Physiological and Biomechanical Responses during High Intensity Upper Body Exercise

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STATEMENT OF ORIGINALITY

The accompanying thesis submitted for the degree of Doctor of Philosophy entitled 'Physiological and biomechanical responses during high intensity upper body exercise' is based on work conducted by the author in the School of Health at The University of Northampton mainly during the period between October 2008 and June 2012

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

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

Signed ..... Date .....

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Abstract

Fatigue during sport and exercise substantially affects the intensity and duration of an activity that can be maintained. Upper body exercise (UBE) despite contributing to sport, exercise and health outcomes has received relatively little attention particularly for high intensity exercise. Consequently, the mechanisms of fatigue during UBE are not fully understood. Therefore, the aims of this thesis were to investigate a range of high intensity UBE protocols with respect to performance and the development of fatigue. In the first study participants (n = 13) completed four 30-s Wingate anaerobic tests (WAnT) against four different resistive loadings (2%, 3%, 4% and 5% body mass) thus potentially manipulating force production and cadence. Corrected peak power output (PPO) was independent of load ($P > 0.05$) and uncorrected PPO increased with load ($P < 0.05$). Results from EMG analysis demonstrated that all upper body sites increased EMG activity at the point of fatigue/minimum power output (PO). The biceps brachii was predominately affected by resistive load at corrected and uncorrected PPO. Kinematic analysis revealed significant changes in trunk rotational velocity which was greater for 3% *vs* 4% resistive load ($P < 0.05$). These data suggest that the biceps brachii is an important contributor to PPO and that resistive load influences kinematic responses. In the second study, participants (n = 14) completed four separate high intensity trials (80%, 90%, 100% and 110% of peak minute power; PMP) from an incremental test for peak oxygen uptake (VO_{2peak}) to volitional exhaustion (T_{lim}) at a fixed cadence and PO. There were significant increases in EMG activation over time (s) and in relation to the exercise intensity ($P < 0.001$). Trunk rotational velocity increased with load prior to T_{lim} ($P < 0.001$) although at T_{lim} there were no differences between trials ($P > 0.05$). All participants reached their maximum cardiorespiratory responses (oxygen uptake & heart rate; beats·min^{-1}) at fatigue. The data suggested that prior to T_{lim} changes in EMG activation and movement patterns were related to the exercise intensity. In general, all EMG activity increased with intensity and exercise duration, with the kinematic data indicating that trunk rotational velocity rather than trunk stabilisation occurred throughout all trials. Overall, untrained participants altered their body movement to maintain PO between 30 & 120 s, however between 120 s & T_{lim}, no further significant changes occurred. In the final study, participants (n = 12) completed a 6-week arm crank training programme. Preliminary performance tests included a WAnT, VO_{2peak} and 100% PMP test to exhaustion. Each test was repeated following the training programme. Corrected and uncorrected PPO and fatigue index (FI) increased in the WAnT test post training ($P < 0.01$, $P \leq 0.05$, respectively). Muscles of the shoulder (anterior deltoid & infraspinatus) demonstrated reduced activation following training ($P \leq 0.05$) with trunk rotational velocity increasing at corrected PPO during the WAnT ($P \leq 0.01$). Therefore, increases in WAnT PO may be related to changes in technique rather than muscle activation. Following training there was a significant increase in PMP ($P < 0.01$) during the VO_{2peak} test and a significant increase in T_{lim} ($P < 0.01$) for the repeated 100% PMP test. Following training there was a significant decrease in triceps brachii EMG activation ($P \leq 0.05$), changes in external oblique activation ($P < 0.001$) at 120 s and a significant increase in trunk rotational velocity at 30 s ($P \leq 0.05$). Although at T_{lim} the kinematic responses were the same. The results of this training study indicated that changes in performance were due to physiological adaptations and changes in technique. The three studies have demonstrated the importance of changes in EMG activity, trunk rotational velocity, and technique to arm crank PO rather than specific physiological changes alone which has implications for the use of arm cranking in testing, training and performance outcomes.

Contents

List of Tables and Figures

Research publications generated from the thesis

Presentations and publications

Talbot, C. & Price, M. (2009) Familiarisation for upper body Wingate testing. 25th *annual conference of the British Association of Sport & Exercise Sciences, Leeds*, United Kingdom, September 1-3, 2009. Journal of Sports Sciences, 27 (S2): S126-S127.

Talbot, C & Price, M. Load optimisation for upper body Wingate testing. 26th *annual conference of the British Association of Sport & Exercise Sciences, Glasgow*, United Kingdom, September 6-8, 2010. Journal of Sports Sciences, 28 (S1): S154-S155.

Talbot, C., Kay, T. & Price, M. A comparison of two Wingate Anaerobic Test software packages. 26th *annual conference of the British Association of Sport & Exercise Sciences, Leeds*, United Kingdom, September 6-8, 2010. Journal of Sports Sciences, 28 (S1): S154-S155.

Talbot, C., Kay, T., Walker. N. & Price, M. Electromyography during upper body Wingate exercise. 27th *annual conference of the British Association of Sport & Exercise Sciences, Colchester*, United Kingdom, September 6-8, 2011. Journal of Sports Sciences, 29 (S1): S131.

Chapter 1

1.0 Introduction

In sport and exercise fatigue substantially affects the intensity and duration of an activity that can be maintained and therefore, performance. Lower body performance factors such as physiology and biomechanics contributing to fatigue are extensively reported in the literature. Research into fatigue during upper body exercise has received comparatively little attention despite contributing to a number of sports, exercise and health outcomes and having substantially different physiological and biomechanical responses to lower body exercise. A small number of studies have examined upper body exercise in relation to physiology and biomechanics (Bressel and Heise, 2004; Frauendorf *et al.*, 1989; Hopman *et al.*, 1995; Marais *et al.*, 2004; Price *et al.*, 2007; Smith *et al.*, 2008; Smith *et al.*, 2007a; Smith *et al.*, 2006c). Recent physiological research has examined optimal cadence for peak oxygen consumption (Smith *et al.*, 2007a, Smith *et al.*, 2001) and body position in relation to the ergometer (Leicht and Spinks, 2007, van Drongelen *et al.*, 2009, Miller *et al.*, 2004). These studies indicate cadence and body position have an effect on physiological responses to arm crank ergometry (ACE). Additionally, ACE testing has received specific recommendations from the British Association of Sport and Exercise Science (Smith and Price, 2007) and research interest in this field is increasing.

Physiological markers of performance during upper body exercise are generally reported at a low intensity (less than 70% of peak oxygen uptake) although the majority of training for sport and exercise is undertaken at higher intensities (Bouhlel *et al.*, 2007, Billat *et al.*, 1996, Fernandes *et al.*, 2008b). A number of tests have been developed to evaluate performance at high intensities and the Wingate anaerobic test (WAnT) is one of these. The Wingate anaerobic test is a maximal test over 30 s duration and is used in upper and lower body exercise testing (Lovell *et al.*, 2011a, Zagatto *et al.*, 2008, Smith *et al.*, 2007b). The WAnT measures power output, cadence and fatigue. The majority of muscular power generated during the test comes from the anaerobic metabolic pathways (Beneke *et al.*, 2002, Bediz *et al.*, 1998, Smith and Hill, 1991) and is a useful and reliable measure of peak power output and fatigue (Bar-Or *et al.*, 1977, Inbar *et al.*, 1996). Manipulating the test load alters cadence and power output (a lower load generally results in a faster cadence) and therefore fatigue which

will affect the subsequent physiological and biomechanical responses (Inbar *et al.*, 1996, Patton *et al.*, 1985, Dotan and Bar-Or, 1983). Although a number of studies have reported arm crank ergometry during WAnT performance (Kounalakis *et al.*, 2008, Weber *et al.*, 2006, Jemini *et al.*, 2006) EMG responses during an upper body WAnT have not been reported in the literature. However, near-infrared spectroscopy studies have found that changes in muscle recruitment patterns exist (Kounalakis *et al.*, 2009) and a high intensity exercise study using EMG analysis indicated changes in shoulder girdle kinematics and muscle co-ordination in the infraspinatus and deltoid muscles (Ebaugh *et al.*, 2006). In addition, the optimal resistive load for an upper body WAnT has not been thoroughly examined since the original suggestion of 6% body mass resistive load (Dotan and Bar-Or, 1983). Therefore, the use of motion analysis and EMG may highlight significant changes in limb kinematics and muscle recruitment patterns to enhance our understanding and interpretation of power production and the effects of fatigue across a range of resistive loads.

Arm crank ergometry during exercise at higher intensities (80%-110% of peak oxygen uptake; VO_{2peak}) has generally only been reported through examining protocols for VO_{2peak}. The combined physiology and biomechanics at and around such high intensities continued to volitional fatigue for ACE has not been published. Whether responses at these intensities fit within the severe exercise domain reported for lower body studies requires further investigation, especially as many sport and exercise endeavours are associated with paced rather than incremental effort(s) to exhaustion (Atkinson *et al.*, 2003, Grant *et al.*, 1997, Lambert *et al.*, 1995). High intensity responses such as changes in efficiency and oxygen uptake have been attributed in part to unmeasured work of the trunk and lower body (Stamford *et al.*, 1978, Bar-Or and Zwiren, 1975, Blasio *et al.*, 2009) and increases in trunk rotation and shoulder range of motion have been linked with a decrease in cadence (Price *et al.*, 2007). Whether these responses are the same for a fixed cadence but different resistive loads and therefore exercise intensities and the influence of training has not to the not been reported in the literature. Ratings of perceived exertion (local and central) have been used as indicators of physiological response to ACE with a local response generally greater than central (Pandolf *et al.*, 1984). These responses are supported by physiological evidence that shows oxygen uptake is restricted by local rather than central (cardiovascular) fatigue (Magel *et al.*, 1975). This is

probably due to a smaller muscle mass (Sawka, 1986, Washburn and Seals, 1984) in the arms and a reduced stroke volume (Astrand *et al.*, 1965) and can be linked to the lesser aerobic capacity of the exercising muscles (Davies and Sargeant, 1975).

Lower body studies have indicated that training can increase the duration and/or power output achieved for the WAnT test (Ziemann *et al.*, 2011, Buśko, 2011) and tests to volitional exhaustion at high intensities (Ziemann *et al.*, 2011, Burgomaster *et al.*, 2005). A number of ACE training studies have shown that peak oxygen uptake can be increased through aerobic training programmes (Magel *et al.*, 1978, Loftin *et al.*, 1988, Franklin, 1989) or weight training only (Swensen *et al.*, 1993). Additionally, comparisons with upper body trained compared to untrained participants indicates improved performance such as an increased work capacity (Volianitis *et al.*, 2004a) and aerobic capacity (Franklin, 1985). Wingate anaerobic test comparisons between different levels of ability in sports show that a greater ability is reflected in a higher peak and mean power output in wrestlers (Horswill *et al.*, 1992, Terbizan and Seljevold, 1996) and gymnasts (Jemini *et al.*, 2006). These studies indicate that upper body training can increase performance, although exact physiological (i.e. changes in respiratory measures) and biomechanical responses (such as EMG and motion analysis) have not been fully explored in the literature. Further research is required to establish the changes in physiological and biomechanical responses that may result from ACE training.

Exercise duration and/or intensity is frequently restricted by fatigue (Ament and Verkerke, 2009, Enoka and Duchateau, 2008). There are many different definitions of fatigue of which the majority confirm that it results in a reduction in performance/force and can be physical and/or mental (Szygula *et al.*, 2003, Fitts, 1996, Kay *et al.*, 2001, Sargeant, 1994). Fatigue during exercise has a central and/or local source (Sahlin, 1992, Davis, 1995, Bigland-Ritchie, 1981). During high intensity exercise a number of fatigue mechanisms may reduce performance, such as an increase of inorganic phosphate interfering with sarcoplasmic reticulum Ca^{2+} handling and the cross-bridge cycle (Westerblad *et al.*, 2002, McLester, 1997, Bangsbo *et al.*, 1996). Additionally, as muscular contraction produces metabolic by-products these may change the feedback from group III-IV afferents (Taylor *et al.*, 2000, Girard *et al.*, 2011) and therefore the

responses from central nervous system and effect physiological responses such as cardiovascular and ventilatory responses (Christine M. Adreani *et al.*, 1997, Amann, 2012). Whilst it is possible to detect fatigue via reductions in power output e.g. WAnT reductions from peak power to minimum power output or the cessation of power output via a constant load and cadence test to volitional exhaustion this does not provide a complete analysis and biomechanical changes also need to be considered.

In upper body exercise changes in feedback have been linked to fatigue and may be associated with changes in electromyographic (EMG) responses such as increased EMG activity of the biceps and triceps brachii (Martin *et al.*, 2006). Additionally, upper arm postural muscles such as the infraspinatus may increase in amplitude in response to fatigue and changes in position (Rudroff *et al.*, 2007). Current EMG studies specific to ACE emphasise the biceps and triceps brachii to power production (Bressel and Heise, 2004, Marais *et al.*, 2004, Bressel *et al.*, 2001, Smith *et al.*, 2008) and the contribution of muscles of the shoulder (Smith *et al.*, 2008, Frauendorf *et al.*, 1989). Although muscles of the trunk have been suggested to be important to ACE (Bar-Or and Zwiren, 1975, Stamford *et al.*, 1978, Shiomi *et al.*, 2000, Smith *et al.*, 2008) only two published studies have analysed abdominal muscles activation during ACE which showed greater activity during synchronous rather than asynchronous ACE (Hopman *et al.*, 1995), and greater activity whilst sitting on a stability ball versus a chair (Marks *et al.*, 2012). These studies highlight the importance of different muscles to the power production during ACE, whether similar patterns of activation exist during maximal and high intensity ACE remains to be reported.

Fatigue can also be detected by changes in motion analysis. Changes in motion during lower body exercise have been used as markers of fatigue with changes in movement patterns in runners (Millet *et al.*, 2010, Geiser *et al.*, 2010). With upper body fatigue, using isokinetic dynamometry, greater limb movement was required before limb movements can be detected (Taylor *et al.*, 2000, Lee *et al.*, 2003a). During ACE at VO_{2peak} changes in shoulder range of motion and trunk angle were reported to be greater at 50 and 70 rev·min^{-1} compared to 90 rev·min^{-1} (Price *et al.*, 2007). At a low intensity of exercise differences have been observed in wrist flexion and muscle activity (Bressel and Heise, 2004). Therefore, as fatigue can be detected by motion analysis, it may be possible to

identify such markers of fatigue during maximal and high intensity exercise. To the author's knowledge no publications have examined the physiological and biomechanical responses to fatigue during maximal and high intensity upper body exercise.

The key aim of this thesis is to establish, using physiology and biomechanical analysis, how fatigue effects performance during maximal and high intensity upper body exercise. Such an integrated approach is novel in this area of research as previous studies have only reported analysis independent of the other factor. Further aims are to establish an optimal protocol for maximal intensity 30-s arm cranking that elicits maximal performance and also determine the optimal exercise intensity for the assessment of continuous high intensity (anaerobic) upper body exercise performance. Finally, a training study could establish how training effects ACE performance and therefore the physiological and biomechanical variables associated with fatigue during maximal and high intensity exercise.

Chapter 2

Literature Review

2.0 Introduction

This chapter aims to draw together the available research and present a background to upper body exercise, in particular arm crank ergometry (ACE) with specific reference to maximal and high intensity ACE. The applications of ACE, protocols employed, factors influencing performance and fatigue in upper body exercise were examined to set out the main areas of research that this thesis reviewed. Two review papers on upper body exercise have been published; Franklin (1985) published a review on arm ergometry training and testing while Sawka (1986) reviewed the physiology of upper body exercise. More recently, BASES guidelines have provided recommendations for upper body exercise testing (Smith and Price, 2007). This chapter aims to present a more detailed and specifically focussed review since these papers were published, and critique studies that have examined the physiological and biomechanical responses to high intensity upper body exercise. Additionally, the role of muscle anatomy, function and metabolism will be explored with reference to upper body exercise.

2.1 Muscle anatomy and function

Each muscle group, which contains hundreds to thousands of muscle fibres, and tapers into a tendon or broad tendinous sheet at each end which connects to bone (Hijikata *et al.*, 1993). The outside of the muscle is wrapped in a sheath of collagen fibres the epimysium. Bundles of muscle fibres are wrapped in perimysium, and each muscle fibre is wrapped in endomysium (Figure 2.1) which also ties together adjacent muscle fibres. The sheaths support each cell and protect the muscle.

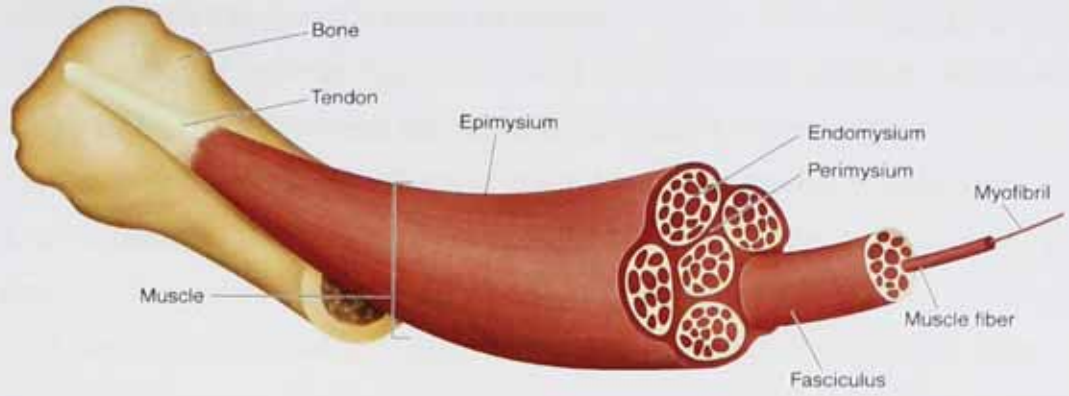


Figure 2.1 Skeletal muscle structure (from Wilmore and Costill, 1999).

Each muscle fibre (Figure 2.2) is enveloped in a thin elastic membrane, the sarcolemma which surrounds the sacroplasm. The sarcoplasmic reticulum consists of vesicles and channels that wrap around and into the spaces of the myofibrils; its major function is to regular intracellular levels of ionic calcium. The transverse tubules are continuous with the sarcolemma and run deep into the muscle fibre and severe to propagate the nerve-initiated electrical impulse further into the muscle cells and sarcomere. It is the electrical impulse that acts a signal to the release of calcium ions into the sarcoplasm which can lead to muscle fibre contraction (Morgan and Allen, 1999).

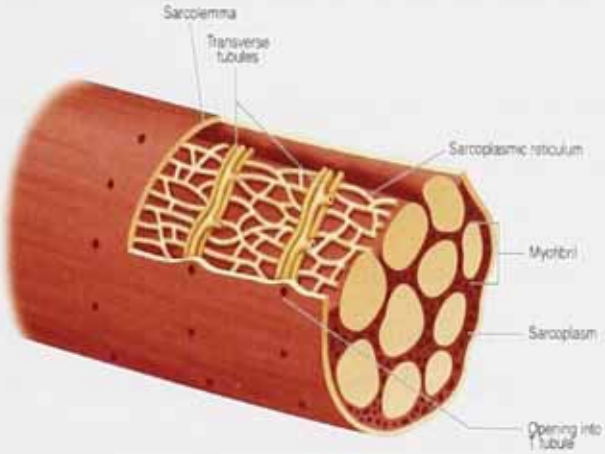


Figure 2.2 A muscle fibre (Wilmore and Costill, 1999).

Each muscle fibre is further subdivided into a myofibril (bundles of myofilaments). Each muscle fibre contains hundreds to thousands of myofibrils. Within and around the myofibrils are mitochondria and granules of glycogen. A sarcomere (Figure 2.3) is a section of myofibril and is the contractile unit of the muscle. Each myofibril consists of about 10,000 sarcomeres (Morgan and Allen,

1999). Each sarcomere contains myofilaments. Each myofilament contains of thick bundle of myosin molecules and a thin strand of actin molecules (Figure 2.3). Each thick filament contains around 500 myosin molecules. Each myosin molecule contains a tail which is bound to other myosin molecules and a head and hinge that allows the myosin head to move. The myosin head interacts with the thin filaments during a contraction (Herzog *et al.*, 2008). The thin filament contains three proteins: F actin, tropomyosin, and troponin. F actin contains individual globular molecules of G actin which contains an active binding site. The binding site can bind to a thick filament (Holmes, 1998). Tropomyosin and troponin assist in making and breaking the contact between thick and thin filaments during contraction.

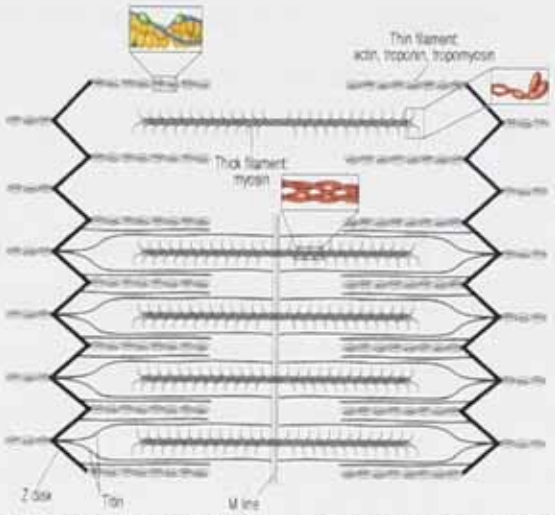


Figure 2.3 Filament arrangements in a sarcomere (Wilmore and Costill, 1999).

For muscle contraction to occur the F actin binding sites need to be exposed by a change in position of the troponin-tropomyosin complex. This change in position occurs when calcium ions bind to receptors on the troponin molecules (Morgan and Allen, 1999). This binding enables the cross-bridges from myosin to attach onto the G actin. Once bound the myosin head pivots towards the centre of the sarcomere (M line) pulling the actin strands closer together. The process of contraction is called sliding filament theory from a hypothesis by Huxley in 1954 (Huxley, 2000). The myosin head is unbound when by the attachment of ATP and hydrolysis of ATP, which results in the recocking of the myosin head and (Holmes, 1998, Rayment *et al.*, 1993). Provided that there is sufficient calcium ion concentrations still present then the process is repeated and the myosin head again pivots towards the centre of the sarcomere. The process end when the calcium ions are pumped back into the sarcoplasmic reticulum (Morgan and Allen, 1999).

2.2 Muscle metabolism

The power for the muscles to contract comes from chemical energy in the form of adenosine triphosphate (ATP). The bonds that join the three phosphates that form part of ATP when broken release energy. This breakdown of ATP provides an immediate energy source releasing adenosine triphosphate (ADP) and providing energy for mechanical work to be complete (Rayment *et al.*, 1993). The store of ATP is sufficient for a few seconds of work (Astrand and Rodahl, 1986). The breakdown of ATP takes place when it is combined with H_2O; this reaction is catalysed by adenosine triphosphatase. The lack of sufficient stores of ATP means that the cells are dependent on further mechanisms to provide ATP. Some of these processes take place whether there is oxygen present or not and are therefore referred to as anaerobic. The rephosphorylation of ATP is provided the catalysation of ADP and creatine phosphate by creatine kinase (Astrand and Rodahl, 1986). Myoadenylate kinase can also convert two molecules of ADP to one molecule of ATP and one of AMP (Brooks *et al.*, 2005). The above energy sources are rapid and provide an immediate supply of energy. The amount of ATP that is available by these sources, including stored ATP, can only supply energy lasting no more than 5–15 seconds (Brooks *et al.*, 2005). Therefore, during exercise of a short duration but high intensity these energy systems will be predominant especially during the early stages of the exercise period (Figure 2.4).

