass et al., 2008; Hannah et al. 2012; Folland et al., 2013). However, it is unclear if the variability in agonist activation exerts a strong influence on the expression of explosive strength over latter time points (> 50 ms). Chapter 3 observed a similar variability in force and agonist EMG for the early phase of contraction (50 ms), but a substantially different level of between session within participant reliability for latter time points (100 and 150 ms and at MVF). If agonist EMG is a strong determinant of force production, then you would expect the two variables to fluctuate similarly. Further interventions as well as cross-sectional studies investigating the relationship between activation and force were carried out to elaborate the role of agonist activation on explosive force production.

Chapter 6 reported a moderate relationship between agonist EMG and isometric explosive force production ($r^2 = 0.36$), but only a tendency for a relationship over 100 ms ($r^2 = 0.15$). EMG during this study was sampled from EMG onset, and therefore EMG over 100 ms also included the 0-50 ms time window. Therefore, it could be argued that there was little contribution of agonist EMG to explosive force production over 50-100 ms. In deed, recent research investigating the determinants of RFD, reported virtually identical relationship between agonist EMG of the quadriceps and knee extensors force production after 50 ms ($r^2 = 0.37$), but observed no contribution of agonist EMG to the variability in RFD for time points greater than 50 ms (50-100 or 100-150 ms, $P > 0.05$, Folland et al., 2013).

The early phase of explosive force development (0-50 ms) was particularly susceptible to fatigue, and the reduction in normalised explosive force compromised force development throughout the rising force-time curve. The decline in normalised explosive voluntary force in the early phase of contraction was likely due to the greater impairment of explosive neural drive, and/or the more marked reduction in the explosive contractile response to sub-maximal activation (reflected by an evoked twitch). There is good evidence that the twitch properties are important determinants of early phase explosive force. Andersen and Aagaard (2006) reported that twitch pRFD explained 20-36% of the variability in RFD during the early phase of contraction from force onset (0-50 ms). This has been supported by Folland et al. (In Press) who reported that twitch force properties explained 40% of the variability in RFD over the initial 50 ms of contraction. Taken together, it appears that the level of agonist activation and response to a given level of stimulation (twitch force responses) are major determinants of early phase explosive force production. As a whole, key findings from this thesis alongside the literature would appear to indicate that the level of agonist activation and twitch properties are important determinants of explosive strength during the early phase of force production, in which the levels of activation are low, but they do not exert a strong influence on the variability in force output at latter time period after force onset, when activation is high/maximal (> 50 ms).

9.2.3.2 Stabiliser Activation

Chapters 6 and 7 aimed to begin to elucidate the role of stabiliser activation on maximal muscle performance. Key findings from chapter six was that the level of stabiliser activation did not directly influence explosive force, but there was a strong relationship between the level of stabiliser activation and agonist EMG, indicating that stabiliser activation had a

strong indirect role on explosive strength. The work was supported by chapter 7 which reported no change in explosive strength, despite large changes in stabiliser activation.

However, from chapter 5 it does appear that the level of stabiliser activation may directly influence the expression of explosive strength. The BLD in RFD observed in chapter 5 was reported in the absence of changes in agonist and antagonist activation, and was suggested that the BLD was due to different postural stabiliser requirements between UL and BL tasks. Jakobi and Chilbeck (2001) reported that greater BLDs can be observed during multiple joint than single joint tasks. Furthermore the BLD has been shown to be higher for tasks requiring greater activation of postural stabilising muscles (leg press versus hand grip, Magnus & Farthing, 2008). As Chapter 5 only utilised a relatively simple task (isometric single joint action), it is possible even more pronounced BLD could be observed for explosive than maximal strength during more complex tasks, involving a greater postural stability.

9.2.3.3 Antagonist Activation

Chapter 6 reported that the level of antagonist co-activation was not related to either the level of force or agonist EMG during explosive force production. It was negatively related to the level of stabiliser activation over the initial 150 ms. It does appear the relationship between antagonist co-activation is complicated. Antagonist EMG has been suggested to be important for joint stability. During isometric contractions of an agonist muscle, the level of antagonist activation appears to scale to agonist activation with increments in force (Krishnan & Williams, 2009) and it is thought that the level of antagonist co-activation is mediated by a central descending common drive to the agonist-antagonist motoneuron pools (Krishnan & Williams, 2009). Furthermore, Tillin et al. (2011) has reported that RT elicited an increase in antagonist activation, but reduced level of antagonist activation for any given level of agonist activation. Finally, Cacchio et al. (2008) reported that RT enhanced stabiliser activation, which allowed for a lower level of antagonist and agonist activation during sub-maximal strength tasks. Collectively, it would appear that those with higher capacity for explosive force production, would be expected to have a higher level of agonist activation and resultant higher level of antagonist activation. Therefore, a positive relationship with antagonist activation and force could be expected. The fact they are unrelated would appear to support that those with enhanced motor control strategies may be able to achieve a lower level of antagonist co-activation for any given level of agonist activation and force and thereby, there was no relationship between force and the level of co-activation. Consequently, when

examining the level of co-activation, consideration of agonist and stabiliser activation and the level of force appear important.