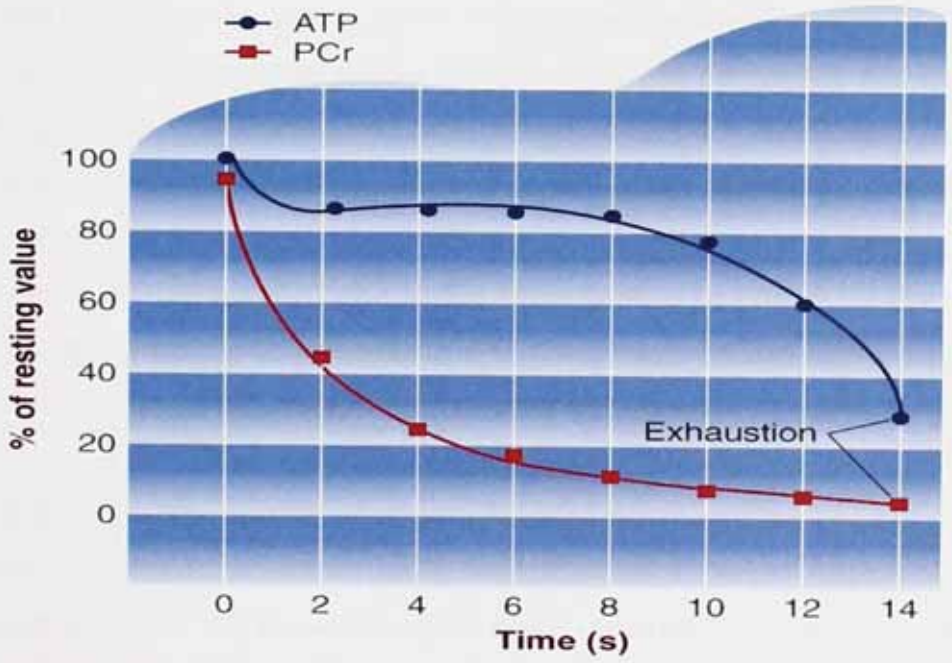


Figure 2.4 ATP and PCr during sprinting (Wilmore and Costill, 1999).

If the energy supply is to last longer than a few seconds then additional sources can be provided by glycolysis and to a lesser extent, during the WAnT, oxidative energy sources. During glycolysis, glucose or glycogen is broken down by a series of enzymatic reactions to produces pyruvic acid. This process yields two molecules of ATP and three when glycogen is used and provides significantly more ATP than from the immediate energy supply but is still limited to 30 – 90 seconds. When oxygen is absent the pyruvic acid is converted to lactic acid which can have a fatiguing effect by the acidification of muscle fibres, for further information see section 2.5.3.

The oxidative energy system can provide considerably more ATP than the anaerobic energy system. In the presence of oxygen pyruvic acid is converted into acetyl coenzyme A (acetyl CoA). The compound enters the Krebs cycle in the mitochondria. The main purpose of the Krebs cycle is to breakdown the acetyl CoA into carbon dioxide and hydrogen atoms. The hydrogen atoms produced are carried to the electron transport chain where they are oxidised to provide energy to phosphorylate ADP and form ATP. At least 38 molecules of ATP can be produced from this process. Although the supply of ATP is much greater from the oxidative energy system it is activated more slowly and produces the energy less rapidly than from the immediate and anaerobic energy systems. Therefore, during short duration high intensity exercise this energy system does not predominate. However, as the duration of the exercise increases this energy system becomes more dominant and Smith and Hill (1991) suggested that during the later stages of a 30 s WAnT test that the oxidative system can provide a significant contribution to energy production. Therefore, during the early stage of short duration high intensity exercise encountered during a 30 s a WAnT the initial contribution to energy is the via the immediate and anaerobic energy system and then towards the latter stages of the test the oxidative energy system contributes substantially to energy production.

2.3 Development of upper body exercise research

Investigation into upper body exercise and different physiological responses compared to leg ergometry has been reported from as early as 1924 by Collett and Liljestrand (1924). Since this publication the majority of arm crank ergometry studies concentrated on comparing physiological responses to lower body exercise such as cardiovascular performance at a given power output

(Secher *et al.*, 1974, Reybrouck *et al.*, 1975, Astrand *et al.*, 1965), metabolism and performance (Karlsson *et al.*, 1975, Pendergast *et al.*, 1979), the effects of cadence on peak physiological responses (Sawka *et al.*, 1983, Weissland *et al.*, 1997) or thermoregulatory responses (Price and Campbell, 1998, Price and Campbell, 2002, Pimental *et al.*, 1984). Furthermore, a number of studies have reported the health benefits of arm crank exercise for cardiac rehabilitation (Fardy *et al.*, 1977), wheelchair based populations (Hjeltnes, 1977, Dicarlo, 1988), increasing high-density lipoprotein cholesterol (Mukherjee *et al.*, 2001, El-Sayed and Younesian, 2005) and reduced rate-pressure product in men with previous myocardial infarction (Franklin *et al.*, 1994).

Since the mid-1980s a number of studies have focused on the physiology of ACE rather than comparing ACE to leg ergometry. Most studies undertaken have been at low submaximal workloads (less than 100 W) and examined the efficiency of arm cranking (Kang *et al.*, 1997, Marais *et al.*, 2002b, Powers *et al.*, 1984). Relatively few studies, other than those addressing development of peak oxygen uptake (VO_{2peak}) protocols, have examined the duration of exercise at or above maximal aerobic capacity (Marais *et al.*, 1999). The mean power outputs associated with such maximal aerobic and high intensity anaerobic arm cranking have been demonstrated through VO_{2peak} and WAnT tests. Power output values for these tests are generally much higher than for the submaximal tests previously noted (~100 W) and can reach up to 1000 W for upper body Wingate anaerobic tests (Smith *et al.*, 2007b, Sawka *et al.*, 1983, Kounalakis *et al.*, 2008). A number of studies have used the Wingate anaerobic test (WAnT) in arm cranking to examine high intensity exercise such as in relation to nutritional interventions for power-based athletes (Aschenbach *et al.*, 2000), comparison between different levels of ability within a specific sport (Evans *et al.*, 1993, Hübner-Woźniak *et al.*, 2006b, Jemini *et al.*, 2006), between sporting and non-sports populations and younger and older men (Marsh *et al.*, 1999) and synchronous and asynchronous WAnTs (Lovell *et al.*, 2011b). Recently standing arm cranking has been applied to the performance of America's cup sailors to help understand their physiological characteristics (Neville *et al.*, 2009, Bernardi *et al.*, 2007). Therefore, performance during an upper body WAnT test has implications for sport, exercise and health although in general, studies have only reported the performance results rather than the mechanisms that contributed to those results.

Recent research using the WAnT has concentrated on assessing the upper body fitness of athletes participating in sailing (Easton *et al.*, 2007), gymnastics (Jemini *et al.*, 2006), martial arts (Franchini *et al.*, 2005, Artioli *et al.*, 2008) and prediction of swimming performance (Invernizzi *et al.*, 2008, Guglielmo and Denadai, 2000). In addition to submaximal intensity ACE being effective training for individuals with spinal cord injury (Dicarlo, 1988, El-Sayed and Younesian, 2005), ACE training can improve walking performance and pain tolerance in patients with symptomatic peripheral arterial disease (Tew *et al.*, 2009, Zwierska *et al.*, 2005) and in the treatment of patients with hypertension (Westhoff *et al.*, 2008). Furthermore, motor coordination and speed of movement required during ACE at submaximal power has been used to predict all-cause mortality in men (Metter *et al.*, 2004) and can also be used as a predictor of cardiovascular and all-cause mortality in an older population with lower limb disabilities (Ilias *et al.*, 2009). Therefore, if arm crank performance is linked to athletic ability and health outcomes further analysis is needed to explain how these adaptations in performance may occur.

The peak oxygen uptake and WAnT tests have not extensively examined the physiological responses associated with arm cranking at high intensities. Furthermore, few studies have examined the biomechanical responses to ACE at either high or low exercise intensities (Hopman *et al.*, 1995, Marais *et al.*, 2004, Bressel *et al.*, 2001, Bressel and Heise, 2004, Mossberg *et al.*, 1999, Frauendorf *et al.*, 1989, Frauendorf *et al.*, 1986, Smith *et al.*, 2008, Zehr and Chua, 2000, Bernasconi *et al.*, 2006). Studies examining muscle activation (EMG) and motion analysis during ACE have been less well studied. Currently only three studies (Price *et al.*, 2007, Bressel and Heise, 2004, Smith *et al.*, 2008) have examined the integration of physiological and biomechanical responses during ACE and these will be discussed later in the chapter. A small number of studies have examined the physiological and biomechanical mechanisms that contribute to the fatigue process resulting in the termination of exercise at high intensities (Hopman *et al.*, 1995, Frauendorf *et al.*, 1989). No studies have reported such responses during upper body WAnTs and during continuous high intensity upper body tests to exhaustion. Examining the physiology and biomechanics at exhaustion will help to develop the understanding of fatigue during upper body exercise; this could have implications for rehabilitation exercises and upper body training for athletes and power output.

2.4 Comparison of physiological responses to upper and lower body exercise

2.4.1 Incremental exercise to exhaustion

When the limit of oxygen uptake is reached during ACE it is normally referred to as peak oxygen uptake (VO_{2peak}). Values are referred to as 'peak' rather than 'maximal' for arm exercise as oxygen uptake is limited by peripheral (local muscular) fatigue rather than central (cardiovascular) fatigue (Magel *et al.*, 1975). Peak oxygen uptake is lower due to a smaller muscle mass (Sawka, 1986, Washburn and Seals, 1984), reduced stroke volume (Astrand *et al.*, 1965) and additional peripheral factors such as, a smaller diffusion area and larger diffusion distance (Calbet *et al.*, 2005). Expected VO_{2peak} values from incremental exercise tests to exhaustion are shown in Table 2.1. Although stroke volume is influenced by the muscle mass recruited and anaerobic threshold (Lepretre *et al.*, 2004), the impact of training status or training on these parameters and upper body exercise, as much as lower body exercise is not known. It has been suggested that the differences in oxygen uptake and heart rate for the legs are due to the rate of oxygen delivery, and for the arms, it is linked to the active muscle mass with the author concluding that there was 'an unidentified peripheral factor' (Warren *et al.*, 1990). Additionally, peak oxygen uptake during ACE is 70% to 75% of that achieved during leg ergometry (Sedlock, 1991, Lyons *et al.*, 2007, Kang *et al.*, 1997). Therefore, upper body exercise has different physiological responses (e.g. heart rate, oxygen uptake) to exercise at the same intensity which suggested that there may be different mechanisms of fatigue present.

Table 2.1 Comparison of VO_{2peak} and heart rate between arm and leg ergometry (mean ± *SD*).

Author(s)	VO_{2peak} ($l \cdot min^{-1}$)		Peak HR ($beats \cdot min^{-1}$)	
	Arm	Leg	Arm	Leg
Davis *et al.*, 1976	2.43 (0.39)	3.68 (0.41)	184 (12)	193 (10)
Kang *et al.*, 1997	2.24 (0.54)	2.98 (0.52)	170 (17)	180 (14)
Lyons *et al.*, 2007	2.20 (0.25)	3.10 (0.38)	No data	No data
Ramonatxo, 1996	2.52 (0.41)	3.17 (0.63)	178 (15)	184 (12)
Rosler *et al.*, 1985a	2.72 (0.13)	3.66 (0.12)	185 (4)	188 (4)
Sedlock, 1991	1.94 (0.57)	2.68 (0.73)	186 (10)	188 (10)
Tulppo *et al.*, 1999	2.44 (0.27)	3.70 (0.47)	178 (11)	188 (13)

2.4.2 Submaximal responses

Oxygen uptake kinetics have been found to be slower (Koppo *et* al., 2002, Smith *et al.*, 2006c) and heart rate and ratings of perceived exertion have been found to greater at the same power output (49 W, 74 W and 98 W) when compared to leg and both leg and arm exercise combined (Eston and Brodie, 1986). Heart rate was also greater for ACE at the same relative exercise intensity (50%, 60% and 70% of VO_{2peak}) when compared to cycle ergometry (Kang *et al.*, 1997). When the absolute power output is matched between upper and lower body exercise at 70% of ventilatory threshold no differences in minute ventilation at low intensities of exercise have been observed if the power output does not elevate blood lactate. At 90% of mode specific ventilatory threshold relative carbohydrate oxidation was significantly greater than leg exercise (Casaburi *et al.*, 1992, Yasuda *et al.*, 2002). At an exercise intensity half way between anaerobic threshold and VO_{2peak} (Schneider *et al.*, 2002) and above ventilatory threshold the response from the arms indicated a greater recruitment of type II muscle fibres compared to leg exercise (Bernasconi *et al.*, 2006) and when the intensity was at 90% of VO_{2peak} (Koppo *et al.*, 2002). During incremental ACE the VO_2 excess has been observed as the result of an increase in trunk and lower body stabilisation (Smith *et al.*, 2006c). However, no specific EMG or biomechanical data has been reported to support this and further studies are required to inform this area of research.

If work efficiency, which excludes resting energy expenditure, is considered then at 50%, 60% and 70% of VO_{2peak} compared to leg exercise then ACE is significantly less efficient (Kang *et al.*, 1997). Compared to leg exercise, arm exercise at 30%, 50%, and 80% of VO_{2peak} utilised more carbohydrate (reflected in a higher lactate output) due to a greater reliance on the anaerobic system (Ahlbory and Jensen-Urstad, 1991). Such unmeasured work, e.g. additional limb movement or limb stabilisation, may be due to the reduction in unmeasured work during ACE at lower intensities and conversely an increase in unmeasured work during high intensity ACE (Kang *et al.*, 1997, Shiomi *et al.*, 2000, Eston and Brodie, 1986) (Ahlborg and Jensen-Urstad, 1991). The increase in unmeasured work for higher intensities remains speculative although a number of studies have suggested it may be due to isometric contraction of arm and trunk muscles (Shiomi *et al.*, 2000, Washburn and Seals, 1984, Bar-Or and Zwiren, 1975, Bernasconi *et al.*, 2006) or the trunk muscles contribution to power generation

(van Drongelen *et al.*, 2009, Stamford *et al.*, 1978). The potential for lower body and torso contribution may be significant during arm exercise. Therefore, upper body exercise is not necessarily limited by the power output of the arms as the torso may contribute to upper body fatigue or aid in power production. Further research targeting the activity of the muscles noted above (EMG) and both lower limb and trunk movement (kinematics) could give a clearer understanding of their contribution to power production and/or unmeasured work during high intensity exercise.

2.4.3 Wingate anaerobic test

The Wingate anaerobic test is a 30 s maximal test and has been widely used in both upper and lower body exercise testing (Winter, 1991, Bar-Or, 1987). Typical values for leg and arm WAnT are shown in Table 2.2 along with a figure of a typical power profile observed (Figure 2.5). The test itself purports to measure maximal and mean power output and fatigue over a short duration of time. However, a standard definition for the variables measured during the WAnT has not been reported in the literature. An accurate definition is important for consistency of reporting data and when comparisons are made to previous literature. From the review of published literature the most frequent term for the 30 s WAnT is to describe it as an 'anaerobic power' test. Tests of a longer duration (greater than 30 s or repeated sprints) are generally referred to as 'high intensity' or 'supramaximal' and those of a short duration (less than 30 s) tend to be referred to as 'sprint' or 'all-out' (Appendix 1).

Table 2.2 Comparison between peak power output from an arm or leg WAnT (mean ± *SD*). Note: *Balmer et al., 2004* used two methods to measure uncorrected peak power output.

Authors	Peak power output (W)			
	Corrected		Uncorrected	
	Arm	Leg	Arm	Leg
Balmer *et al.*, 2004	609 (179)		440 (97)	
			472 (117)	
Bouhlel *et al.*, 2007			720 (133)	1208 (272)
Greer *et al.*, 2006				1049 (192)
Giovani and Nikolaidis, 2012			445 (80)	910 (138)
Guglielmo and Denadai, 2000			527 (79)	
Lutoslawska *et al.*, 2003			667 (243)	844 (167)
Patton *et al.*, 1985				770 (94)
Smith *et al.*, 2007b	629 (169)		507 (109)	
Weber *et al.*, 2006			743 (37)	1055 (42)
Winter *et al.*, 1996		1005 (32)		915 (35)
Zagatto *et al.*, 2008			375 (56)	772 (94)

The WAnT is not necessarily conducted over 30 s duration which may contribute to the differences in definitions for this test (Baker *et al.*, 2001b, Baker *et al.*, 2001a, Stickley *et al.*, 2008, Marquardt *et al.*, 1993, Smith *et al.*, 2007b). A 20 s Wingate test may be used (Marquardt *et al.*, 1993, Smith *et al.*, 2007b) as this can reduce nausea, vomiting and dizziness associated with the 30 s test (Inbar *et al.*, 1996, Stickley *et al.*, 2008, Marquardt *et al.*, 1993). As the peak power output (PPO) is normally achieved within 10 s, the 20 s duration will not affect this measure although there will be changes in mean and especially minimum power output (Inbar *et al.*, 1996). Therefore, the test duration may be influenced by whether the main objective is to measure peak or mean power output. Changes in minimum power output are important as this is one of two variables used to calculate the fatigue index (FI; [PPO 1 s – minimum power output 1 s] / PPO 1 s). Therefore, if peak power output increases and minimum power does not increase by the same amount then the FI will increase. As corrected peak power is greater than uncorrected and both power outputs have

similar minimum power then the FI for corrected peak power output will be greater (Bogdanis *et al.*, 2008). Therefore, it is important that the FI is analysed using corrected and uncorrected data as this will affect the analysis and reporting of the measure of fatigue.

During the WAnT, the energy contribution is predominately anaerobic and therefore performance is largely governed by this energy system (Minahan *et al.*, 2007, Smith and Hill, 1991). However, aerobic metabolism provides a significant contribution to power output with a greater contribution towards the latter half of the test (Smith and Hill, 1991, Medbo and Tabata, 1989, Gastin, 2001). Depending on the type of measurement estimates of the aerobic contribution are between 16% (Smith and Hill, 1991), 19% (Beneke *et al.*, 2002, Bediz *et al.*, 1998), 22% (Micklewright *et al.*, 2006), 28% (Serresse *et al.*, 1988) and 40% (Medbo and Tabata, 1989) during leg exercise. The aerobic contribution during an upper body WAnT has not been established. However, it has been established that there is a greater percentage of type II fibres in the arms and a lower capillary to fibre ratio (Pendergast, 1989, Sawka, 1986), earlier and/or greater recruitment of type II muscles fibres (Ahlborg and Jensen-Urstad, 1991, Koppo *et al.*, 2002, Smith *et al.*, 2006c, Kang *et al.*, 1997) and a high anaerobic energy release measured in ACE against leg ergometry at submaximal intensities (Jensen-Urstad *et al.*, 1993, Koga *et al.*, 1996) and incremental exercise (Schneider *et al.*, 2002). Muscle oxygenation desaturation during an upper body WAnT is less than found during a WAnT performed with the legs and indicates that for the upper body that the aerobic contribution is less than for leg exercise (Kounalakis *et al.*, 2009). A greater anaerobic energy contribution to the WAnT may therefore be assumed for ACE compared to leg ergometry for the same power output.

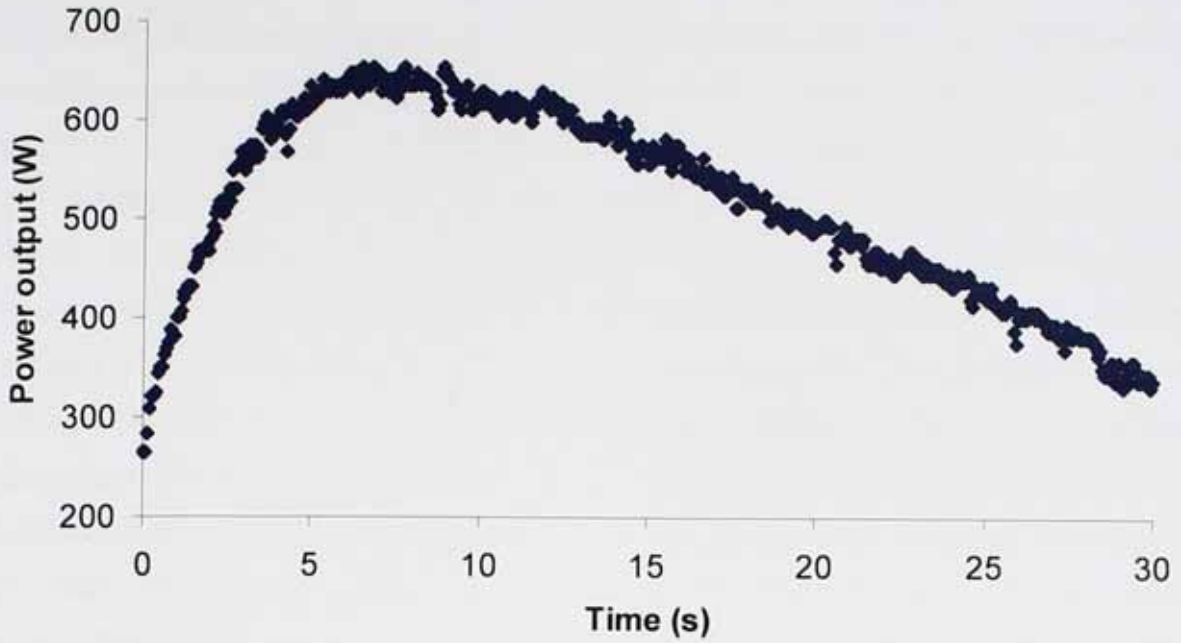


Figure 2.5 Example of a 30 s upper body WAnT at 5% body mass load using a 18 Hertz data interval.

2.4.3.i Wingate test considerations

Previous studies have shown that the WAnT is valid and reliable for assessing power output in leg ergometry (Vandewalle *et al.*, 1987, Bar-Or *et al.*, 1977, Inbar *et al.*, 1996, Bar-Or, 1987) and upper body ergometry in both able bodied (Smith *et al.*, 2007b) and spinal cord injured populations (Jacobs *et al.*, 2005, Jacobs *et al.*, 2003). However, differences exist between studies in terms of the initial cadence used prior to the application of the resistive load, the resistive load applied and the use of corrected and uncorrected power output values. These will be discussed in the following sections.

Starting cadence

The suggested cadence before the mass is applied ranges from a stationary start with the mass already applied (MacIntosh *et al.*, 2003) to achieving maximal cadence prior to the load being applied (Bassett, 1989, Bediz *et al.*, 1998, Inbar *et al.*, 1996). A maximum cadence prior to application of the resistive load results in neuromuscular fatigue prior to the start of the test (MacIntosh *et al.*, 2003) and does not accurately reflect time to peak power (Wright *et al.*, 2007), although a stationary start can be difficult to accelerate the flywheel (Winter and MacLaren, 2001). In general a starting cadence of between 50-70 rev·min^{-1} is recommended (Winter and MacLaren, 2001, Smith and Price, 2007).

Resistive load

The workload, measurement and reporting of values has not been thoroughly investigated (Sawka, 1986, Smith and Price, 2007) which makes comparing results between studies problematic. Although it is acknowledged that a single test cannot optimise for both peak and mean power output (Dotan and Bar-Or, 1983). An extensive review of the literature found no standardised test criteria for upper body maximal intensity exercise since the research by Dotan and Bar-Or (1983) suggested a workload of 6% of body mass and The British Association of Sport and Exercise Sciences guidelines suggest between 3% and 6% of body mass depending on training status (Smith and Price, 2007). The majority of studies either employed a resistive load of 4% body mass (Hubner-Wozniak *et al.*, 2004, Weber *et al.*, 2006, Aschenbach *et al.*, 2000, Biggerstaff *et al.*, 1997) or 5% body mass (Aziz *et al.*, 2002, Lovell *et al.*, 2011b, Buśko, 2011, Smith *et al.*, 2007b). It is not clear whether a 4% or 5% body mass loading produces a significant difference in power output. Therefore, determination of the optimal load will be beneficial to exercise testing procedures and guidelines for testing. Additionally, biomechanical responses to different loads have not been reported and therefore further analysis is required to inform of the possible mechanisms that may result in the different power outputs and FI reported with different WAnT loads.

Corrected and uncorrected power output

Corrected power output takes into account the force required to accelerate the flywheel (Lakomy, 1986, Lakomy, 1985, Bassett, 1989) and is useful when examining acceleration and is applicable to a sprint start or finish in sport. Uncorrected peak power output occurs when maximal flywheel velocity is reached (Vandewalle *et al.*, 1985b, Lakomy, 1986) and may be useful when analysing maximal limb cadence and load. To calculate the load required for corrected power output optimisation is not required (Winter *et al.*, 1996, Martin *et al.*, 1997, James *et al.*, 2007b) although in upper body WAnT, due to the variability in upper body power output, more than one test may be needed (Vanderthommen *et al.*, 1997). A number of tests using different body mass loadings may be required before optimal uncorrected peak power output is achieved (Winter *et al.*, 1996, Dotan and Bar-Or, 1983). When analysing WAnT power output the results should indicate whether the data is corrected or uncorrected (Lakomy, 1985) and the sample time which influences power output,

as the sample time increases peak power output is reduced (Lakomy, 1986, Winter, 1991). The original WAnT and early studies (Bediz *et al.*, 1998, Bar-Or *et al.*, 1977) used a 5 s averaging period to calculate performance indices. With more powerful computing methods and further refinement of the test reported values can now be analysed per second or fractions of a second (typically 0.5 s and 0.25 s) (Lakomy, 1986). Corrected peak power output is greater and occurs earlier than uncorrected peak power output (Lakomy, 1986, Lakomy, 1985, Balmer *et al.*, 2004, James *et al.*, 2007b) although corrected and uncorrected mean power output over 30 s is not significantly different (Balmer *et al.*, 2004).

The WAnT model allows for manipulation of resistive load in determining performance (Goosey-Tolfrey *et al.*, 2006, Jacobs, 2003, Johnson *et al.*, 2004, Dotan and Bar-Or, 1983). In general, for lower body WAnT the greater the resistance results in a slower cadence and less resistance results in faster cadences and therefore differences in power output. As such manipulation of resistive load would enable analysis of physiological and biomechanical responses to fatigue at different cadences and power outputs and help to understand the requirements to training adding to the current paucity of published information in this area.

2.4.4 Continuous high intensity exercise

Very few studies have investigated the physiology and biomechanics of ACE at higher intensities continued to volitional fatigue rather than stopping after a predefined period of time. Studies have typically investigated transitions in VO_2 kinetics due to changes in crank rate (Smith *et al.*, 2006c), prior with or without the legs active (Ogata and Yano, 2005) and the influence of prior arm exercise (Koppo and Bouckaert, 2005). To date no studies have reported the physiology or biomechanics during a series of increment high intensity ACE test to volitional exhaustion.

During incremental arm cranking the small amount of muscle mass, compared to the legs, may be limiting to performance, rather than the oxygen transport system (Bar-Or and Zwiren, 1975, Muraki *et al.*, 2004, Bhambhani, 2004). Muraki *et al.*, (2004) observed that the triceps brachii experienced muscle deoxygenation at 50% of VO_{2peak} despite an adequate oxygen supply, indicating that the limiting factor for exercise may be the triceps brachii ability to extract

and/or utilise oxygen The lower ability to extract and/or utilise oxygen was related to a lower ratio of slow twitch muscle fibres, which promoted the use of the anaerobic energy supply for this muscle group (Muraki *et al.*, 2004). This is illustrated by the local fatigue mentioned in section 2.2.1. At exercise intensities at and above VO_{2peak} such fatigue may be accentuated. For example, local fatigue from gripping the crank handles (pseudo-occlusion) may result in isometric contractions and impair venous return. As exercise intensity increases muscle grip may increase, which would further impair venous return (Koga *et al.*, 1996, Schneider *et al.*, 2009, Davis *et al.*, 1976). Further study of forearm muscle activation at various intensities may add to the limited knowledge in this area.

During continuous high intensity exercise for the lower body there is a severe exercise domain in which maximal oxygen uptake occurs. (Caputo and Denadai, 2008, Xu and Rhodes, 1999). There is an upper limit and lower limit to the domain in which VO_{2max} cannot be achieved (Hill *et al.*, 2002). With the upper limit fatigue occurs before VO_{2max} can be reached. The relationship of power and time fits a hyperbola (Figure 2.6) i.e. as intensity increases time to achieve VO_{2max} decreases. Whether, this relationship exists in upper body exercise is not clear as at present studies have only indicated time to exhaustion at peak oxygen uptake in swimmers and kayak paddlers (Billat *et al.*, 1996, Leveque *et al.*, 2002, Fernandes *et al.*, 2008b). Time to exhaustion in the severe exercise domain has not been reported for ACE. Given the physiological and biomechanical difference to lower body exercise this warrants further investigation, and may aid in informing upper body training programmes. As previously stated VO_{2peak} and submaximal oxygen consumption compared to WAnT and high intensity arm cranking is relatively well investigated. A number of studies have examined physiological responses at intensities below VO_{2peak} (Jensen-Urstad, 1992, Kang *et al.*, 1999). Very few studies have examined responses at or above VO_{2peak} (170% and 200% of VO_{2peak}; Tabata *et al.*, 1997, 110% and 120% of maximal power; Marais *et al.*, 1999). These are isolated studies and so far no study has examined responses below, at and above peak oxygen consumption. A study linking various exercise intensities (e.g. 80%, 90%, 100% and 110% of VO_{2peak}) would be useful as it would enable comparisons of and differences in fatigue at a range of high intensity exercise intensities to be examined.

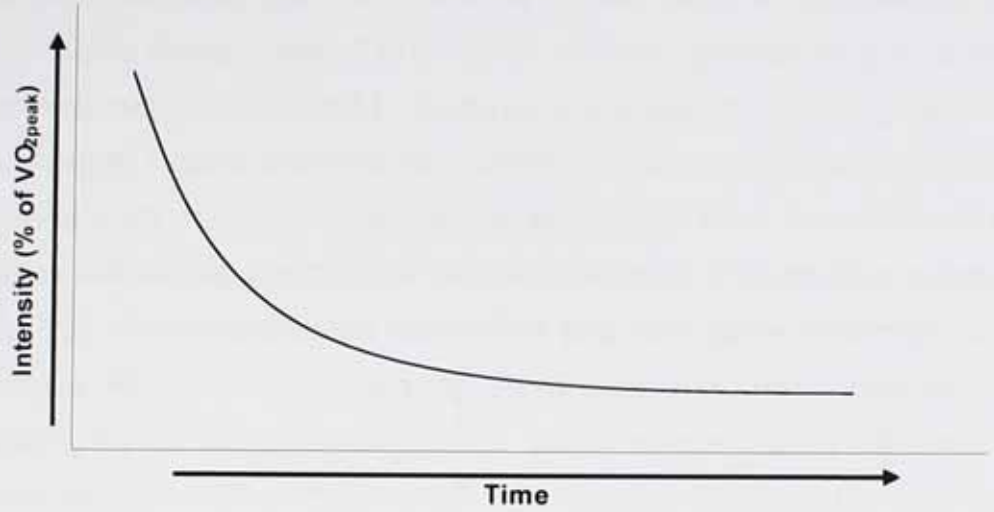


Figure 2.6 Schematic diagram with hypothetical data showing the relationship between exercise intensity and time with the curved line representing upper and lower point to achieve peak or maximal oxygen uptake.

2.5 Physiology of fatigue

There are a number of definitions of fatigue. For example fatigue has been regarded as;

'a decrease in performance and can be both physical and mental (Szygula *et al.*, 2003, Fitts, 1996)

'a continuous process that transforms the functional state, with exhaustion being the point at which exercise is terminated.' (Kay *et al.*, 2001)

'the failure to generate or maintain the required or expected force or power output, resulting from muscle activity and reversible by rest.' (Sargeant, 1994)

Fatigue is specific to the exercise being undertaken (McLester, 1997, Fitts, 1996) For the exercise intensities examined in this thesis is taken as either a reduction in power from maximal to minimum or the inability to maintain a given exercise intensity.

Fatigue may be categorised as central or peripheral fatigue (Sahlin, 1992, Davis, 1995, Bigland-Ritchie, 1981). Central fatigue is the inability to generate the drive from the central nervous system whereas peripheral fatigue is the inability

to generate a contraction in the peripheral nerve or contracting muscle (Sahlin *et al.*, 1998, Davis, 1995, Taylor *et al.*, 2000). Central fatigue is relatively unexplored (Davis, 1995). It may have a role in high intensity exercise through a reduced neural drive (Green, 1997), as suggested during six 1-min sprints during a 60 min cycle test (Kay *et al.*, 2001). Here reductions in efferent drive observed during sprints 2-4 and were seen as a protective mechanism via central control. Additionally, the discomfort and pain of the exercise may contribute to fatigue with the longer duration the greater the impact (Sahlin, 1992, Katch and Henry, 1972, Taylor *et al.*, 2000). Local fatigue during high intensity exercise may be the result of number of physiological mechanisms such as, afferent feedback, interference from metabolic by-products, fibre type rather than one isolated factor (Green, 1997). Recent molecular data has indicated that the muscle proteins troponin and tropomyosin are disrupted by the by-productions of metabolism (Debold, 2012). The potential fatigue mechanisms are explored below.

2.5.1 Components of fatigue

In high intensity exercise the increase of inorganic phosphate from the breakdown of creatine phosphate interferes with sarcoplasmic reticulum Ca^{2+} handling e.g. inhibition of Ca^{2+} uptake or release and also with the cross-bridge cycle (Westerblad *et al.*, 2002, McLester, 1997, Bangsbo *et al.*, 1996). Furthermore, the metabolic by-products of contractions may affect the feedback from group III-IV afferents that are sensitive to metabolic products and ischaemia (Taylor *et al.*, 2000). This may be important to upper body exercise as previous research suggested that in response to a sustained 2 min maximal voluntary contraction there was a reduction in triceps brachii and an increase in biceps brachii EMG activity (Martin *et al.*, 2006). Additionally, during static exercise, upper limb postural muscles (e.g. infraspinatus) may increase EMG amplitude in relation to postural fatigue and arm position more significantly than during dynamic force production (Rudroff *et al.*, 2007). No studies so far have reported changes in EMG amplitude in these muscles during dynamic upper body exercise. However, WAnT testing of the lower limb indicated an accumulation of metabolite and/or reduced afferent command does not alter EMG amplitude due to a constant electrical input (Rana, 2006, Hunter *et al.*, 2003).

Muscle performance is influenced by the fibre types recruited, fast twitch (FT) fibres are able to produce more power resulting in a high concentration of

lactate, a lower pH and greater concentration of Pi. Therefore, FT fibres are potentially more susceptible to fatigue during tests such as the WAnT and resulting in a greater decline in peak power output (Bar-Or *et al.*, 1980, Mannion *et al.*, 1995, Fitts, 2008). Of the few studies available comparing upper and lower body FI during the WAnT (Weber *et al.*, 2006, Zagatto *et al.*, 2008) upper body exercise does appear to have an increased FI compared to lower body exercise (Table 2.3).

Table 2.3 Comparison of fatigue indexes (%) from arm and leg Wingate anaerobic tests (mean ± *SD*).

	Fatigue index (%)	
Authors	Arms	Legs
Franchini *et al.*, 2005	48 (8) Elite judo	45 (11) Non-elite judo
Guglielmo and Denadai, 2000	42 (7)	
Hawley and Williams, 1991	26 (10) Swimmers	
Kounalakis *et al.*, 2009	49 (10) Athletes	55 (9) Students
Stewart *et al.*, 2011		58 (14)
(Stickley *et al.*, 2008		41 (10)
Üçok *et al.*, 2005		53 (6) Corrected PO
Weber *et al.*, 2006	63 (1) Corrected PO	52 (2) Corrected PO
Zagatto *et al.*, 2008	49 (5) Table tennis	43 (6)

During the WAnT the initial loss of power following attainment of peak power output is primarily governed by the speed of ATP regeneration (the first 5 s) and the considerable depletion of phosphocreatine (Sahlin *et al.*, 1998). From peak power output to the end of the test at 30 s would represent fatigue i.e. the ability or inability to sustain peak power output. Using EMG and kinematic analysis could aid in understanding the effects of fatigue by analysing changes in muscle activation and limb movements at specific time points. Furthermore, different

resistive loads may evoke different fatiguing effects. Thus, the examination of responses (EMG and kinematics) at difference intensities is needed have not been reported in the literature.

2.5.2 Fatigue during high intensity lower body

At intensities above and below VO_{2max} there is a curvilinear relationship between intensity and endurance time (known as the force-velocity time curve; Figure 2.7). The force velocity time curve demonstrates as the exercise intensity increases (force) the performance duration (velocity) is reduced. With appropriate training the curve shifts to the right (Sahlin, 1992). If this is due to a technique change with performance then biomechanical analysis may help to explain adaptations to training. Assessing how ACE technique changes at fatigue with training may address this aim.

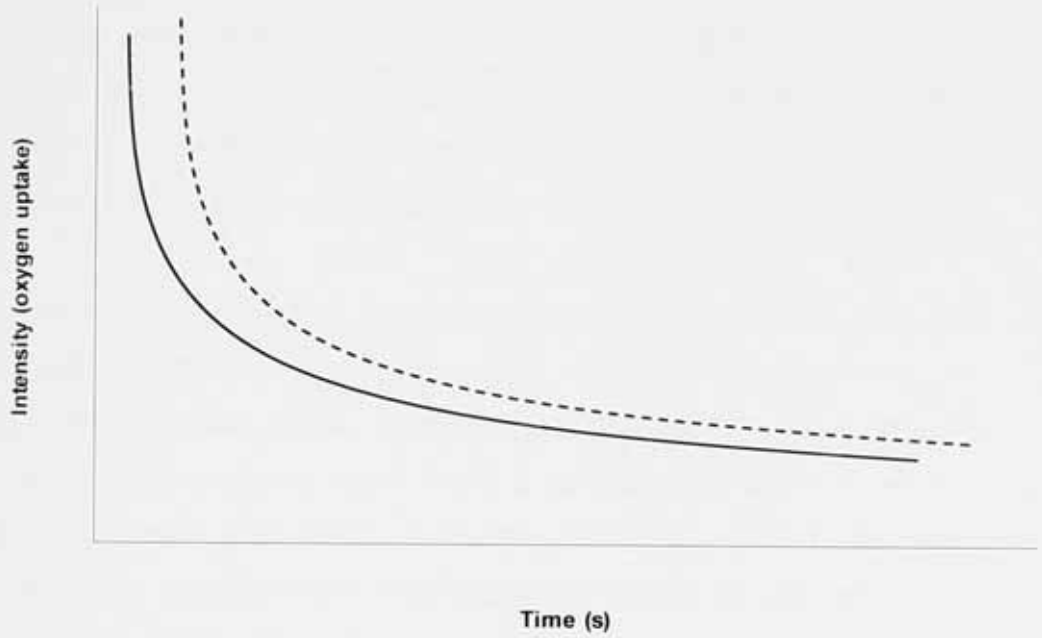


Figure 2.7 Schematic diagram with hypothetical data showing the relationship between intensity (force) and time (velocity). The curved line representing upper and lower point between oxygen uptake and duration (time; s) of exercise. The dashed line represents a shift to the right in the curve as a response to training.

The contributors to fatigue may change as the exercise intensity decreases, for example, high intensities, muscle and ATP recruitment and at lower intensities substrate stores, dehydration and motivation (Davis, 1995, Korge, 1995, Mannion *et al.*, 1995, McLester, 1997). To date studies have not examined the

relationship between exercise intensity and fatigue during upper body exercise using ACE over a range of intensities. An analysis of change in technique may help to understand the mechanisms of fatigue in a relatively small muscle mass compared to lower body exercise. Most studies of lower body exercise have that have linked biomechanics and physiology and have focused on running economy and therefore this topic is relatively unexplored in the literature for ACE. Furthermore, performance at different intensities of exercise may evoke different fatiguing effects, thus the examination of responses at difference intensities is needed.

2.5.3 Fatigue during high intensity upper body exercise

As previously discussed there are a number of theories regarding fatigue. The type of fatigue experienced, central or local, is often duration/intensity based and can be manipulated by speed of limb movement and cadence. As with most areas of upper body exercise the mechanisms of fatigue remain relatively unexplored and given the differences in physiology when compared to lower body exercise, these differences should be examined to determine differences in fatigue during upper and lower body exercise.

As noted in sections 2.4.1, fatigue in incremental upper body exercise may be limited by local muscular fatigue over central fatigue (Sawka, 1986, Mossberg *et al.*, 1999, Franklin, 1985, Enders *et al.*, 1994). The greater recruitment of type II muscle fibres during upper body exercise may result in exercise termination due to neuromuscular fatigue (Bernasconi *et al.*, 2006). Neuromuscular fatigue in ACE may be due to the build-up of metabolic by-products (e.g. inorganic phosphate, H^+ and lactate) interfering with the process of muscle contraction (Bernasconi *et al.*, 2006, Taylor *et al.*, 2000). Further specific studies examining motion analysis and EMG may improve understanding of the mechanisms contributing to arm fatigue (Section 2.7 & 2.8).

Activation of a smaller muscle mass such as during upper body exercise may concentrate perceptions of fatigue more so than during leg exercise at 70% of VO_{2peak} (Kang *et al.*, 1998). However, perception of fatigue at higher intensities of upper body exercise, and the determination of whether at higher intensities oxygen uptake and ratings of perceived exertion can be associated to the same extent as lower body fatigue remains to be reported. During maximal

incremental arm there may be an additional afferent feedback from the arms and torso (Ishida *et al.*, 1994) which may increase neuromuscular activity and, due to the increased load on the respiratory system (Ramonatxo, 1996), increase perceptions of fatigue. These changes in perception, or actual fatigue, appear to be affected by cadence. For example at 50 rev·min^{-1} there is greater local RPE than central RPE and participants fatigued earlier in a VO_{2peak} test at this cadence than when compared to 90 rev·min^{-1}, even though ventilation rate increased with cadence (Smith *et al.*, 2006b). Therefore, perception of fatigue may be greater and more limiting than during lower body exercise.

In comparison to lower body exercise the possibly greater anaerobic contributions associated with upper body exercise such as greater proportion of fast twitch fibres (Muraki *et al.*, 2004, Kounalakis *et al.*, 2009) would increase the accumulation of inorganic phosphate and as the duration of exercise increased the accumulation of ADP would also contribute to the fatigue process (McLester, 1997). Additionally, late in exercise, and especially in ischaemic conditions, pain develops which may contribute to sensation of fatigue through local RPE and the termination of exercise (Taylor *et al.*, 2000). In isometric conditions it is possible that local muscular fatigue may be accentuated at greater exercise intensities thus increasing the potential for localised (forearm) muscle ischaemia. At comparable exercise intensities local and central RPE are greater in the arms than the legs (Marais *et al.*, 2001). Therefore, studies reporting this difference may aid in the understanding of the mechanism of fatigue associated with ACE.

Electroencephalograph (EEG) activity following incremental ACE to exhaustion has been found to differ when compared to cycle and treadmill ergometry. These responses suggest the local muscular fatigue experienced and the inexperience of the participants to this activity contributed to the greater local fatigue (Schneider *et al.*, 2009). The change in EEG may indicate differences in central drive and fatigue that could be reflected in changes in EMG although this has not been reported in the literature for ACE and requires further studies to investigate this possible cause of fatigue and possible changes in EMG after habituation to ACE.

A number of suggestions for the mechanisms of fatigue have been proposed and these can be examined through physiological and biomechanical methods as well as perceptual methods such as RPE which may aid detection of different types of fatigue. Once the key factors contributing to fatigue, analysed within the scope of this thesis, have been identified it will be useful to examine how these factors can be manipulated by exercise intensity and training. Training may result in reduced fatigue and therefore improve performance such as maximal power output or time to exhaustion. This could also facilitate/optimise the volume of work that can be achieved in the context of clinical rehabilitation which could have implications for exercise adherence.

2.6 Upper body training studies

2.6.1 Upper body aerobic training studies

There are few studies on the effects of arm crank training compared to leg training especially in non-clinical or healthy populations. However, there are also cross sectional studies involving upper body athletes and how their trained state differs from untrained participants. Increased lactate release and greater aerobic output in trained rowers compared to untrained individuals at volitional fatigue (Volianitis *et al.*, 2004a) has been shown. Furthermore, arm crank training has produced significant improvements in central and peripheral circulatory function and increase in time to exhaustion (Loftin *et al.*, 1988). In males, with quadriplegia, eight weeks of arm crank training improved cardiopulmonary functions and wheelchair propulsion endurance (Dicarlo, 1988) and five weeks training improved submaximal wheelchair exercise (Sedlock *et al.*, 1988). Training programmes of ten weeks (Magel *et al.*, 1978) and five weeks (Clausen *et al.*, 1973) of arm cranking training resulted in significant improvements in VO_{2peak} (16% and 10% increase respectively) which was reflected in a significantly enhanced a-vO_2 difference, a peripheral rather than a central adaption. There was no significant change in stroke volume, cardiac output or heart rate (HR). Although, Clausen *et al.*, (1973) found a reduction in HR at a submaximal exercise intensity which may indicate a central adaptation at submaximal loads. This suggested that peripheral adaptations can be adapted separately and may be more important for upper body exercise than central adaptations. Helge (2010) reviewed low-intensity arm and leg training studies below VO_{2peak} and suggested that there are specific adaptations to the arm and leg and that adaptations are peripheral rather than central for the upper body.

Adaptations to high intensity ACE using an anaerobic training programme would be beneficial as lower body high intensity training studies have shown performance improvements in time to exhaustion (Burgomaster *et al.*, 2005), maximal oxygen uptake (Gibala *et al.*, 2006) and WAnT power output (Ziemann *et al.*, 2011). However, there are no reports of training studies that have incorporated high intensity upper body training.

2.6.2 Upper body strength training studies

Although there is a lack of research reporting the effects of high intensity upper body exercise training, the effects of conventional resistance training or circuit training have been reported. A study involving four weeks strength training (four upper body exercises, three sets of ten repetitions at 60% of one repetition maximum) observed increases in strength and VO_{2peak} in previously sedentary men (Swensen *et al.*, 1993). Suggested reasons for an increase in performance were increased recruitment of muscle fibres and/or more efficient coordination. Although not measured, this training may have improved technique and therefore biomechanics which could be measurable by motion analysis and EMG before and after training. The authors recommended a training study of longer duration but such effects may be cancelled out by muscle hypertrophy reducing mitochondrial and capillary density (Swensen *et al.*, 1993). Therefore, this study indicated that strength/power training could improve ACE peak oxygen uptake. As ACE training can have a positive impact on performance through reducing local fatigue (Helge, 2010), a training study examining how technique potentially contributes to a reduction in local fatigue would be informative. Any changes in fatigue indicated by a reduction in the difference between local and central ratings of perceived exertion may indicate through local and central RPE any physiological and/or biomechanical adaptations.