9.3 Implications for Athletic Training

There are various implications from this thesis for athletic training. Firstly, from both this thesis and from the literature as a whole, it is clear that the ability to increase agonist EMG during the very early phase of contraction (0-50 ms) appears to be important for explosive neuromuscular performance (EMD and RFD 0-50 ms). As time from force onset increases EMG becomes high/maximal and other factors than the level of agonist activation appear to largely influence explosive force production. Chapter 6, as well as an array of other studies has reported that the level agonist activation explains about half of the variability in explosive force capabilities during the early phase of contraction (40-50 ms, de Ruiter et al., 2004; Del Balso & Cafarelli, 2007; Klass et al., 2008; Hannah et al., 2012; Folland et al., 2013). The response to a given level of activation (i.e., twitch force responses) likely explains the majority of the remaining variance (Andersen & Aagaard, 2006; Folland et al., 2013; Chapter 5). Therefore, training to enhance the level of agonist activation during the early phase of contraction appears to be an important aspect of enhancing explosive neuromuscular performance. This component of activation can be considered as EMG rise, the ability to increase agonist activation up to maximal levels as quickly as possible. As time is limited during explosive sporting tasks such as sprint running and injury avoidance situations, this can be considered an essential component of explosive sporting performance and injury risk.

At present it is not clear as to the best methods of training for EMG rise. It has been suggested that addition of an explosive strength to conventional strength training is sufficient to train the level of agonist EMG during explosive force production and subsequent explosive strength. However, chapter six reported that conventional isoinertial RT, even with maximal intention to lift the weight as quickly as possible did not improve RFD through neural adaptations. It is thought that exercises that involve transition from low to high force levels is required to develop this component of muscle performance. The majority of research that has reported increased EMG and force following strength training has used isometric contractions with maximum RFD (i.e., Rich & Cafarelli, 2000; Pucci et al., 2006; Del Balso et al., 2007; Tillin et al., 2012a) or ballistic contractions using low loads (i.e., Van Cutsem et al., 1998) and therefore there is no or low EMG either prior to force production (isometric rest period or

low EMG during the eccentric phase of ballistic actions). Equivocal findings are typically observed with those studies adopting conventional isoinertial type models (although have not included the maximal intention to lift the weight) (i.e., Aagaard et al., 2002a; Bruhn et al., 2007; Andersen et al., 2010). In the light of the current thesis an available evidence it is suggested that separate specific training targeted specifically at training the ability to increase agonist EMG at onset (EMG rise) be included in athletic development programmes.

As it was reported that fatigue may exert more pronounced effects on explosive than maximal strength, this indicates that there is a requirement to avoid high levels of fatigue, to maintain the levels of neuromuscular performance which are essential for joint stability and injury avoidance. Fatigue is thought to be an important risk factor for injury and an enhanced EMD and reduced RFD would be expected to increase the time required for joint stability following mechanical perturbation and therefore increased injury risk. The decline in explosive neuromuscular performance was thought to be due to both neural and contractile mechanisms. Advice from chapter 8 would be that avoidance of high levels of fatigue is an essential aspect of injury prevention. As RT did not enhance explosive neuromuscular performance, fatigue resistance would appear to be an essential component of athletic training to reduce injury risk.

The observed 15% deficit in RFD, despite no influence of BL actions on MVF suggests specific training to offset this deficit should be performed in order to maximise the performance of BL explosive sporting tasks. The BLD observed was thought to be explained by reduced inter-muscular coordination (lower stabiliser activation) during BL efforts and suggests that specific practice of coordinated explosive BL tasks and improved core/joint stability could be expected to improve the expression of BL explosive sporting tasks through reducing this explosive force/RFD BLD.

Although there is limited research available, greater consideration of stabilising muscles for athletic development and injury prevention may be sought. It is possible that stabiliser activation could act as a constraint to optimal agonist activation and therefore exert strong indirect effects on muscle performance. It is apparent that instability creates a reduced force output (for a review see Behm & Anderson, 2005). Therefore, insufficient joint stability through low levels of stabiliser activation could exert similar intrinsic influence on adjacent muscle performance. Including specific training to enhance the stabiliser muscle system may

be expected to benefit neuromuscular performance and thus sports performance as well as reduced injury risk.

9.4 Directions for Future Research

1. The findings of stabiliser activation from this thesis are intriguing, but the assessment of stabiliser activation and muscle performance during isometric single joint situations, unlikely reveals the full contribution of the stabiliser muscle system to muscle performance during more functional situations. A body of research investigating the importance of stabiliser activation on maximal and explosive strength, during isoinertial situations would appear to be important in order to fully understand the contributions of muscle activation strategies to maximal muscle performance.

2. As isoinertial RT did not enhance agonist EMG during explosive muscle actions, further research into the influence of RT on explosive neuromuscular performance is required. It is possible that this type of exercise does not offer the transition in EMG from low to high levels required to train this aspect of neural function. Documenting the EMG patterns during conventional isoinertial lifting tasks throughout the eccentric and concentric phases, and reporting the changes in EMG rise during this concentric phase would improve our understanding as to why isoinertial RT with maximal intention to lift the weight as quickly as possible did not enhance agonist EMG during the early phase of contraction.

3. Chapter six was the higher agonist EMG observed during isoinertial 1RM than at iMVF. The exact reasons are not known, but it is thought that activation is maximal or at least close to maximal during isometric MVC of the elbow flexors. Research comparing activation during isometric and isoinertial tasks, and understanding the possible mechanisms associated is sought.

4. Research to examine the BLD during explosive multiple joint tasks and document the neural, methodological factors which might explain this including assessment of agonist, antagonist and stabiliser muscle activation of major muscle groups is needed.

5. Research examining if duplicate EMG electrodes improves the reliability of normalised EMG amplitude should be undertaken.

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