2.6.3 Comparing the physiological responses between untrained individuals and trained upper body athletes

The differences in leg compared to arm exercise responses may be in part due to the relatively untrained state of the arms, i.e. lack of use of the arms in everyday activities when compared to the legs (Yasuda *et al.*, 2002, Koga *et al.*, 1996, Clausen *et al.*, 1973, Davis *et al.*, 1976). However, it has been shown that at submaximal exercise intensities (30%, 50% and 80% of VO_{2peak}) lactate release is similar between untrained and arm-trained athletes (Jensen-Urstad, 1992)

which may be related to similar circulatory adaptations in the arms regardless of training status. It is likely that lactate accumulation could be changed by undertaking a period of ACE training and the respiratory exchange ratio (RER) could be reflective of changes in muscle pH and bicarbonate buffering of lactate acid (Casaburi et al., 1992). Although not specifically ACE trained, during maximal arm cranking trained rowers were found to have a threefold increase in lactate release compared to untrained subjects, additionally the rowers had a higher arm blood flow and larger oxygen extraction (Volianitis *et al.*, 2004a).

The two studies noted above examined training status in relation to peak oxygen uptake or during low intensity exercise (30–90 W). These studies were not concerned with measuring EMG responses to investigate changes in muscle activation, or motion analysis to analyse changes in ACE techniques and whether these can reduce fatigue and improve ACE performance. The use of physiology and biomechanics would aid in analysing how adaptations to training could improve ACE performance during high intensity anaerobic upper body exercise.

2.7 Motion analysis

Despite being recommend as an area of research by Bar-Or and Zwiren (1975) and later by Inbar *et al.*, (1996, p.75) motion analysis for ACE is considerably under reported in the literature. An extensive literature search revealed studies have examined motion analysis relating to upper body sport or exercise and these will reviewed. In able-bodied participants undertaking 7 weeks of wheelchair training improvements in mechanical efficiency and metabolic cost of the experimental group where likely to be the result of significant increased stroke angle observed compared to the control group (de Groot *et al.*, 2008). Similarly, during wheelchair ergometry, wheelchair-dependent participants where more mechanically efficient than able-bodied participants (Brown *et al.*, 1990)which suggests that upper body exercise performance can be improved by specific training. During submaximal one minute handcycling in nondisabled participants, trunk range of motion was observed to be significantly greater in asynchronous mode compared to synchronous mode (Faupin *et al.*, 2011). As asynchronous handcycling is comparable to ACE (Faupin *et al.*, 2011) it is probable that trunk function is important to ACE performance and requires further study.

Two studies have examined motion analysis during upper body exercise (Price *et al.*, 2007, Bressel and Heise, 2004). Differences in wrist flexion and muscle activity at a low exercise intensity (25 W) where observed between forward and reverse arm cranking (Bressel and Heise, 2004) whereas differences in shoulder range of motion and trunk angle were observed at VO_{2peak} during ACE at different cadences (Price *et al.*, 2007). Both these studies showed technique difference with exercise intensity. However, there are no reported studies involving motion analysis of high intensity anaerobic upper body exercise. Given the extensive use of this test in a variety of settings it would appear that further research may be beneficial to examine performance and the effect of technique on fatigue. This lack of literature may be an indication of the methodological problems associated with the high speed of movement in high intensity exercise tests such as the WAnT.

2.7.1 Biomechanical changes and fatigue

Previous studies suggest that fatigue changes movement patterns and can be detected by motion analysis in runners during a treadmill run to exhaustion (Millet *et al.*, 2010). Furthermore, inducing hip abductor fatigue prior to treadmill running increased the changes in knee position (Geiser *et al.*, 2010). Changes in joint movement and position due to fatigue may be the result of the fatiguing process itself through an impaired ability to detect movement i.e. as an individual fatigues greater limb/torso movements are required before those movements can be detected (Taylor *et al.*, 2000). Shoulder external rotation has been shown to significantly increase due to fatigue desensitising the muscle mechanoreceptors (Lee *et al.*, 2003a). Changes in kinematics via alterations in crank length can alter power production at a cadence of 120 $rev \cdot min^{-1}$ (Barratt *et al.*, 2011). After a fatiguing WAnT lower body test metabolic fatigue may weaken dynamic knee joint stability (increase movement), and training may help control body movement and lessen the chances of injury (Ortiz *et al.*, 2010). Given the likely extensive contribution of the shoulder muscles in ACE performance an investigation of the kinematic responses is needed to inform us of technique changes or limitation to movement patterns or force production and whether the forms of fatigue differs over a range of resistive loads observed for lower limb studies is applicable for upper body exercise

A previous study of VO_{2peak} during ACE (Price *et al.*, 2007) suggested that the biomechanics of ACE change due to both cadence and exercise intensity. Further investigation is needed when cadence is set at the current BASES and literature recommendation of 70 rev·min^{-1} (Price *et al.*, 2007, Smith and Price, 2007) and how different intensities at 70 rev·min^{-1} affect the motion of the limbs and body during ACE performed to volitional fatigue. This may help explain how biomechanical variables (i.e. technique) have an influence on power production and fatigue, as this represents/describes the muscle movement path which is affected by muscular activity and fatigue.

At low intensity (50%-60% of VO_{2peak}) at 50 rev·min^{-1} respiratory frequency synchronized with arm movement more than leg exercise (Vokac *et al.*, 1975). Whether this could influence high intensity ACE performance is not clear, although later studies on optimal cadence for peak incremental ACE testing have suggested that cadences below 70 rev·min^{-1} were not optimal for performance (Smith *et al.*, 2001, Price and Campbell, 1997, Price *et al.*, 2007, Sawka *et al.*, 1983).

2.8 Muscle activation

Although power can be recorded by the ergometer being used and cardiorespiratory and motion analysis add to the picture of how this power is being produced, muscle activation can provide a more detailed analysis of individual muscle or muscles activation and time of activation and indicate how different exercise intensities alter these parameters within the muscle(s) being studied. As for motion analysis, muscle activation studies during ACE are not extensively reported in the literature and further examination of this area is therefore required. The available literature pertaining to ACE will be reviewed below.

2.8.1 Muscle activation during arm crank ergometry

Reflexes of the upper limb have been studied and indicated amplitudes changed for the first dorsal interosseus, carpi ulnaris (flexor and extensor), brachioradialis, biceps and triceps brachii and deltoid (anterior and posterior) throughout the duty cycle (Zehr and Chua, 2000), therefore muscles are activated at different times during the duty cycle. Due to the method of analysis there was no statistical analysis performed on the differences in magnitude. In a

later study, during unloaded ACE, the biceps and triceps brachii, deltoid (anterior, posterior and medial), erector spinae (cervical, thoracic, lumbar) and carpi radiallis (flexor, extensor) showed significant differences in EMG magnitude between certain clock positions for each individual muscle (Klimstra *et al.*, 2011). Therefore, each individual muscle is not activated to the same magnitude during the whole of the duty cycle. For Klimstra *et al.*, (2011) not all muscles showed the same duration of maximal activation, for example the triceps brachii was not activated for as long a duration as the medial deltoid.

When a resistive load is applied to the ACE at increasing low intensities (15W, 30 W and 45 W) EMG activation was found to increase in four sites including the external oblique and rectus abdominis regardless of whether participants were conventionally seat or seat on a stability ball (Marks *et al.*, 2012). Additionally, using the stability ball significantly increased oxygen uptake and rectus femoris activation over sitting on a chair. This study indicated that muscle activation during ACE increased with resistive load and a stable position for the lower limbs is need to accurately access upper body work measured via oxygen uptake. Increased EMG activation was observed in males and females during one arm ACE with power output between 5 W and 35 W (Frauendorf *et al.*, 1986). This relationship was found at higher intensities by Marais *et al.*, (2004) via muscle activation in the biceps and triceps brachii at intensities from 20% to 80% of peak power out, and found that muscle activation increased with work load. The EMG responses during sub-maximal and asynchronous ACE showed triceps brachii to be activated for 50% of the duty cycle and the rectus abdominis to act only as a stabiliser (Hopman *et al.*, 1995). However, Hopman *et al.*, (1995) only analysed the EMG data descriptively not statistically and a more detailed analysis of muscle patterns of activation would aid understanding in this area. As part of an examination of ACE hand grip position Bressel *et al.*, (2004) found that the triceps brachii at 25 W was activated for 52% of the time which would support the 50% observation of Hopman *et al.*, 1995.

During constant load exercise between ventilatory threshold and VO_{2peak}, for 6-min, increased muscle activation has observed (biceps, triceps, deltoid and infraspinatus) which could be linked to changes in oxygen uptake and recruitment of additional type II muscle fibres (Bernasconi *et al.*, 2006). Further recommendations were made for the study of EMG, in relation to handgrip and

torso and shoulder stabilisers (Bernasconi *et al.*, 2006). Smith *et al.*, (2007a) observed that during submaximal ACE engagement of leg, torso and arm muscles occurred and activation increased with load, especially in the prime movers (biceps and triceps). The above authors suggested further investigation into muscle activation at higher exercise intensities than the 50 W and 100 W studied with recommendations for measurement of a number of additional muscles including the torso.

During incremental ACE to exhaustion were differences in EMG activation between upper body trained and non-upper body trained participant (Frauendorf *et* al., 1989). Swimmers showed an increasing level of EMG activity in upper body sites with an increasing load, where as there were no significant changes in EMG activity for trained runners (Kilen *et al.*, 2012) and untrained males (Frauendorf *et al.*, 1989). This observation suggests upper body athletes exhibit a different muscle recruitment response during ACE to lower body athletes and this is likely to be due to the their upper body training. At present no studies have investigated EMG and ACE during the WAnT, at high intensity constant load exercise to volitional exhaustion or following adaptations following ACE training. Such studies would assist in providing a comprehensive analysis of ACE continued to volitional fatigue and aid in the understanding of contributory muscle(s) and changes in technique to performance.

2.8.2 Muscle activation and fatigue

Lower limb EMG studies may give an indication of how fatigue affects muscle force and recruitment during ACE. For example during cycling to exhaustion at 80% of maximal power output, changes in movement patterns due to fatigue resulted in compensatory increases and earlier recruitment of additional muscles (hip extensor) to attenuate the loss of force production of knee extensor muscles (Dorel *et al.*, 2009). In addition different types of exercise that induce neuromuscular fatigue (short duration; repeated squats and submaximal cycling) have been shown to alter the biomechanical response to landing performance (James *et al.*, 2010). Whilst comparing incremental asynchronous versus synchronous ACE to volitional exhaustion, Mossberg *et al.*, 1999, suggested that the triceps brachii fatigue was likely to contribute to fatigue and more so in synchronous ACE. To provide further information additional muscles contributing to ACE power output require further study as only the triceps brachii and anterior

deltoid was included in the Mossberg *et al.,* (1999) study. Therefore, fatigue induces change in these patterns of muscle recruitment and/or changes in kinematics. Whether these changes exist in upper body ACE remains to be investigated.

2.9 Summary

The physiological responses comparing upper and lower body exercise have observed differences in a number of variables such as oxygen uptake, local fatigue and ratings of perceived exertion. Although upper body exercise does exhibit some similar responses in comparison to leg exercise, such as responses to exercise at 100% of VO_{2peak} - although the time to exhaustion is different, less is known about anaerobic upper body work, especially during constant work tests. Furthermore, in these scenarios studies examining biomechanics during lower body activities have indicated that technique is related to fatigue. Therefore, the following areas of research were undertaken;

The aims of this thesis will be realised through a series of three studies. Study 1 will examine the biomechanical and physiological responses to maximal intensity 30-s arm cranking. The model used will involve the manipulation of cadence and subsequently power output. The first study will also aim to determine an optimal protocol for maximal intensity 30-s arm cranking that elicits maximal performance. Study 2 will examine the biomechanical and physiological variables associated with fatigue during continuous high intensity upper body exercise. The model used will enable the examination of fatigue at a constant cadence but different exercise intensities. The second study will also attempt to determine the optimal exercise intensity for the assessment of continuous high intensity upper body exercise performance. Studies 1 and 2 will therefore examine the limiting factors of different types of high intensity upper body exercise from physiological and biomechanical perspectives. The final study will determine the effect of anaerobic training on physiological and biomechanical responses in order to assess how these limiting factors are affected or potentially offset by training.

2.10 Hypothesis

Null hypothesis (Ho_1): There will be no significant difference in performance measures with changes in resistive load during a 30-s Wingate anaerobic test.
Alternative hypothesis (H_1): There will be a significant difference in performance measures with changes in resistive load during a 30-s Wingate anaerobic test.

Null hypothesis (Ho_2): There will be no significant difference in biomechanical and physiological responses with changes in load during a 30-s Wingate anaerobic test.
Alternative hypothesis (H_2): There will be no significant difference in biomechanical and physiological responses with changes in load during a 30-s Wingate anaerobic test.

Null hypothesis (Ho_3): There will be a significant difference in cardiorespiratory and biomechanical responses with changes in exercise intensity during high intensity exercises completed to volitional exhaustion.
Alternative hypothesis (H_3): There will be no significant difference in cardiorespiratory and biomechanical responses with changes in exercise intensity during high intensity exercises completed to volitional exhaustion.

Null hypothesis (Ho_4): There will be a significant difference in performance measures with changes in exercise intensity during high intensity exercises completed to volitional exhaustion.
Alternative hypothesis (H_4): There will be a no significant difference in performance measures with changes in exercise intensity during high intensity exercises completed to volitional exhaustion.

Null hypothesis (Ho_5): There will be a significant difference in physiological and biomechanical responses measured via a Wingate anaerobic test and tests of high intensity exercise completed to volitional exhaustion following a 6-week training programme.
Alternative hypothesis (H_5): There will be no significant difference in physiological and biomechanical responses measured via a Wingate anaerobic test and tests of high intensity exercise completed to volitional exhaustion following a 6-week training programme.

Null hypothesis (Ho_6): There will be a significant difference in performance measures for a Wingate anaerobic test and tests of high intensity exercise completed to volitional exhaustion following a 6-week training programme.
Alternative hypothesis (H_6): There will be a no significant difference in performance measures for a Wingate anaerobic test and tests of high intensity exercise completed to volitional exhaustion following a 6-week training programme.

Chapter 3

General methods

3.1 Recruitment, ethics and testing considerations

Participants were recruited via posters, email and face to face meetings. All participants were provided with a participant information sheet prior to beginning each study and completed an informed consent form (Appendix 2) along with a pre-test medical questionnaire prior to each exercise session (Appendix 3). Any participants known to have high blood pressure (greater than 139/89 mmHg; Stage 1 hypertension; Pescatello *et al.*, 2004) or taking blood pressure medication were excluded from participating. Specific participant details will be given in each chapter. All studies were approved by the University's Post Graduate Research Ethics Committee (Appendix 2).

All data were held in a locked filing cabinet or stored on a password protected computer and it was not possible to identify participants from any published outputs from the research. On completion of the data collection and research all data collected, apart from pre-test medical questionnaires and informed consent, were either destroyed or returned to the individual as per the University of Northampton guidelines. Pre-test medical questionnaires and informed consent documentation are to be held securely for six years before being destroyed as confidential waste. On completion of the study a short summary of the results were sent to each participant.

To control for any possible differences in power output and fatigue between male and females, only male participants were used in the main studies in accordance with previous study protocols (Szygula *et al.*, 2003, Hopkins *et al.*, 2001, Hicks *et al.*, 2001). To minimise possible variation in power output within studies due to circadian rhythms (Souissi *et al.*, 2007, Hill and Smith, 1991, Bernard *et al.*, 1998), each participant was tested within ± 1 hour of their initial testing session with a minimum of 48 hours between tests. In addition, participants were instructed not to conduct new training regimes, or any vigorous training prior to each test. All tests were conducted in the same laboratory with the temperature between 18-21°C.

3.2 Arm Crank Ergometer

All studies utilised a Monark cycle ergometer (894E, Monark Exercise AB, Sweden) adapted for use as an arm crank ergometer (ACE) as frequently used for studies examining upper body exercise (Kounalakis *et al.*, 2009, Johnson *et al.*, 2004, Kang *et al.*, 1998, Volianitis *et al.*, 2004b). The seat post was removed and the pedals replaced with handgrips (Monark part number 9145-71). The ACE was raised on wooden blocks attached to a table with the blocks and ACE subsequently bolted through the table (Figure 3.1), with the table also bolted to an external wall. Ten-kilogram metal disks were placed on each corner of the table to further minimise movement during exercise tests.

Figure 3.1 The adapted Monarch cycle ergometer.

Each participant was seated on a sturdy padded metal chair with the backrest removed. To minimise movement of the chair each of the four chair legs were bolted to a wooden board. The participant was positioned such that the centre of their glenohumeral joint was horizontal to the centre of the crank arm on the ACE (Sawka *et al.*, 1983; Sawka, 1986) via a metre rule and spirit level. The

chair height was adjusted to within ± 10 mm by a series of wooden boards and rubber matting. Participants were instructed to find the most comfortable horizontal distance from the ACE, but not to have their elbows locked at the point of furthest extension (Sawka *et al.*, 1983, Washburn and Seals, 1984, Price and Campbell, 1997, Smith *et al.*, 2001, Miller *et al.*, 2004). During the warm-up, participants were able to adjust their position from the ACE as required. To increase inter-test reliability the chair position was noted and kept the same for all tests (Leicht and Spinks, 2007; Miller *et al.*, 2004). Participants were instructed to keep their feet shoulder width apart with their knees at 90° to the floor and not to move their feet during each exercise test.

3.2.1 Arm crank ergometer calibration

The Monark cycle ergometer was checked as per the manufacturer's guidelines for calibration of the height of the weights cradle. In addition, all weights used for determining resistance were checked. The mass of each weight disc used and the cradle mass were each weighed three times on an electronic balance readable to 0.01 g (Sartorius MP 8/8-1, Sartorius AG, Goettinge, Germany) and the mode mass used to be representative of each disc.

For studies involving the Wingate Anaerobic test (studies 1 and 3), the following calibration procedure was undertaken. Each morning (9:00 a.m.) and afternoon (14:00 p.m.) the ACE was calibrated using Cranlea, Wingate software version 4.00 (Cranlea & Company, Birmingham, UK). The calibration involved accelerating the flywheel against a series of resistances (0.5 to 2.5 kg) in 0.5 kg increments. At each stage cranking stopped when 135 rev·min^{-1} was achieved and flywheel deceleration was measured to provide a value of the moment of inertia and friction torque for the ACE (Lakomy, 1986). The calibration was accepted if the calibration regression coefficient was greater than 0.9900, the moment of inertia (MI; reluctance of an object to rotate) was between 0.96 – 1.06 kg·m^2 and friction torque (FT; resistance in the bearings and chainset) was between was between 0.2–0.4 N.m (Wingate Power Test, Cranlea and Company, UK). If the ACE did not calibrate to the required standard then it was re-calibrated.

3.3 Exercise protocols

3.3.1 Wingate Anaerobic test

The Wingate Anaerobic test (WAnT) was used in studies 1 and 3. After resting heart rate was recorded participants completed a 5 minute warm-up at 60 rev·min^{-1} (Winter and MacLaren, 2001) on the unloaded ergometer. After 2 minutes and a count of 3, 2, 1 the resistive load (4% of body mass; Smith and Price, 2007) was released automatically via a manual trigger, with participants maintaining 60 rev·min^{-1} (Winter and MacLaren, 2001). On the command of "Go" participants were instructed to crank as hard and as fast as they could. After 3-4 s the ACE was unloaded and the flywheel allowed to decelerate and participants were instructed to continue arm cranking at 60 rev·min^{-1}. This process was repeated at the start of the third and fourth minutes. Following the third practice sprint the ACE was unloaded and participants continued to crank until the 5 minute warm-up was complete. After completing the warm-up participants continued to crank at 60 rev·min^{-1} on the unloaded ACE and then advised the experimenter when they were ready to start the full 30 s duration WAnT. The same instructions and procedures were given as for the practice sprints. During the 30 s test, all participants were given strong verbal encouragement and an indication of time elapsed (every 10 s). After the test the resistive load was removed and participants was instructed to continue arm cranking at 60 rev·min^{-1} on the unloaded ergometer for at least 5 minutes in order to prevent venous pooling (Weber *et al.*, 2006).

NB: to avoid any conflict between the verbal instruction to stop the test, and to ensure participants had not reduced their maximal effort in anticipation of the end of the test, the last second of WAnT data was omitted from the analysis.

3.3.1.i Software comparison

Two commercially available software programmes were available to record WAnT data from a Monark ergometer; Cranlea Wingate (v.4.00; Cranlea & Company, Birmingham, UK) and Monark Wingate (v.2.20; Monark, Varberg, Sweden). Both the Cranlea software (Baker *et al.*, 2001a, Balmer *et al.*, 2004, Baker *et al.*, 2001b, Franklin *et al.*, 2008) and the Monark software (Zagatto *et al.*, 2008, Dupont *et al.*, 2007, Rana, 2006) have been used for a number of published research studies. As both programmes could be run simultaneously and no previous comparison of the merits and differences between the two systems

have been reported, both systems were trialled during pilot testing to inform the selection of software for the main studies in the thesis. Therefore, this study is presented as part of the methods chapter.

3.3.1.ii Software comparison participants

Twenty participants (Table 3.1), completed a total of 58 Wingate ACE tests. As a compromise between recommended resistive loads for males and females (Smith and Price, 2007) for all participants, resistance was set at 4% of body mass (BM). Participants undertook a range of team and individual sports at competitive or recreational level. None were specifically upper body trained or involved in predominantly upper body sports. Male and female participants were recruited to compare as wide a range of power outputs a possible.

Table 3.1 Participants' characteristics (mean ± *SD*).

	Male	Female	Whole group
n	11	9	20
Age (y)	26.1 (9.2)	22.2 (3.7)	24.4 (7.4)
Mass (kg)	87.0 (18.3)	67.9 (16.8)	78.4 (19.7)
Height (m)	1.78 (0.05)	1.65 (0.05)	1.73 (.09)

3.3.1.iii Software and data

The Monark Wingate programme records from a single sensor located within the crank of the flywheel whereas the Cranlea Wingate programme records from a strip, consisting of black and white bars along its length, applied to the perimeter of the flywheel (Figure 3.1). As the flywheel rotates, the Cranlea data logger records the interruption in signal between the black and white strips enabling flywheel velocity to be calculated, with the data logger operating at 18 Hz. When calibrating for moment of inertia and friction torque the Monark programme assumes a standard moment of inertia value (0.91), while the Cranlea programme requires a calibration sequence in order for these figures to be determined (section 3.2.i). The Monark software records peak power output based on one revolution of the flywheel and peak power output as a mean of data recorded per second. Following data collection, in order to provide a comparison against the Monark software Cranlea peak power output was averaged per 0.5 s and 1 s. It is possible to extract data from both systems for further analysis. Raw data from the Monark can be downloaded but the current

version (v.2.2) does not provide data for the full duration of the test. Therefore, only 24 s was available for comparison between software packages. However, this would not affect analysis of the peak values recorded and as this is past 20 s where there is a rapid decrease in power output it is unlikely to substantially affect the fatigue index (Chtourou *et al.*, 2011). An overview of the data collections and analysis systems is shown in Table 3.2.

Table 3.2 An overview of the Cranlea and Monark data collections and analysis systems.

Monark	Cranlea
Single sensor within the flywheel	Black and white strip on flywheel
No calibration required	Rundown calibration required
Assumes MI and friction torque (FT)	MI and FT calculated from calibration
Power recorded per revolution and per second (1s; mean)	18 Hz sampling
Automatic and manual cage drop	Manual cage drop

Both software systems produce values for corrected and uncorrected power outputs for the WAnT. Uncorrected data does not take into account the energy required to overcome the inertia of the flywheel and power is calculated as the load applied multiplied by cadence (rev·min^{-1}). Corrected power output accounts for the inertia of the flywheel, including friction, and the necessary power needed to overcome the inertia. The following performance variables (all corrected PO) were analysed:

peak power output 1 s
peak power output 0.5 s
peak power output 5 s
mean power output over 24 s
cadence (rev·min^{-1}) at peak power output (1 s)
mean cadence (rev·min^{-1})
time to peak power output (1 s)
end power (1 s mean at 24 s)
fatigue index (FI; [PPO 1 s – minimum power output 1s] / PPO 1 s).

All power variables are in Watts (W). A paired samples t-test was used to analyse differences between systems along with Bland Altman plots to establish the Limits of Agreement.

3.3.1.iv Results

Key performance variables for the WAnT from both the Cranlea and Monark systems are shown in Table 3.3. All power output variables are for corrected power output. A range of power output values from 179 W to 1000 W were recorded for the Cranlea software (1 s), 137 W to 911 W for Monark (1 s) and 216 to 1192 W for Monark software peak power (1 revolution).

Table 3.3 Key performance variables for the WAnT from both the Cranlea (Cr) and Monark (Mk) systems, with *P* values for the T-test, (mean ± *SD*).

	Cranlea v.4.0	Monark v.2.2	R	Mean difference	Cr *vs* Mk (*P* value)
Peak PO	---	546 (264)	0.99 €$		<0.01
Peak PO 0.5 s	589 (267)	----	0.97$¤		<0.01
Peak PO 1.0 s	509 (239)	454 (222)	0.99$	55 (38)	<0.01
Peak PO 5.0 s	443 (216)	411 (198)	0.99$	32 (32)	<0.01
Mean PO 24.0 s	339 (141)	317 (134)	1.00$	22 (14)	<0.01
End PO	242 (88)	245 (91)	0.91$	-3 (38)	0.515
Peak cadence	114 (35)	114 (35)	1.00$	1 (1)	0.678
Mean cadence	100 (29)	98 (28)	1.00$	1 (1)	<0.01
Time to peak PO 1 s	4.46 (2.78)	4.21 (2.04)	0.82$	0.25 (1.60)	0.25
Fatigue index (%)	0.56 (0.16)	0.43 (0.12)	0.64$	0.12 (0.16)	<0.01

€ compared to Cranlea 1 s

¤ compared to Monark peak power

^ R values for X & Y

$ Significant difference ($P < 0.01$) between variables.

Note: cadence ($rev \cdot min^{-1}$); power (Watts; W); PO (power output).

Strong correlations were observed between variables, in particular between all measures of peak power output (PPO), and mean power output (MPO). The weakest correlation was time to peak power output (PPO_{time}), which may be

expected given the data recording methods of both systems. However, mean values were similar. Bland and Altman plots show the closest agreement between Monark (peak) and Cranlea (1 s and 0.5 s) PPO (Figure 3.3 and 3.2, respectively). As PPO output increased there was a tendency for the disparity of measurements to increase (heteroscadasticity). Peak power output values for Cranlea (1 s) vs Monark (1 s), Cranlea (1 s) vs Monark (peak) and Cranlea (0.5 s) vs Monark (peak), were all significantly different ($P < 0.01$).

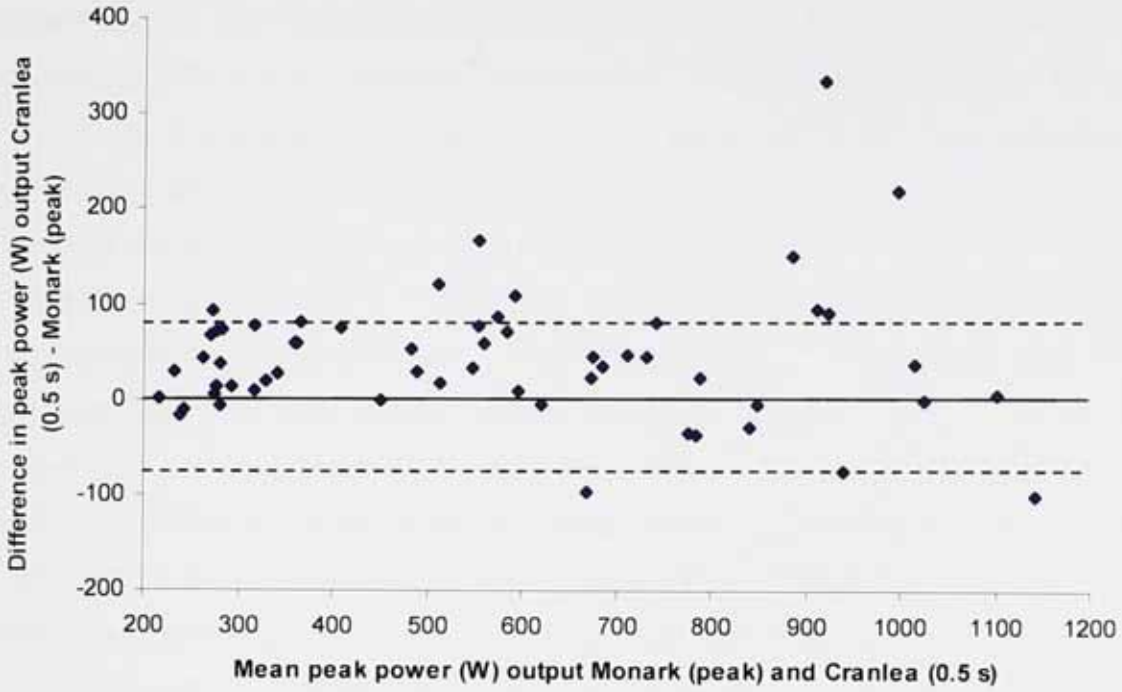


Figure 3.2 Bland and Altman plot with 95% limits of agreement (dashed lines) for peak power output between the two measurement devices.

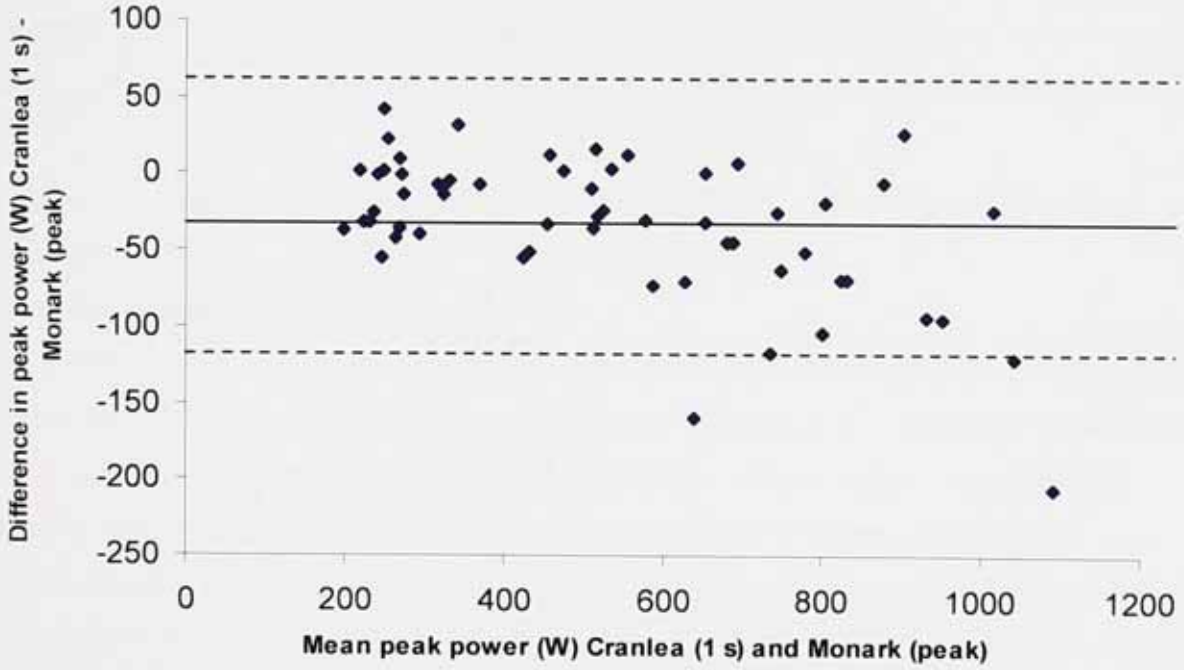


Figure 3.3 Bland and Altman plot with 95% limits of agreement (dashed lines) for peak power output between the two measurement devices.

There was a significant difference between PPO values from the Cranlea software when averaged over 0.5, 1.0 and 5.0 second durations ($P < 0.05$). Peak power output values were 589 (± 267), 513 (± 239) and 443 W (± 216) ($P < 0.05$) respectively. Post-hoc analysis revealed differences between each pairwise comparison ($P < 0.05$).

The results of this study suggest that there were significant differences in peak power output (PPO) regardless of the sample time. Furthermore, as sample time decreases PPO values increase for systems. Peak power output for Monark and Cranlea at 5 s was closer, which is likely to be reflected of the averaging smoothing the higher power values and the inability to hold a high power output for long duration. Also, Monark and Cranlea PPO (0.5 s) was closer, which was probably a reflection of the recording time for the Monark being closer to the recording time for the Cranlea. The physical reliability of both systems was comparable, with both failing to record data on two occasions. This has been noted in a previous study using Cranlea software (Smith *et al.*, 2007b). An advantage of the Cranlea software is the recording interval (18 Hz) allowing more detailed power analysis. Provided the recording start time is synchronised with other analysis equipment it may be possible to provide detailed analysis per pedal revolution, or unit of time, enabling a more detailed analysis than is available from the Monark software. Given the ability to analyse sections of the duty cycle of the cranks, raw data available for 30 s, and that the sampling frequency (18 Hz) was greater than the minimum (5 Hz) recommended (Santos *et al.*, 2010), it was decided to use the Cranlea software for the main studies.

3.3.1.v Familiarisation and reliability of the upper body Wingate anaerobic test

Familiarisation rationale

Familiarisation to exercise testing procedures is recommended in order to reduce measurement error such as learning effects, fatigue, and biological or mechanical variation (Atkinson and Nevill, 1998). Although many authors of scientific studies note within their methods sections that participants were fully familiarised with procedures prior to testing, data regarding the nature of such familiarisation and magnitude of its effects on performance is lacking. However, a small number of studies have specifically considered the effects of test familiarisation on a range of performance based trials such as, 1000m outrigger

canoeing (Sealey *et al.*, 2010) and 2000m cycling time trials (Corbett, 2009), prolonged exercise with a sprint or performance based component (Tyler and Sunderland, 2008, Marino *et al.*, 2002) and repeated sprint tests (McGawley and Bishop, 2006, Spencer *et al.*, 2006). These studies have generally reported improvements in performance after three (Tyler and Sunderland, 2008, Marino *et al.*, 2002) or four trials (Sealey *et al.*, 2010) with accompanying improvements in the coefficient of variation for each performance trial (Marino *et al.*, 2002, Spencer *et al.*, 2006). Where studies have examined self-paced time trial performance, significant changes in pacing strategy across trials have been observed with familiarisation (Tyler and Sunderland, 2008, Corbett, 2009). It is important to note that changes in pacing strategy were also noted when improvements in performance were not observed but were accompanied by changes in the pattern of energy expenditure.

Although the above studies have provided useful information regarding familiarisation, these relate predominantly to self paced trials (Tyler and Sunderland, 2008, Corbett, 2009), performance following a long duration exercise pre-load (Marino *et al.*, 2002), sprint performance with one hour of submaximal exercise (Marino *et al.*, 2002) or sprints protocols (McGawley & Bishop, 2006; Schabort *et al.*, 1999, Hopker *et al.*, 2009). Although the WAnT has been demonstrated to be a valid and reliable test for assessing power output in both leg (Vandewalle *et al.*, 1987, Inbar *et al.*, 1996, Bar-Or, 1987) and arm ergometry (Jacobs *et al.*, 2003, Jacobs *et al.*, 2005, Smith *et al.*, 2007b) no studies have reported the familiarisation effects of conventional laboratory based tests, such as the WAnT in upper body ergometry. Furthermore, the majority of cycle ergometry studies have considered well trained participants with only two considering participants not well accustomed to laboratory procedures by familiarising with repeating two and three sprint trials (Marino *et al.*, 2002, respectively, Barfield *et al.*, 2002). Familiarisation may be of specific importance for upper body exercise testing due to the uniqueness of the testing mode and where not specifically trained participants are often examined (Smith *et al.*, 2007b, Kounalakis *et al.*, 2009, Nindl *et al.*, 1995). Therefore, the aim of this study was to determine the effects of familiarisation on performance and reliability of the WAnT for the upper body.

Familiarisation method

Following institutional ethical approval, 17 students with no previous arm crank ergometry experience volunteered to participate. Participants were either moderately active (recreational walking, cycling) or were involved in team sports (e.g. rugby, football) and/or undertook resistance exercise at least 2 days a week. Participants were instructed not to undertake any new training activities and/or high intensity exercise 24 h prior to testing. Participant details are shown in Table 3.4. This population, male and female, elicited a range of power output values reported in the literature for WAnT of the upper body (Inbar *et al.*, 1996). All participants provided written informed consent and completed a health screening questionnaire prior to each exercise session. The University's Post Graduate Research Ethics Committee approved all studies.

The ergometer was set-up as reported in section 3.2, with participants receiving the same test instructions as reported in section 3.3.1. Participants completed three WAnTs (T1, T2, and T3) with a minimum of 48 hours rest between each test. Corrected and uncorrected peak power output (PPO; over 1 s duration) and mean power output (MPO; over 24 s duration was recorded, as described in section 3.3.1.iii) were recorded using Cranlea UK Wingate software (version 4.0). Peak cadence and PPO_{time} values were also recorded.

Table 3.4 Participants' characteristics (mean ± *SD*).

	Male	Female	Whole group
n	10	7	17
Age (y)	25.0 (9.0)	23.1 (3.7)	24.2 (7.1)
Mass (kg)	87.9 (19.0)	71.7 (17.0)	81.2 (19.5)
Height (m)	1.80 (0.04)	1.66 (0.05)	1.74 (0.08)

Group means were compared using a repeated measures analysis of variance (SPSS v.17.0) with Bonferroni correction and all other statistics were calculated using Microsoft Excel 2003. For reliability analysis, the intra-class correlation (IC), coefficient of variation (CV) and Bland-Altman Limits of agreement (LoA) and bias were calculated from trials T2 and T3.

Familiarisation results

The PPO for each of the three trials is shown in Table 3.5. Correlations were strong between trials (T1 *vs* T2 $r = 0.94$, T2 *vs* T3 $r = 0.96$). The coefficient of variation for trial 1 to 2 was 9.8% and 8.2% for trial 2 to 3. The majority of participants increased their PPO from T1 to T2 (14%, $P = 0.024$) but not between T2 and T3 (3%, $P = 0.874$). As there were no differences in PPO between T2 and T3 these trials were used for reliability analysis. The reliability indices (Table 3.6) indicate good reliability and are similar to previous studies examining upper body exercise (Smith *et al.*, 2007b, Patton *et al.*, 1985). Based on the results of the three trials, unfamiliarised participants were subsequently required to undertake one practice trial before WAnT experimental trials which is in agreement with a lower body cycle ergometry recommendation (Barfield *et al.*, 2002).

Table 3.5 Corrected mean and peak power outputs (W) combined for male and female participants (mean ± *SD*).

	Trial 1	Trial 2	Trial 3
Peak power 1 s (W)	455 (206)	519 (251)	537 (250)
Mean power 24 s (W)	315 (136)	341 (141)	353 (148)

Table 3.6 Intraclass correlation coefficients (ICCs), coefficients of variation (CVs) and *P* values for corrected peak power output (W), uncorrected peak power output (W) and mean power output (24 s; W).

Trial 2 *vs* Trial 3			
	Corrected peak power (W)	Uncorrected peak power (W)	Mean corrected power (W)
ICC	.95	1.0	.99
CV	7.8	4.3	4.6
P	.26	0.06	.13

3.3.2 Peak oxygen uptake test

3.3.2.i Protocol

Peak oxygen uptake (VO_{2peak}) was determined in studies 2 and 3. The following protocol was undertaken. Participants were positioned at the ACE as detailed in section 3.2. The initial load was 50 W for 3 minutes with increments of 20 W every 2 minutes thereafter until volitional exhaustion. Crank rate was set at 70

rev·min^{-1} (Smith *et al.*, 2001, Price and Campbell, 1997, Price *et al.*, 2007, Sawka *et al.*, 1983). Volitional exhaustion was judged to have occurred when the crank rate dropped below 65 rev·min^{-1} for 5 s (Smith and Price, 2007). Peak oxygen uptake was taken as the highest recorded oxygen consumption (l·min^{-1}) over a 15 s average during the test.

Participants had visual feedback for cadence on the Monark ergometer display, and were given verbal feedback when their cadence deviated from 70 rev·min^{-1}. Participants were instructed to reach 70 rev·min^{-1} as quickly as possible and maintain this cadence for as long as possible. Additional pacing from a digital metronome (DM-11, Seiko UK., Ltd., Berks, UK) provided audio feedback (Bressel and Heise, 2004; Hintzy *et al.*, 2008; Kang *et al.*, 1998). Participants indicated their rating of perceived exertion (RPE) using the Borg's 6-20 scale (Borg, 1998a) in the last 20 s of each incremental stage. Participants firstly indicated RPE for local fatigue (RPE_L; arms) and secondly RPE for cardiorespiratory exertion (RPE_{CR}) (Kang *et al.*, 1998; Smith *et al.*, 2006). Participants were given verbal encouragement throughout all tests (Moffatt *et al.*, 1994). After termination of the test, participants were asked for their RPE_L and RPE_{CR} fatigue. Participants then completed a 5 minutes warm-down on the unloaded ergometer at a self-selected cadence, typically 50–70 rev·min^{-1}. Peak oxygen uptake was considered to have been reached if two of the following criteria were met: a respiratory exchange ratio (RER) ≥ 1.1 (Muraki *et al.*, 2004, Marais *et al.*, 1999), RPE_L > 18 (Muraki *et al.*, 2004) and volitional exhaustion (Warren *et al.*, 1990, Yasuda *et al.*, 2006, Yasuda, 2008).

In order to calculate the peak minute power (PMP) the fraction of time spent at the final two exercise stages was calculated (Albertus-Kajee *et al.*, 2010). For example:

Test ended 11 min 30 s

Completed all (2 min) of previous stage at 130 W.

Completed 30 s of next stage.

30 s / 120 s = 0.25 x 100 = 25% of next stage completed.

25% of 20 W (the increase in W from completed stage) = 5 W

Peak minute power is 130 W + 5 W = 135 W

3.3.2.ii Reliability and familiarisation to the VO_{2peak} test

In order to determine the reliability of the VO_{2peak} test 21 participants (Table 3.7) volunteered to undertake repeated VO_{2peak} tests. All tests were completed as section 3.4.3.

Table 3.7 Participants' characteristics (n = 21) for the reliability and familiarisation of the VO_{2peak} test (mean ± *SD*).

Age (y)	Mass (kg)	Height (m)
23.7 (8.1)	79.6 (15.7)	1.77 (0.07)

3.3.2.iii Results

The cardio-respiratory responses at volitional exhaustion are presented in table 3.3.8.

Table 3.8 Cardio-respiratory variables for trial 1 and trial 2 at volitional exhaustion (mean ± *SD*).

	Trial 1	Trial 2
PMP (Watts)	136 (25)	141 (28)
VO_{2peak} ($l \cdot min^{-1}$)	2.33 (0.41)	2.40 (0.46)
Heart rate ($beats \cdot min^{-1}$)	176 (13)	176 (12)
RER	1.28 (0.09)	1.28 (0.09)

Peak physiological responses were representative of those reported in the literature for the population studied (Enders *et al.*, 1994, Swaine and Winter, 1999, Tarara, 1995, Schrieks *et al.*, 2011) and 0.5 $l \cdot min^{-1}$ less than reported in a non-specifically trained population (Price *et al.*, 2007). Previous studies have determined the reliability of peak oxygen consumption at 50 $rev \cdot min^{-1}$ (Bar-Or and Zwiren, 1975) and 60 $rev \cdot min^{-1}$ (Price and Campbell, 1997). Reliability of peak oxygen consumption during ACE has not been reported at 70 $rev \cdot min^{-1}$ therefore each participant completed two VO_{2peak} trials (Table 3.8). Although the PMP was significantly different from test 1 to 2 (P = 0.006; Table 3.9) the increase in power (5 W) is well within the error of measurement expected (Leicht *et al.*, 2009). Furthermore, the limits of agreement and bias for PMP were observed to be ~10W. The intraclass correlation of .96 for peak minute power (Watts; PMP) is similar to reported values of .94 (Price and Campbell, 1997) and indicated that PMP is reliably determined using this protocol.

Table 3.9 Intraclass correlation coefficient (ICC), limits of agreement (95%; LoA), bias (mean difference), coefficients of variation (CVs) & *P* values (paired t-test) for peak minute power (PMP), peak oxygen uptake (VO_{2peak}), heart rate (HR), respiratory exchange ratio (RER).

	ICC	LoA	Bias	CV	Paired t-test
PMP (W)	.96	-9/+19	~10W	3.094	.006
VO_{2peak} ($l \cdot min^{-1}$)	.91	-0.30/+0.44	0.07	4.611	.080
HR ($beats \cdot min^{-1}$)	.82	n/a	n/a	2.266	.731
RER	.52	n/a	n/a	3.809	.945

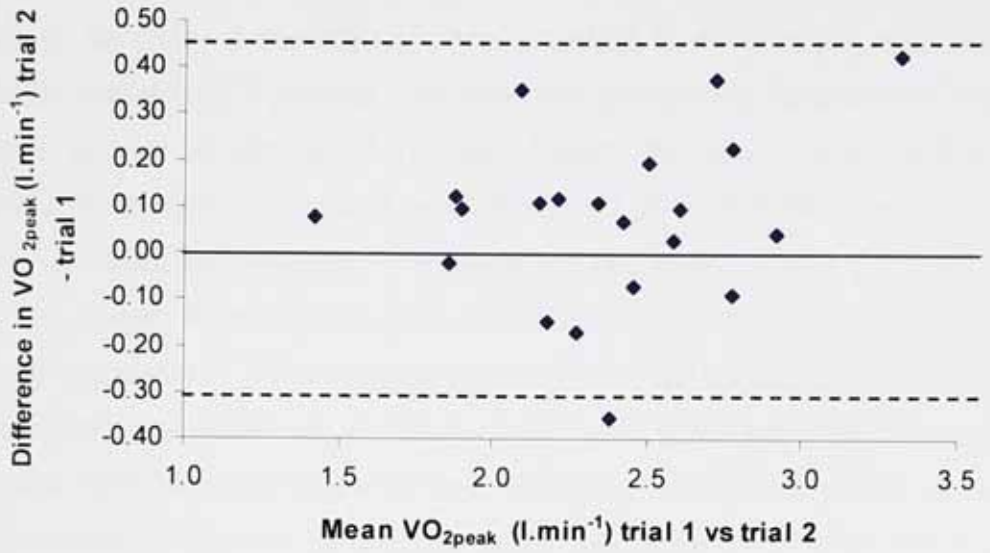


Figure 3.4 Bland and Altman plot with 95% limits of agreement (dashed lines) for mean VO_{2peak} ($l \cdot min^{-1}$) between trial 1 and trial 2.

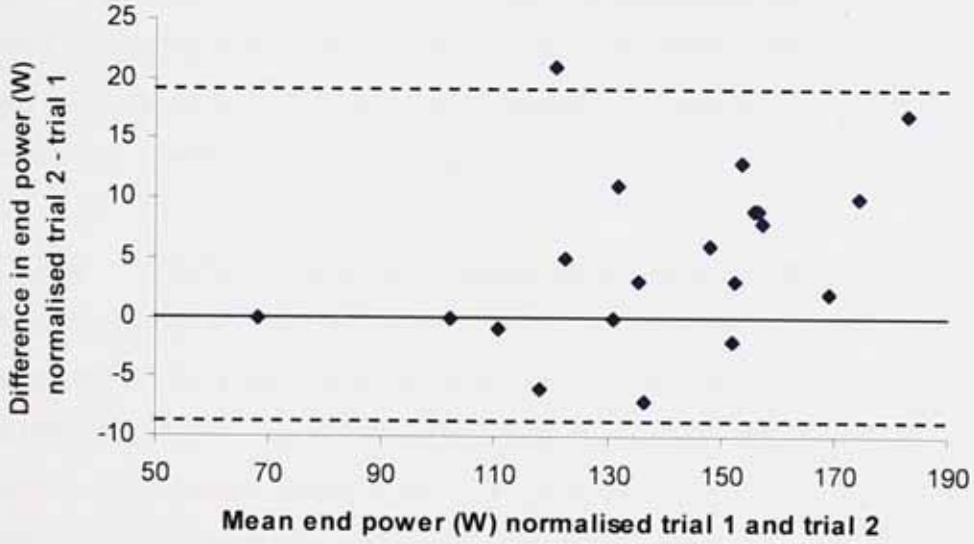


Figure 3.5 Bland and Altman plot with 95% limits of agreement (dashed lines) for end power (W) at VO_{2peak} ($l \cdot min^{-1}$) between trial 1 and trial 2.

3.3.3 Continuous work test

Studies 2 and 3 used a range of continuous work tests to exhaustion in order to measure time to exhaustion (T_{lim}) and difference in physiological and biomechanics responses at exhaustion. Each participant completed a peak oxygen uptake test (section 3.3.2.i) with their PMP calculated (section 3.3.2.i). All subsequent tests were completed after at least 48 hours rests and where completed at the same time of day as the initial test ± 1 hour. In study 2 the VO_{2peak} test was repeated. In study 3 participants only completed a VO_{2peak} test before and after the training programme. All subsequent tests were completed after at least 48 hours rest and at the same time of day as the initial test ± 1 hour. In study 2 participants arm cranked to exhaustion at a work load of 80%, 90%, 100% and 110% of PMP with the workloads balanced using a 4 × 4 × 4 Latin square to allocate the order of each PMP test. In study 3 participants arm cranked to exhaustion at a work load of 100% of PMP before and after training, with an additional 100% PMP based on the PMP achieved following their second VO_{2peak} test. Participants were verbally encouraged to continue each trial for as long as possible. All participants were instructed to completed the trials, including the warm up, at 70 rev·min^{-1} and trials were terminated when the crank rate dropped below 65 rev·min^{-1} for 5 s (Smith and Price, 2007). The PMP trials were preceded by a 5 min warm-up, after 2 minutes the ACE was loaded with a mass corresponding to the participants 80% PMP (100% PMP for study 3) for 20 s and participants were instructed to continue arm cranking at 70 rev·min^{-1}. This process was repeated at the start of the third and fourth minutes. Following the third load the ACE was unloaded and participants continued to crank until the 5 minute warm-up was complete. After completing the test the trial mass was removed and participants completed a minimum 5 minute cool down at a freely chosen cadence.

3.3.3.i Reliability of the continuous work test to exhaustion

In order to determine the reliability of the time to exhaustion tests participants volunteered to undertake repeated PMP resistive loads tests. Two PMP loads (80% and 110%) were repeated. For the 80% PMP test four participants (age = 29.2 ± 10.3 years, mass = 80.5 ± 18.6 kg, height = 1.74 ± 0.07 m) and 110% test three participants (age = 32.0 ± 12.1 years, mass = 84.8 ± 24.9 kg, height = 1.76 ± 0.07 m) completed the repeat tests. All tests were completed as section 3.3.3.

3.3.3.ii Results

Time to fatigue for the 80% PMP trial was 823 s (151) vs 864 s (165) and for the 110% trial was 221 s (16) vs 233 s (43) between trial 1 and 2. With a mean variability of 5% for both 80% and 110% is better than those found for cyclists at 80% (17% variation) and 120% (10% variation) (McLellan *et al.*, 1995, Graham, 1989). Similar reliability would likely be found for time to exhaustion at 90% and 100% of peak normalised power output.

3.4 Physiological measurements

3.4.1 Body mass and Stature

Body mass (Hanson TFA-05, Hanson, Herts, UK) and stature (Holtain stadiometer, Holtain, Dyfed, UK) were recorded on each experimental trial. For studies involving the WAnT body mass recorded on the initial testing session was used to calculate the resistive loading applied during all subsequent tests.

3.4.2 Heart Rate

In all studies heart rate (HR; beats·min^{-1}) was recorded using a telemetric chest strap and watch (Polar Accurex Plus, Polar, Electro Oy, Finland). Resting HR was recorded while participants were seated at the ACE. Further values were continuously recorded throughout each test and during the recovery period.

3.4.3 Expired gas analysis

Expired gas was collected via a Metalyser 3B (Cortex, Lepzig, Germany) breath-by-breath automated system and analysed using MetaSoft v.3.9.7 software (Cortex, Lepzig, Germany). To reduce the 'noise' generated by breath-by-breath gas analysis outliers were removed prior to processing (Midgley *et al.*, 2007). Values were first averaged for 1 s (Koppo *et al.*, 2002) and then further averaged using a 15 s rolling average (James *et al.*, 2007a). Before each test the analyser was calibrated with room air (20.93% oxygen and 0.03% carbon dioxide) and known reference gas mixtures (17.07% oxygen and 5.03% carbon dioxide) certified to Beta standard (BOC Gases, Surrey, UK). The turbine flow meter (Triple V Turbine, Cortex, Lepzig, Germany) was calibrated for volume with a 3 litre calibration syringe (Hans Rudolph, Inc, Kansas City, MO, USA). Barometric pressure was recorded (Fortins Barometer, F. Darton & Co. Ltd., London, UK) to calibrate pressure within the Metalyser 3B. The gas calibration values were checked twice a day at 12:00 and 18:00 hours with room air and the

known reference gas calibrations as noted previously. After each participant completed the first test the same size of face mask (Hans Rudolph, Kansas City, MO) secured with a head-cap (Hans Rudolph, Kansas City, MO) and turbine (see above) was used for all the remaining trials for that participant.

3.4.4 Ratings of perceived exertion

For studies 2 and 3 ratings of perceived exertion (RPE) using the 6–20 Borg scale was used. On the initial laboratory visit, each participant was familiarised with the Borg scale. Participants were instructed to indicated their rating of perceived exertion (RPE), indicating firstly RPE_L and secondly RPE_{CR} (Kang *et al.*, 1998; Smith *et al.*, 2006). To ensure familiarisation was complete these instructions were repeated on the second laboratory visit prior to testing.

3.4.5 Electromyography (EMG)

For study one, eight electrodes were available for data collection. As EMG data for the upper limb, torso and lower limb EMG data were required only the right hand side of the body was used for EMG data collection. Each site used is described in Table 3.10. After analysis of results from the first study (chapter 4) electrodes reference number 7 and 8 were omitted from studies two and three.

Table 3.10 Electromyograph electrode placement sites and rationale for use.

Muscle	Muscle function	Additional information	Used by authors
Flexor carpi ulnaris (FCU)	Flexes and adducts the wrist		Zehr and Chua, 2000, Smith *et al.*, 2008
Biceps brachii (BB)	Agonist and antagonist to triceps	One of the key muscle groups used and studied in ACE	Bernasconi *et al.*, 2006, Bressel *et al.*, 2001, Bressel and Heise, 2004, Burden and Bartlett, 1999, Frauendorf *et al.*, 1986, Frauendorf *et al.*, 1989, Marais *et al.*, 2004, Smith *et al.*, 2008, Zehr and Chua, 2000
Triceps brachii lateral (TB)	Main extensor of the elbow	During a VO_{2peak} trial was considered the primary cause of the test termination due to fatigue in the muscle group (Mossberg *et al.*, 1999)	Bernasconi *et al.*, 2006, Bressel *et al.*, 2001, Bressel and Heise, 2004, Brink-Elfegoun *et al.*, 2007, Frauendorf *et al.*, 1986, Frauendorf *et al.*, 1989, Hopman *et al.*, 1995, Marais *et al.*, 2004, Smith *et al.*, 2008, Zehr and Chua, 2000
Anterior deltoid (AD)	Flexes and medially rotates the arm	Contributed to performance limitation (Mossberg *et al.*, 1999)	Smith *et al.*, 2008, Bernasconi *et al.*, 2006, Bressel *et al.*, 2001, Bressel and Heise, 2004, Balter and Zehr, 2006, Zehr and Chua, 2000
Infraspinatus (IS)	Laterally rotates the arm	Opposite of the anterior deltoid. More active in the	Bernasconi *et al.*, 2006, Bressel *et al.*, 2001, Bressel and Heise, 2004

		pronated grip used in these studies (Bressel *et al.*, 2001)	
External oblique (EO)	Muscles of the back assists in trunk rotation and lateral flexion	Recommended for further study by (Smith *et al.*, 2008, Koppo *et al.*, 2002)	Hopman *et al.*, 1995
Vastus medialis (VM)	Extends leg at knee joint		Smith *et al.*, 2008
Lateral soleus (LS)	Plantar flexes the foot when the knee is flexed		

3.4.5.i EMG site preparation

As recommended by Hermens at al., (2000) all sites were cleaned with isopropyl alcohol to remove oil and dirt to reduce skin impedance. Additionally, the vastus medialis and lateral soleus were shaved to remove body hair. Double-differential (16-3000 Hz bandwidth, x300 gain), bipolar, active electrodes (MP-2A, Linton, Norfolk, UK) were firmly taped to the skin surface with the wires also taped down. The flexor carpi ulnaris, biceps brachii and triceps brachii lateral electrodes were further secured in place using an oversized (to avoid over-compression) compression bandage. Securing the wires, in such a way, prevented movement artefacts and removed the risk of the wires and electrodes coming loose during exercise. A similar technique has been used in leg cycling (Rouffet and Hautier, 2008). A ground electrode was place on the right kneecap of each participant. After the first test, the placement of each electrode was marked on the participant's skin with a surgical marker pen to enable placement of the electrode on the exact site from the previous test.

3.4.5.ii Electromyography processing

Electromyographic data were sampled at 1000 Hz and filtered using a 20 to 500 Hz band-pass filter (Figure 3.6). The electrodes were connected to a high level transducer interface (HLT100C, Biopac, Goleta, CA) and then converted from analogue to digital signal (MP150 Data Acquisition, Biopac). All signals were analysed using a personal computer running Biopac AcqKnowledge (v.4.0.0) software. The average root-mean-squared (RMS; Figure 3.7) value for each muscle was calculated over 250-ms.

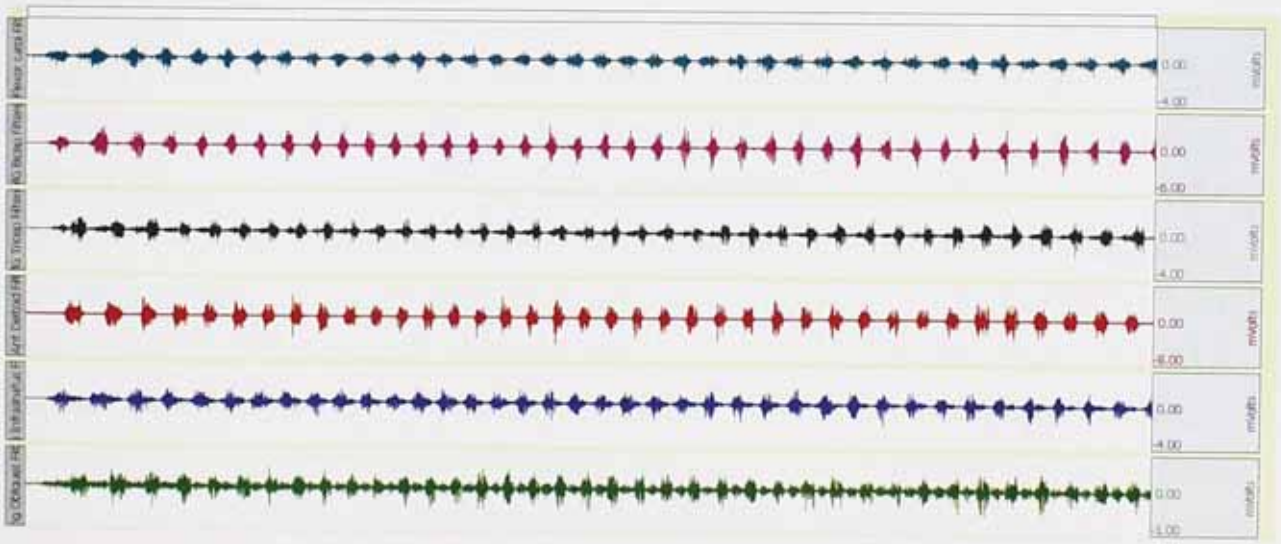


Figure 3.6 Example of EMG signal after filtering but prior to RMS calculation7

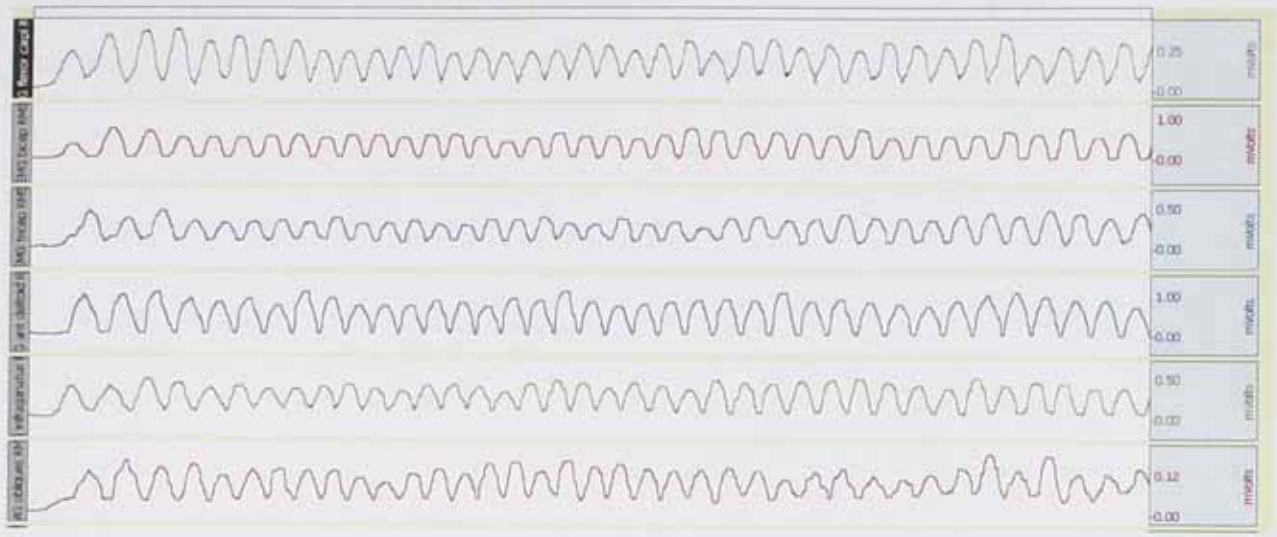


Figure 3.7 Example of EMG signal after RMS calculation

3.4.5.iii Earthing and interference

High noise interference (50 Hz) on the EMG signal on the initial trials of study 1 indicated some interference to the signal. Further investigation suggested that the flywheel was generating a large amount of static electricity. Two earths were subsequently connected to the ergometer frame, which successfully removed this noise.

3.4.5.iv Signal normalisation

Normalisation using isometric maximal voluntary contractions (isometric MVC) is widely used within EMG studies. However, it has been suggested that isometric MVC's cannot by applied to dynamic exercise (Clarys, 2000). Recent research on lower limb EMG activity has indicated that isokinetic MVC's may be more appropriate with dynamic exercise (Burden and Bartlett, 1999, Anders *et al.*, 2005) if not better than isometric MVC's (Rouffet and Hautier, 2008, Albertus-Kajee *et al.*, 2010). Isokinetic MVC's take into account the full range of motion of the joint and muscle length during the activity, can be recorded during the activity (the warm-up) assessing each muscle at the same time, reduce fatigue that may be associated with isometric MVC's and are reliable (Hsu *et al.*, 2006, Rouffet and Hautier, 2008, Albertus-Kajee *et al.*, 2010). Normalisation after a prior full familiarisation may also improve neural drive and therefore reliability across measures (Burden and Bartlett, 1999). The method of isokinetic MVC normalisation has been used effectively during a lower limb WAnT (Rana, 2006, Greer *et al.*, 2006) and during warm-up procedures during previous ACE studies (Smith *et al.*, 2008, Marais *et al.*, 2004, Balter and Zehr, 2006).

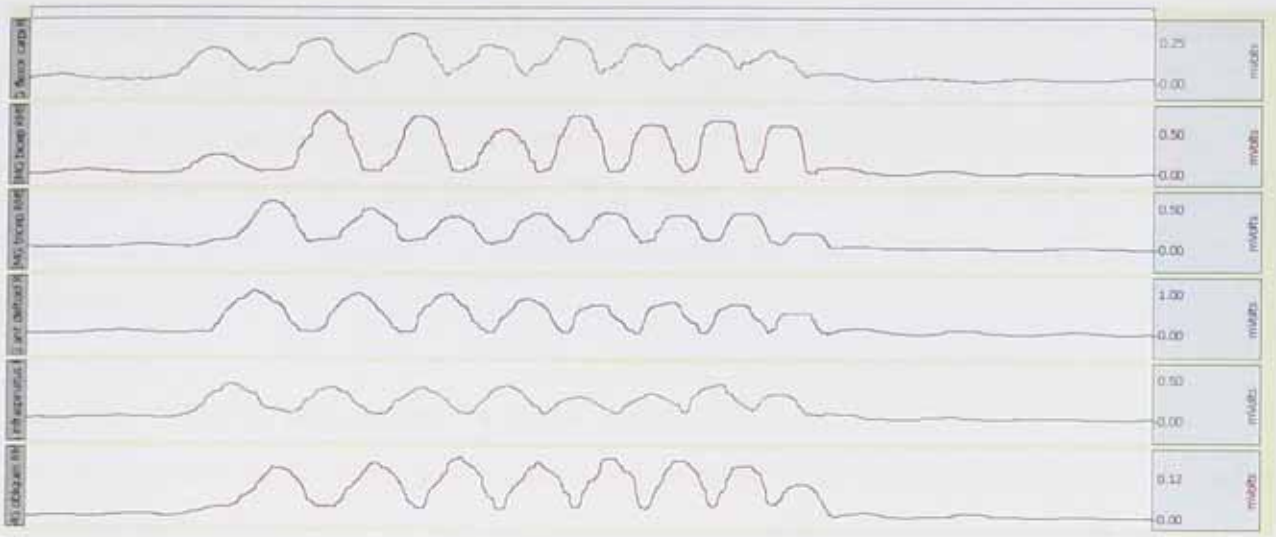


Figure 3.8 Example of one of three RMS EMG 3-4 s 4% BM load sprints during warm-up used to calculate peak normalised RMS EMG.

Signal normalisation for the Wingate anaerobic test

To enable comparison of the EMG signal within the WAnT data of study 1 and study 3 during the warm up all participants complete three 4-5 s sprints against a resistive load of 4% BM. The peak RMS EMG amplitude was calculated as the peak RMS EMG amplitude achieved during the warm-up for each trial (3.8). This peak value was then used to normalise the RMS EMG from each of the EMG recordings for each exercise test. A 4% BM load during the warm up was chosen to provide a sufficient stimulus for EMG normalisation and physiological response without the fatigue that may occur with a 5% load as a too vigorous warm-up may impair performance (Hawley *et al.*, 1989, Bishop *et al.*, 2001). Keeping a 4% BM load throughout all the trials enabled a comparison in normalised RMS EMG activity at 2%, 3%, 4% and 5% of BM. Additionally, a 4% BM load provided a more representative range of values for the abilities of the population being studied.

Signal normalisation for the constant load trials

Prior to the start of the continuous performance trials in studies 2 and 3 a five minute warm up was completed with the unloaded cage (36 W) to provide a sufficient physiological stimulus and enable a smooth loading and unloading of the weight cage. During the warm-up at 1:30, 2:30 and 3:30 minutes a mass, corresponding to 80% of PMP, was added for 20 s. This procedure was repeated for all four trials within study 2 (80%, 90%, 100% and 110% PMP to exhaustion)

and the 100% PMP trials within study 3 to enable a comparison of EMG activity across all PMP trials. The mean RMS EMG amplitude was calculated as the mean of 10 duty cycles during the 20 s 80% PMP load. The three mean RMS EMG amplitudes achieved during the warm-up for each trial (Figure 3.8) were then summed and the mean calculated. This mean value was then used to normalise the RMS EMG from each of the EMG recordings for each PMP resistive load exercise test.

3.4.5.v Electromyography data analysis

For data analysis the RMS EMG data were averaged over three consecutive peaks (Figure 3.9). For study 1, the middle peak corresponded to the time at which peak uncorrected or corrected power occurred with the two peaks either side used was used for analysis. Where the peak power occurred at the base of the RMS EMG the peak to the right was taken as the middle peak. For end power (29 s) the last three peaks at or prior to 29 s were averaged.

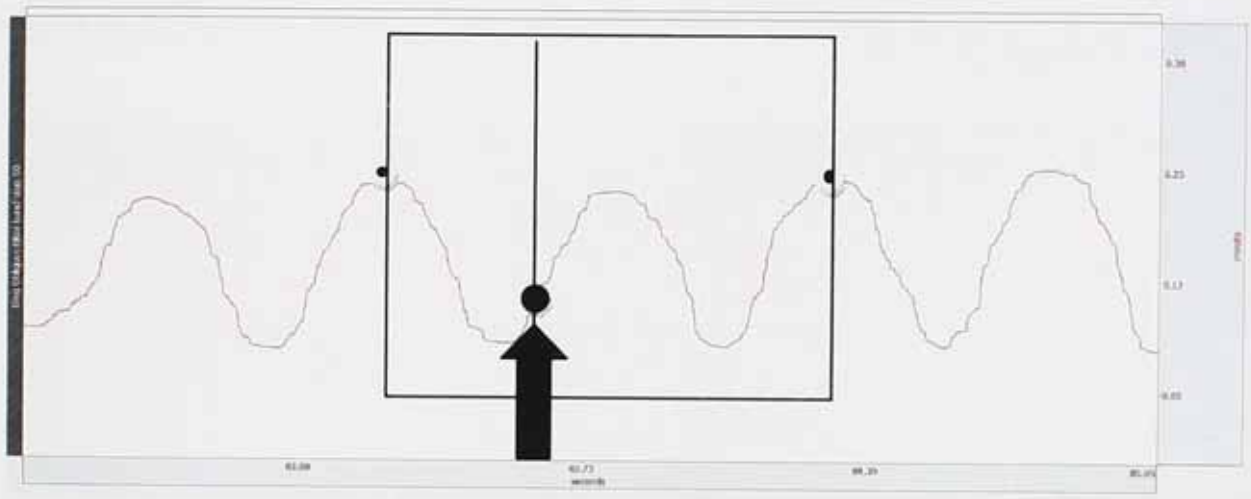


Figure 3.9 Example of method used to calculate average RMS EMG signal. The vertical line and black arrow represent the time point of peak uncorrected power (EMG recording was started prior to the WAnT) and the two peaks (dots) either side make the three EMG RMS signals used. The black arrow represents time of peak power. The box represents the three peak values considered for data analysis.

3.5 Motion analysis

For study 1, three cameras were available for data collection. As the upper limb, torso and lower limb motion analysis data were required for the studies only the right hand side of the body was used for motion analysis data collection. Pilot

testing revealed a three camera system was unable to simultaneously record data from left and right limbs. For studies 2 and 3, four and five cameras were available, respectively.

For 3D recording and computer analysis (Qualisys Track Manager v.2.0.365., Qualisys, Gothenburg, Sweden) fourteen infrared reflective markers were placed on appropriate anatomical landmarks (Figure 3.10 and Table 3.12) and secured using double-sided tape. The markers were tracked by three ProReflex Motion Analysis Cameras (Qualisys, Gothenburg, Sweden) sampled at 100 Hz and smoothed at 100 ms moving average. After each participant had completed the first test, the placement of each marker was marked on the participant's skin with a surgical marker pen as for the EMG measurements. Prior to testing the cameras were calibrated for 10 s, using a calibration frame and wand (Wandkit 750, Qualisys, Gothenburg, Sweden) enabling calibration of X (depth), Y (width) and Z (height) axes within the field of view of the cameras. Once the calibration had passed the calibration test, any markers in the field of view of the cameras could be determined for relative distance.

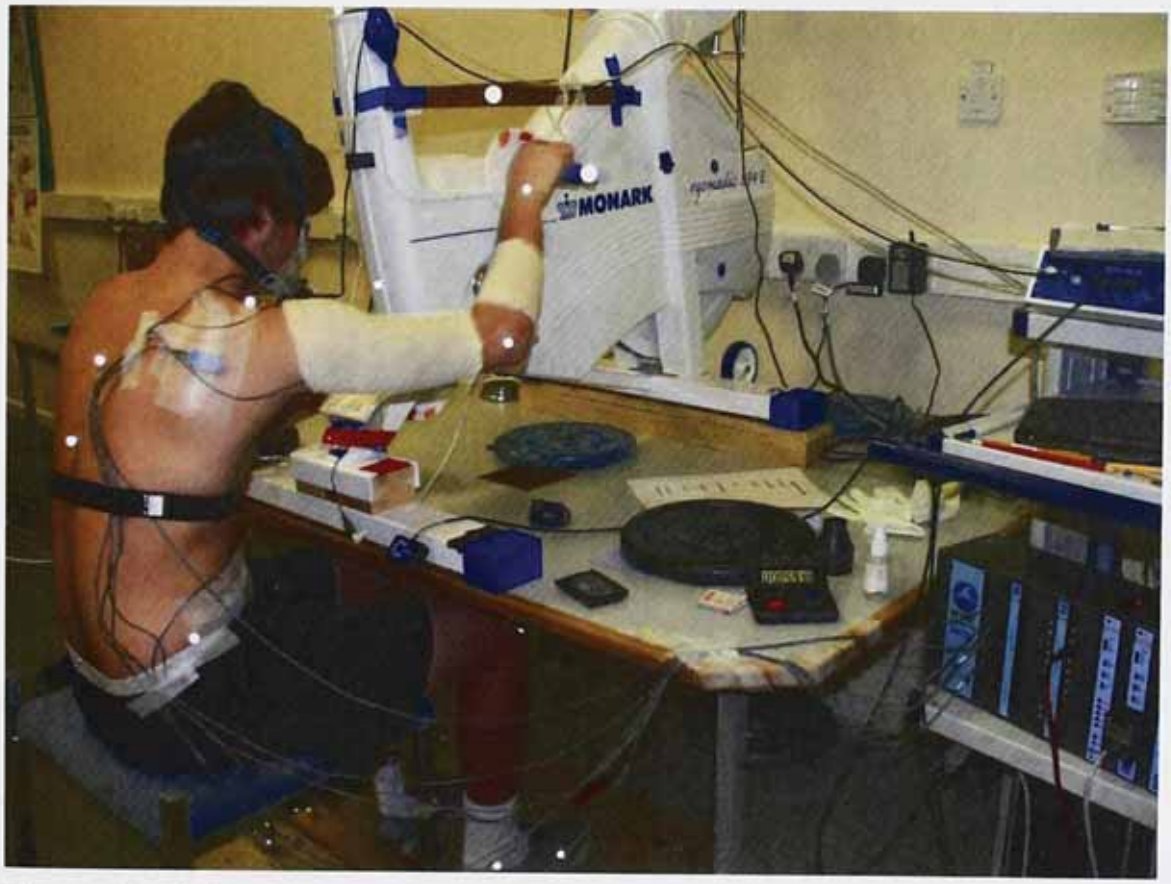


Figure 3.10 Participant arm cranking, showing motion analysis markers, EMG electrodes and gas analysis system.

Table 3.12 Motion analysis marker position and abbreviation.

Marker abbreviation	Marker position
TCSP	Top and centre of seat post pillar
STBB	On seat tube in line with maker letter D
VBB	Directly vertical to centre of bottom bracket
CBB	Centre of bottom bracket
CHg	Centre of handgrip
C7	7th cervical vertebrae C7
T7	7th thoracic vertebrae T7
Ic	Iliocristale
Lc	Joint space between the lateral condyle of the femur and lateral tibia condyle
LmF	Lateral malleolus of the fibula
mT5	Most prominent position where the 5th metatarsal joins the 5th proximal phalange
CtC	Most prominent superior position on the conoid tubercle on the clavical
Acb	The most lateral and superior of the bony process on the acromion border (not used in study 2)
LeH	Lateral epicondyle of the humerous
Us	Ulnar styloid process

After data collection, an automatic identification of markers (AIM) model was created using the Qualisys Track Manager 3D software (v.2.3, Qualisys, Gothenburg, Sweden). The data for one participant's trial was used to identify each marker and from this an AIM model was created for each participant (Figure 3.11). Due to the large number of markers and the close proximity of some of the markers, separate AIM models were used for each participant for greater accuracy. Markers that were that were not identified using AIM were manually identified and assigned to their correct position. Markers not recorded by the cameras were spline-filled to a maximum of 10 frames. Three measures were recorded throughout each WAnT and CWT trial during all three studies and subsequently analysed:

horizontal upper body movement ($C7_D$) the change in distance (mm) between the torso and the ACE (Figure 3.12) the distance between C7 and STBB elbow joint angle (degrees; °) between the ulnar styloid process, the lateral epicondyle of the humerous and the most prominent superior position on the conoid tubercle on the clavicle was recorded to analyse upper limb joint angle angular velocity (degrees/s; °·s) between the 7th cervical vertebrae, the most prominent superior position on the conoid tubercle on the clavicle and the top and centre of the seat post pillar to analyse speed and change in direction of the trunk in relation to the ergometer.

Figure 3.11 Example of AIM model for motion analysis

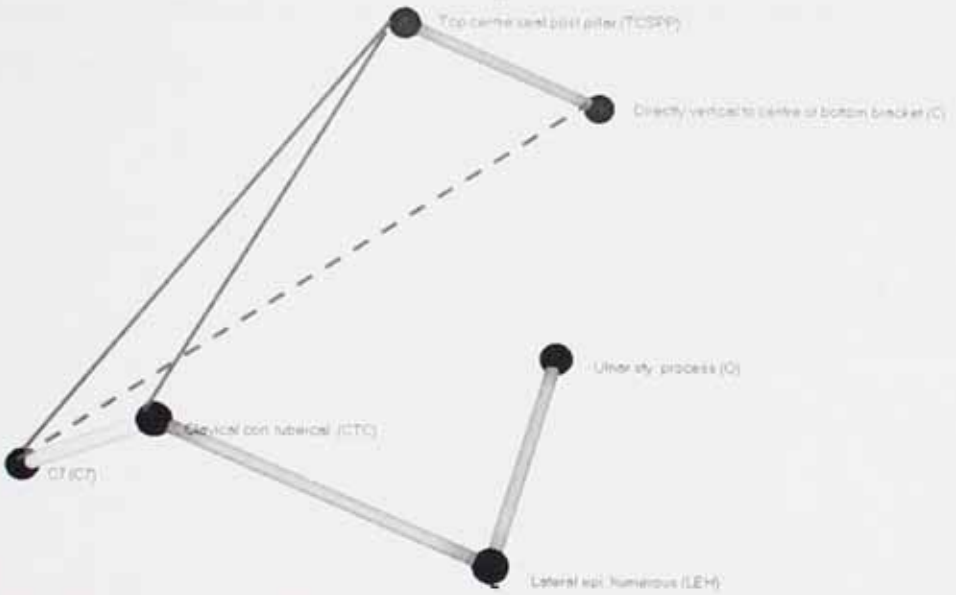


Figure 3.12. Example of kinematic analysis showing anatomical and static markers for analysis. The solid line represents the joined points of trunk rotation. The dashed line represents the joined points of the two markers for distance.

3.5.1 Motion analysis during normalised percent of peak minute power

Preparation and post-test marking of anatomical sites was the same as for the EMG analysis. The most lateral and superior of the bony process on the acromion border was not used in all three studies as the most prominent superior position on the conoid tubercle on the clavical was found to be more reliable for analysis. Although an additional marker was also placed on the centre of the ergometer to enhance analysis of trunk rotation with the 7th cervical vertebrae (C7), this marker was too obscured by the participants to provide accurate analysis.

3.5.2 Data analysis and calculation of joint angles and distance

All data were selected in QTM and filtered before and after calculation (11 frames per filter window). The results were then exported to Microsoft Excel for further analysis. For data analysis, the joint angles were averaged over three peaks in a similar process as for the EMG analysis (Figure 3.14). The middle peak corresponded to the time peak uncorrected or corrected power occurred and the peak either side made the three data points. Where the peak occurred at the base or trough of the data cycle the peak to the right was taken as the middle peak. At the end of each trial the last three peaks at or prior to end of the test were averaged. The angle for each of the peaks was calculated in Microsoft Excel from the difference between the peak and minimum angle for each wave (Figure 3.13).

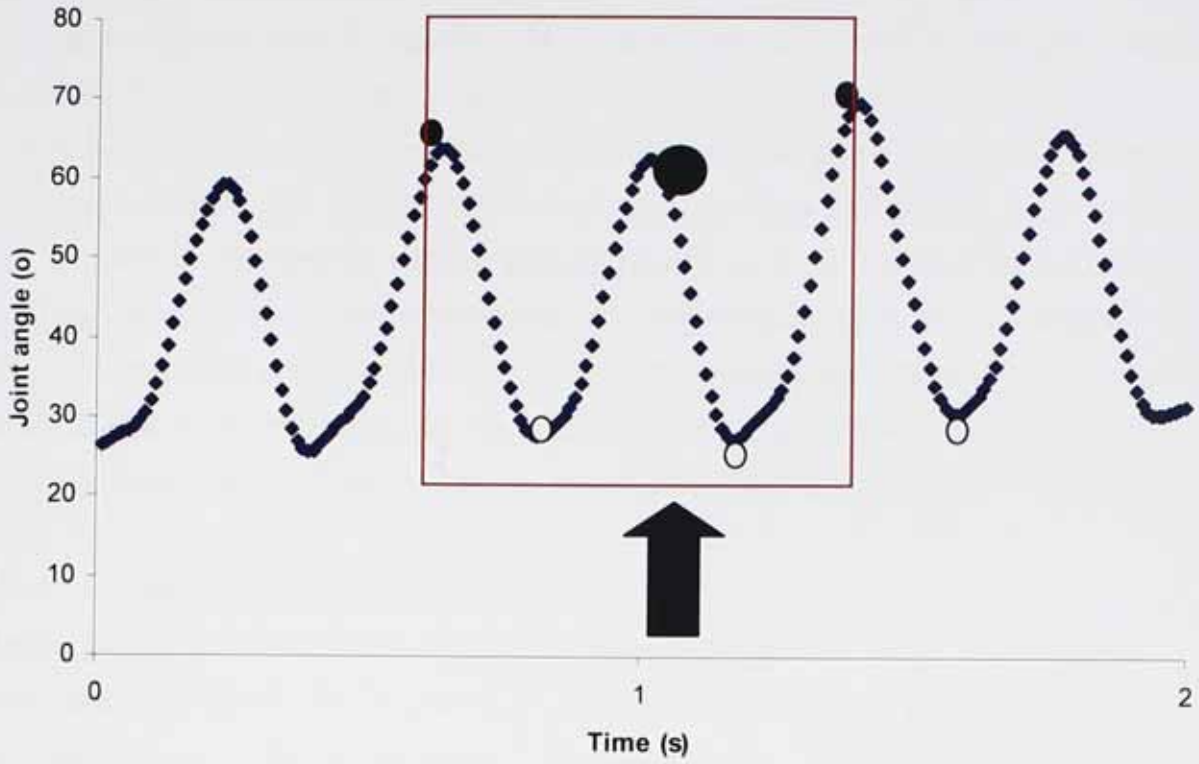


Figure 3.13 Example of joint angle calculation. The large black dot represents peak power corrected and the two peaks (small black) either side make the three joint angles. The unfilled dots mark the minimum of the joint angle. The black arrow represents the time to peak power. Box represents the three peak values considered for data analysis.

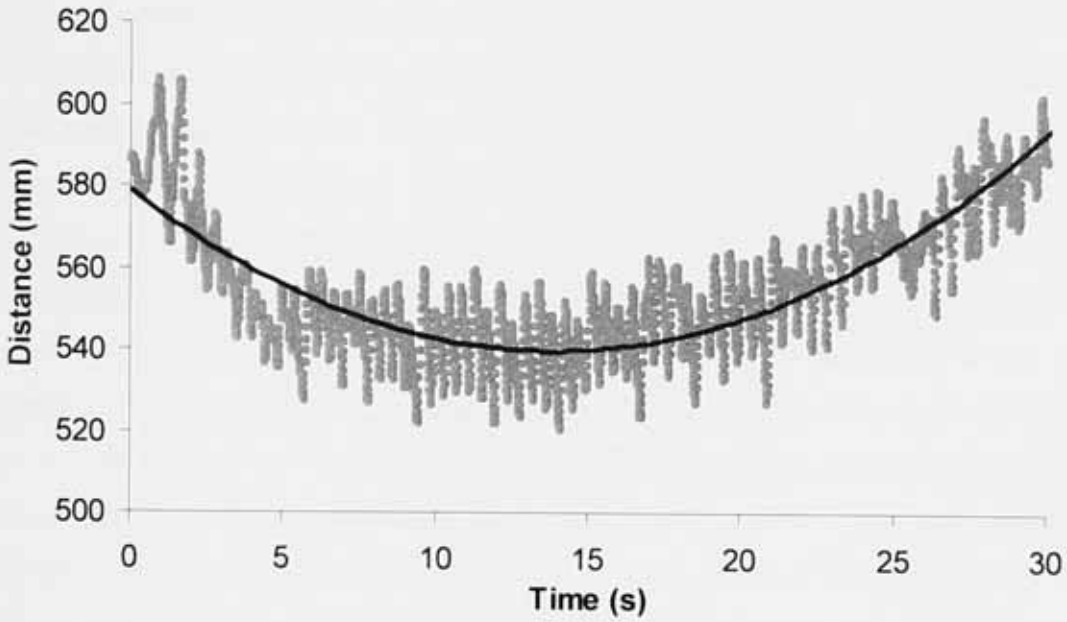


Figure 3.14 Example of change in distance (mm), during a WAnT, between markers C7 and VBB.

3.6 Synchronisation of signals

To synchronise the WAnT (studies 1 & 3), EMG, motion analysis and gas analysis (studies 2 & 3) a number of techniques were used. First, a trigger was connected to the Qualisys motion analysis system and the Biopac EMG system. The trigger started the capture of motion analysis data and placed a signal spike on a channel in the Biopac system (set to record prior to the start of each test). Secondly, as the trigger was depressed a second trigger was also depressed which dropped the weight cage on the Monark ergometer. Thirdly, for the CWTs (study 2 and 3) as both triggers were depressed a marker was placed on the Cortex gas analyser software (set to record prior to the start of each test).

3.7 General statistics

All data are presented as the mean ± standard deviations (*SD*). All analyses were performed using the Statistical Package for Social Sciences (v 17.0; SPSS Inc., Chicago, IL). For all the statistical analysis, the level of significance was set as $P \leq 0.05$. Where statistical significance was approached these results are reported to further inform analysis of the data (Williams and Wragg, 2004, Winter *et al.*, 2001). Statistical results in the text are reported as actual *P* values. All data were tested for normal distribution using tests of skewness and kurtosis (Field, 2009). Where SPSS presented *P* values of $P = 0.000$ these are reported as $P < 0.001$. When data were analysed using an ANOVA, individual differences between means were located using Bonferroni post-hoc correction. Bonferroni correction was undertaken as it provides a conservative control over Type I errors and is more suited than other *post hoc* where the number of comparisons is small (Field, 2009). It is acknowledge that Bonferroni correction may increase the probability of committing a type II error (Field, 2009). Therefore, test-retest reliability data was used to explore and interpret the true meaningfulness of subsequent findings. A number of statistical analyses used a repeated measure design. When repeated measures were used and Mauchly's test of sphericity was not significant ($P > 0.05$) and sphericity was assumed the *F*-ratio and associated degrees of freedom were used to test for statistical significance between groups. When sphericity was not assumed ($P \leq 0.05$), the Greenhouse-Geiser value with the F value with the degrees of freedom corrected to test for statistical significance between groups.

Chapter 4

The physiological and biomechanical responses to short duration, maximal intensity arm cranking

4.1 Introduction

A number of factors affecting aerobic upper body exercise such as cadence (Price and Campbell, 1997; Sawka *et al.*, 1983; Smith *et al.*, 2006b; Smith *et al.*, 2001) and exercise protocol (Sawka, 1986, Smith *et al.*, 2002b, Smith *et al.*, 2006a, Castro *et al.*, 2010, Walker *et al.*, 1986) have been thoroughly examined. Established exercise testing protocols have subsequently been developed (Smith and Price, 2007, Kenney, 2005). However, despite the use of arm crank ergometry (ACE) being beneficial to exercise and health scenarios (Metter *et al.*, 2004, Zwierska *et al.*, 2005, Rosler *et al.*, 1985a, Westhoff *et al.*, 2008) and a range of sports including the specific sport of hand cycling (Kounalakis *et al.*, 2008, Franklin, 1985, Franklin, 1989, Hawley and Williams, 1991, Mermier, 2000), little information has been reported regarding the factors affecting anaerobic aspects of ACE. Although it is known that during aerobic ACE there is a peripheral limitation to exercise (Loftin *et al.*, 1988, Muraki *et al.*, 2004, Sawka, 1986, Franklin, 1985) resulting in peak rather than maximal responses (Magel *et al.*, 1975) and localised rather than cardiorespiratory fatigue (Price *et al.*, 2007), much less is known regarding fatigue for anaerobic upper body exercise.

A commonly used anaerobic test for both the upper and lower body is the Wingate anaerobic test (WAnT). Previous studies examining upper body WAnTs have suggested that during the WAnT, the arms work more anaerobically than the legs (Kounalakis *et al.*, 2009, Lutoslawska *et al.*, 2003). Indeed, muscle biopsy studies indicate a greater proportion of fast twitch to slow twitch muscle fibres within the upper than lower body (Mygind, 1995), with data presented by Inbar *et al.*, (1996), Dotan and Bar-Or (1983) and Marsh (1999) indicating that fatigue during a 30-s WAnT is greater for the upper than lower body. Furthermore, upper body joints such as the shoulder demonstrate a greater range of movement when compared to lower body joints such as the hip (Tortora and Grabowski, 2003). As a result of potentially greater ranges of movement patterns, there may be greater changes to upper body movement patterns in

order to maintain power output than expected for lower body exercise. Although the key reasons for greater fatigue during an upper body WAnT remains unreported, fatigue may affect movement patterns and the subsequent biomechanics by decreasing proprioceptive sense, which increases shoulder movement and impacts performance (Lee *et al.*, 2003b, Taylor *et al.*, 2000, Carpenter *et al.*, 1998, Voight *et al.*, 1996). Although no studies have reported electromyographic (EMG) responses during upper body WAnTs, near-infrared spectroscopy studies suggest changes in muscle recruitment patterns exist (Kounalakis *et al.*, 2009). Changes in shoulder girdle kinematics and muscle coordination during high intensity shoulder elevation exercise has shown fatigue, as measured through EMG activity, in a number of muscles, especially the infraspinatus and deltoid muscles (Ebaugh *et al.*, 2006). Therefore, the use of motion analysis and EMG may highlight significant changes in limb kinematics and muscle recruitment patterns to enhance our understanding and interpretation of power production and the effects of fatigue during upper body exercise (Zehr and Chua, 2000).

In order to examine some of the mechanisms underlying fatigue during short duration high intensity exercise the WAnT was used. The WAnT allows the manipulation of power output and movement speed by using different resistive loads. Measuring biomechanical (EMG and motion analysis) and performance indices (power output; Watts and cadence; $rev \cdot min^{-1}$) over a range of loadings (2%, 3%, 4% and 5% of body mass; BM) will enable the study of fatigue during a range of maximal intensity exercise conditions. By manipulating the force production (resistive load) and potentially the rate of fatigue development, will enable the relationship between physiology, biomechanics and WAnT performance to be analysed and may allow a model of fatigue during upper body WAnTs to be developed. In addition, the optimal resistive load for upper body WAnTs has not been thoroughly examined since the original suggestion of 6% body mass resistive load (Dotan and Bar-Or, 1983), other studies have employed resistive loads of 4% body mass (Hubner-Wozniak *et al.*, 2004, Weber *et al.*, 2006, Aschenbach *et al.*, 2000, Biggerstaff *et al.*, 1997) and 5% body mass (Aziz *et al.*, 2002, Lovell *et al.*, 2011b, Buśko, 2011, Smith *et al.*, 2007b). Although it is acknowledged that an optimal load cannot be achieved for all parameters in the same test (Dotan and Bar-Or, 1983).

Therefore, the aims of this study were to examine the relationship between upper body WAnT performance and the underlying physiological and biomechanical factors (power output, cadence, fatigue index, EMG; amplitude, motion analysis; trunk movement distance, elbow range of motion, trunk rotational velocity), and to examine the optimal resistive loading for a 30 s upper body WAnT.

4.2 Method

4.2.1 Participants

Thirteen participants (age = 21.8 ± 5.2 years, mass = 78.3 ± 9.2 kg, height = 1.77 ± 0.07 m) with no previous arm crank ergometry experience volunteered to take part in this study. Participants did not participate or train in or for upper body sport or exercise. Each participant was tested within ± 1 hour of the first test with a minimum of 48 hours between tests. Participants were instructed not to conduct new training, or any vigorous training prior to each test. All tests were conducted in the same laboratory with the temperature at 20° ± 1°C. The study was approved by the University's Post Graduate Research Ethics Committee (Appendix 2).

4.2.2 Exercise protocol

After a full familiarisation session, participants completed four, seated upper body WAnT's, conducted as reported in section 3.3.1 using Cranlea Wingate (v.4.00; Cranlea & Company, Birmingham, UK). Resistive loads were 2%, 3%, 4% and 5% of body mass. The order of testing was balanced using a 4 × 4 × 4 Latin square to allocate the order of tests, with a minimum of 48-h between trials. Body mass taken at the familiarisation session was used as the reference mass for all subsequent tests. Corrected and uncorrected peak power output (PPO; over 1 s duration) and mean power output (MPO; over 29 s duration) and minimum power output (PO_{min}) were recorded. Mean cadence ($rev \cdot min^{-1}$), final cadence and time to peak power output (PPO_{time}; 1 s) values were also recorded.

4.2.3 Electromyography

Electrodes were placed on the following sites: flexor carpi ulnaris (FCU), biceps brachii (BB), triceps brachii lateral (TB), anterior deltoid (AD), infraspinatus (IS), external oblique (EO), vastus medialis (VM), lateral soleus (LS). A passive reference electrode (Blue sensor M-00-S, Ambu Ltd, Cambs, UK) was placed

centrally on the right patella (Section 3.4.5.i). Prior to electrode placement, all sites were cleaned with isopropyl alcohol to remove oil and dirt. Double-differential (16-3000Hz bandwidth, x300 gain), bipolar, active electrodes (MP-2A, Linton, Norfolk, UK) were firmly taped to the skin surface with the wires also taped down to reduce movement noise artefact. Electrode sites were marked on the participants' skin with a surgical marker pen to enable reliable electrode placements during subsequent tests, for full processing information e.g. collection frequency, normalisation etc are described in section 3.4.5.

4.2.4 Kinematic analysis

Infrared reflective markers were attached using double-sided tape on the following anatomical landmarks; ulnar styloid process; lateral epicondyle of the humerous; most prominent superior position on the conoid tubercle on the clavical and 7th cervical vertebrae (C7) (Figure 4.1). The ACE was also marked with infrared reflective markers at the following static points; directly vertical to centre of bottom bracket (C), and top centre of seat post pillar (A) (Figure 4.1). For additional anatomical landmarks and ACE markers are given in Table 3.12. After each participant's first test the placement of each marker was marked on the participant's skin with a surgical marker pen as for the EMG measurements.

The joint angle (°) between the wrist, elbow and inner shoulder was used to determine the elbow range of motion and is analogous to that of the knee joint in cycling (Zehr and Chua, 2000). The distance (mm) between C7 and the static point directly vertical to the centre of ergometer bottom bracket gave an analysis of forwards and backwards upper body movement. Angular velocity ($°\cdot s^{-1}$) between C7, shoulder inner and top and centre of seat post pillar, gave an analysis of the velocity and change in direction of the trunk (trunk rotation) in relation to the ergometer. The above variables were analysed at points corresponding to corrected and uncorrected peak power output and at minimum power output (29 s). A full description of each marker position is presented in Table 3.12. The motion analysis system and EMG system data where synchronised at the start of each test, further details are given in section 3.6.

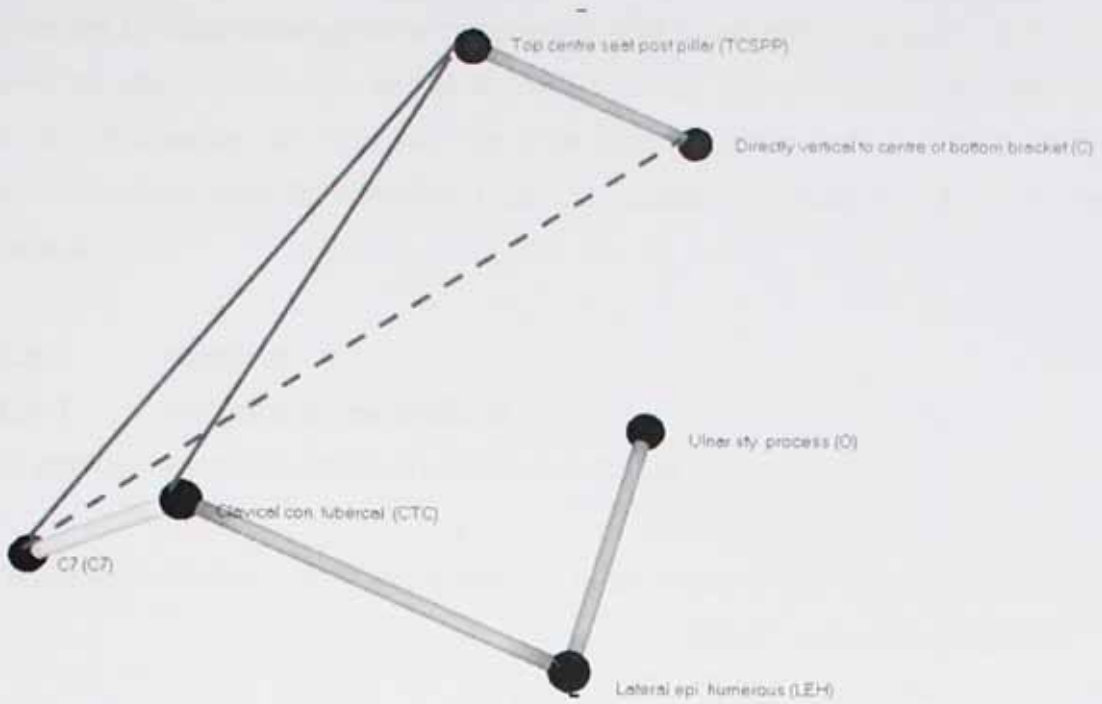


Figure 4.1. Example of kinematic analysis showing anatomical and static markers for analysis. The solid line represents the joined points of trunk rotation. The dashed line represents the joined points of the two markers for distance.

4.2.5 Statistical analysis

The data are presented as the mean ± standard deviations (*s*). To aid clarity standard deviations are plotted on line graphs for top and bottom lines as the standard deviations were fairly equal across data sets. All analyses were performed using Statistical Package for Social Sciences (v 17.0; SPSS Inc., Chicago, IL). Measures of peak power output (corrected and uncorrected), mean power output, peak and mean cadence were analysed between resistive loadings using separate single factor analysis of variance. The EMG activity analysed corresponded to corrected and uncorrected PPO and PO_{min} time points. Therefore, EMG data was analysed over time (PPOcorrected *vs* PPOuncorrected *vs* PO_{min}) and between resistive loadings (2%, 3%, 4% and 5% body mass) using a two-factor with repeated measure on both factors (power output × loading). Each muscle was analysed separately. The kinematic variables were analysed at the same time points and using the same statistical tests as for the EMG data time points corresponding to the occurrence of corrected PPO, uncorrected PPO and PO_{min}. Where SPSS presented *P* values of $P = 0.000$ these are reported as $P < 0.001$. Bonferroni post-hoc correction was undertaken when there were multiple comparisons for data sets for, performance indices, heart

rate data, electromyography responses and kinematic analysis. A two-tailed level of significance was set at $P \leq 0.05$ for all tests unless otherwise specified. In the discussion, to interpret the true meaningfulness of the data, test-retest power output data for the WAnT (as discussed in section 3.3.1.i) has been included.

4.3 Results

4.3.1 Performance indices

All performance variables are shown in Table 4.1.

Table 4.1 Performance variables for each upper body WAnT (mean ± *SD*).

		Resistive loading (% BM)			
		2%	3%	4%	5%
Uncorrected	PPO (W)	276 (42)	367 (46)[a]	427 (86)[a]	482 (76)[b]
	MPO (W)	239 (30)[c]	317 (30)[d]	364 (59)	405 (47)
	PO_{min} (W)	219 (19)	270 (25)	301 (47)	321 (48)
	PPO_{time} (s)	11 (3)	10 (4)	11 (3)	9 (3)
	FI (%)	20 (9)[e]	26 (7)	28 (11)	32 (12)
Corrected	PPO (W)	621 (149)	627 (100)	591 (143)	613 (95)
	MPO (W)	296 (30)[c]	351 (31)[e]	389 (60)	423 (50)
	PO_{min} (W)	196 (79)	223 (51)	275 (62)	285 (69)
	PPO_{time} (s)	4 (3)	3 (3)	5 (2)	6 (3)
	FI (%)	65 (19)[f]	64 (9)	52 (11)	53 (12)
Cadence	Peak ($rev \cdot min^{-1}$)	180 (18)[c]	160 (12)[d]	139 (19)	125 (13)
	Mean	156 (14)[c]	138 (10)[d]	119 (14)	106 (11)
	Minimum	143 (14)[c]	118 (13)[d]	99 (16)	85 (16)

a. Significantly different from 2% resistive loading.

b. Significantly different from 2% and 3% resistive loading.

c. Significantly different from 3%, 4% and 5% resistive loading.

d. Significantly different from 4% and 5% resistive loading.

e. Significantly different from 5% resistive loading.

f. Significantly different from 4% resistive loading.

Note: FI is fatigue index. BM is body mass (kg)

4.3.1.i Peak power output

Significant differences were observed between resistive loads for uncorrected PPO ($F = 23.578$, $P < 0.001$) with mean values increasing with resistive load (Table 4.1). Post-hoc analysis revealed that uncorrected PPO using the 5% resistive loading was greater than for both the 2% and 3% resistive loads ($P < 0.001$, $ES = 1.70, 1.35$). Differences were also noted between the 2% and 3% resistive loads ($P = 0.005$, $ES = 1.43$) and the 2% and 4% resistive loads ($P < 0.001$, $ES = 1.48$). No differences were observed for corrected PPO although there were variations across all four resistive loads. Mean values for corrected PPO were 591-627 W. Therefore, this reflects the method of calculation which accounts for the power required to accelerate the flywheel and the data suggested that corrected peak power is independent of resistive load.

4.3.1.ii Mean Power Output

Uncorrected MPO demonstrated a significant difference between resistive loads ($F = 35.490$, $P < 0.001$; Table 4.1) with values increasing with each resistive load. There was a significant difference between 2% *vs* 3%, 4% and 5% ($P = < 0.001$) and 3% *vs* both 4% and 5% ($P = 0.048$ and $P < 0.001$, respectively). Significant differences between resistive loads were also observed for corrected MPO ($F = 19.607$, $P < 0.001$; Table 4.1) with values increasing with each resistive load ($P < 0.05$). Significant differences were observed between 2% and 3%, 4% and 5% ($P = 0.017$, $P < 0.001$, $P < 0.001$, respectively) and between 3% *vs* 5% ($P = 0.001$). Therefore, this reflects the method of calculation for corrected power whereby deceleration from corrected PPO results in a greater loss of power for a lighter than heavier resistive load.

4.3.1.iii Time to peak power output

Although time to peak power output for uncorrected and corrected data were different ($F = 81.378$, $P < 0.001$, $ES = 1.32$) values were not significantly different between resistive loads ($P > 0.05$). Mean values for corrected and uncorrected time to PPO were approximately 4-5 s and 10 s, respectively ($P < 0.001$). Therefore, time to peak power is not dependent on load but time to PPO is dependent on whether corrected or uncorrected power data is used.

4.3.1.iv Cadence

There was a significant difference in the peak cadence achieved between resistive loads ($F = 29.927$, $P < 0.001$; Table 4.1). As resistance increased, peak cadence decreased with significant differences observed between the 2% *vs* 3%, 4% and 5% ($P = 0.011$, $P < 0.001$, $P < 0.001$, respectively) and 3% vs both 4% and 5% resistive loadings ($P = 0.009$, $P < 0.001$, respectively). Similarly to peak cadence, there was a significant difference in mean cadence across resistive loads ($F = 41.124$, $P < 0.001$). As resistive load increased mean cadence decreased (Table 4.1) with significant differences observed between 2% *vs* 3%, 4% and 5% ($P = 0.004$, $P < 0.001$, $P < 0.001$, respectively) and 3% *vs* both 4% and 5% ($P = 0.001$, $P < 0.001$, respectively). There was a significant difference in minimum cadence across resistive loads ($F = 38.966$, $P < 0.001$). As resistive loads increased minimum cadence decreased (Table 4.1) with significant differences between 2% and 3%, 4% and 5% ($P < 0.001$) and 3% vs 4% and 5% ($P = 0.008$, $P < 0.001$, respectively). The absolute decrease in cadence (~40 rev·min^{-1}) was similar for each resistive load (Table 4.1). Therefore, as resistive load increases all measures of cadence at that load decrease.

4.3.1.v Fatigue Index

There was a significant difference in the fatigue index for uncorrected PPO ($F = 4.068$, $P < 0.022$; Table 4.1). As resistance increased fatigue index increased with significant differences observed between 2% *vs* 5% ($P = 0.016$). There was a significant difference in fatigue index for corrected PPO ($F = 4.068$, $P = 0.012$; Table 4.1). As resistance increased fatigue index decreased with significant differences observed between 2% *vs* 4% ($P = 0.05$).

4.3.2 Heart rate data

There were no significant differences for peak heart rates (HR; beats·min^{-1}) at 29 s between resistive loads of 2%, 3%, 4% and 5% (166, 167, 166, 169, respectively). Therefore, peak heart rate can be achieved regardless of resistive load.

4.3.3 Electromyography responses

Electromyographic activation was measured for each muscle at corrected PPO, uncorrected PPO and minimum power output. For bicep brachii there were

differences in peak EMG activity between resistive loads for uncorrected PPO (F = 3.01, P = 0.04), corrected PPO (F = 4.12, P = 0.011) and PO_{min} (F = 4.92, P = 0.005). Post-hoc testing for uncorrected and corrected PPO revealed significant differences between 2% and 5% (P = 0.03, ES 1.11, P = 0.012, ES = 0.94, respectively) and both 2% and 3% *vs* 5% (P = 0.006, ES = 1.18 and P = 0.027, ES = 1.04, respectively; Figure 4.2) for PO_{min}. Therefore, the data suggested that as resistive load increases biceps brachii activation also increases.

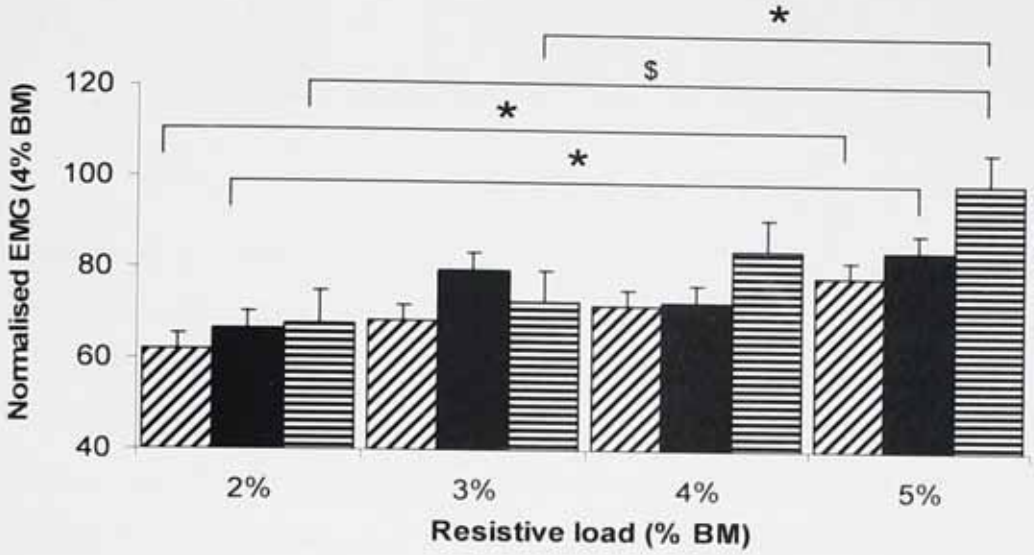


Figure 4.2 Biceps brachii normalised EMG (4% BM) against resistive loads (% BM) for uncorrected and corrected PPO and PO_{min}.
* significant difference ($P \leq 0.05$) between resistive loads.
$ significant difference ($P < 0.01$) between resistive loads.
Note: hashed lines represent uncorrected power output, full boxes represent corrected power output and vertical lines represent minimum power output.

For triceps brachii there were differences between resistive loads for uncorrected PPO (F = 3.01, P = 0.04) and PO_{min}, (F = 8.714, $P < 0.001$). Triceps brachii approached significance for uncorrected PPO between 2% *vs* 5% (P = 0.081). For PO_{min} post-hoc testing revealing significance between 2% *vs* both 3% and 5% (P = 0.015, ES = 1.00 and $P < 0.001$, ES = 1.21, respectively) and significance being approached at 2% *vs* 4% (P = 0.081, ES = 1.05; Figure 4.3).

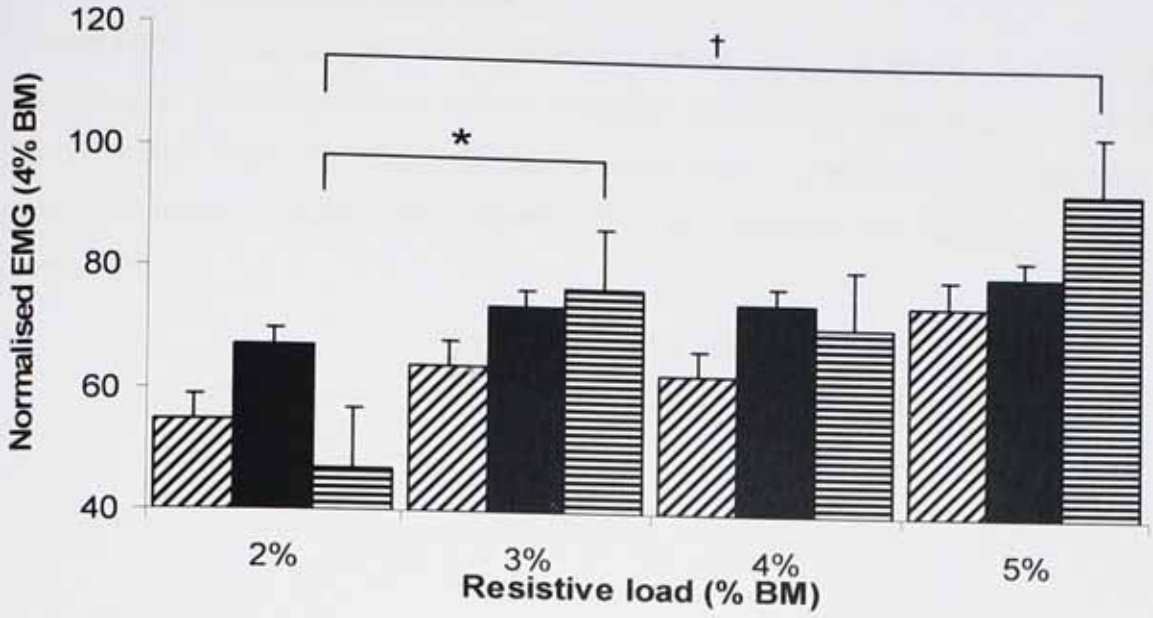


Figure 4.3 Triceps brachii normalised EMG (4% BM) against resistive loads (% BM) for uncorrected and corrected PPO and PO_{min}.

* significant difference ($P \leq 0.05$) between resistive loads.

† significant difference ($P < 0.001$) between resistive loads.

Note: hashed lines represent uncorrected power output, full boxes represent corrected power output and vertical lines represent minimum power output.

There was a significant difference for flexor carpi ulnaris at PO_{min} ($F = 3.13$, $P = 0.034$), with post-hoc testing revealing significance being approached at 2% *vs* 5% resistive load ($P = 0.57$, $ES = 0.97$). Anterior deltoid was significant at PO_{min} ($F = 6.55$, $P = 0.001$) with post-hoc testing indicating significantly greater activation at 5% *vs* 2%, 3% and 4% ($P = 0.02$, $P = 0.04$, $P = 0.036$, respectively). The external oblique demonstrated a significant difference for corrected PPO ($F = 2.787$, $P = 0.052$) with post-hoc testing indicating significance was approached between 2% *vs* 5% ($P = 0.068$, $ES = 0.84$). Power output minimum was also significant ($F = 6.034$, $P = 0.002$) with post-hoc testing indicating significantly reduced activation between 2% *vs* both 4% and 5% ($P = 0.038$, $ES = 1.27$, $P = 0.001$, $ES = 1.23$, respectively). There were no significant differences for vastus medialis and lateral soleus EMG activation. Therefore, vastus medialis and lateral soleus activation is not dependent on resistive load.

4.3.4 Kinematic analysis

There were no significant differences for elbow range of motion between resistive loadings or for comparison within resistive loads compared to corrected PPO, uncorrected PPO and PO_{min}, although there were variations across all four resistive loads (Figure 4.4). Figure 4.5 is an example of changes across a single load.

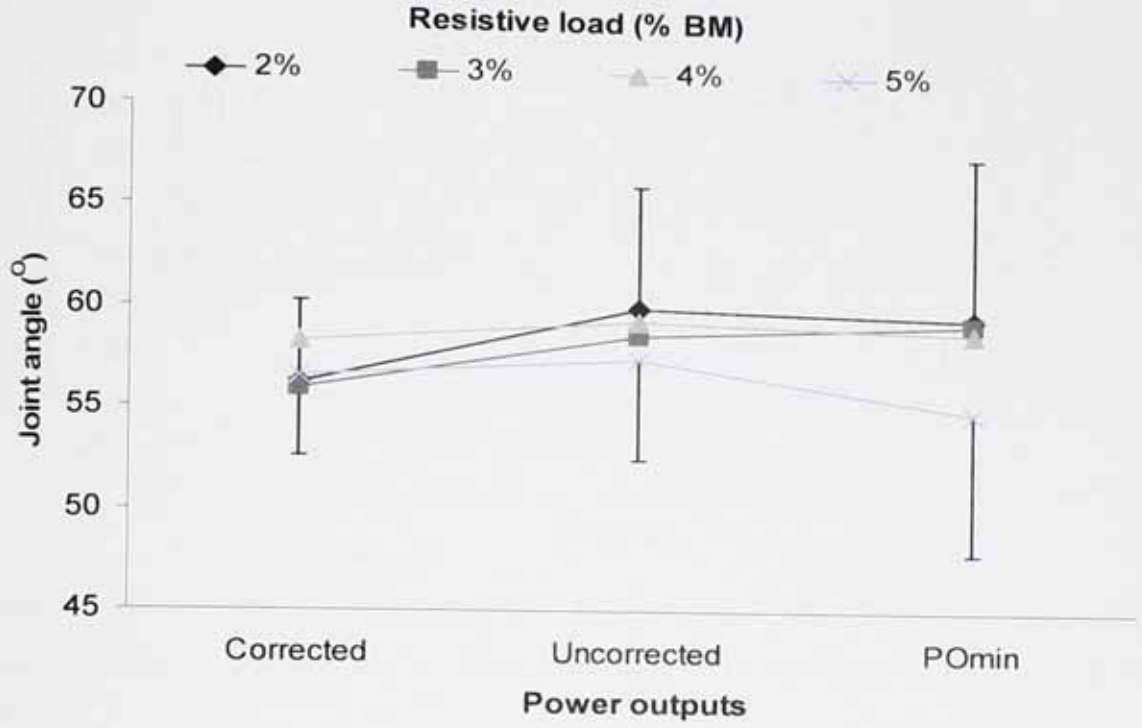


Figure 4.4 Changes in elbow joint angle (°) against resistive loads corresponding to corrected and uncorrected PPO and PO_{min}.

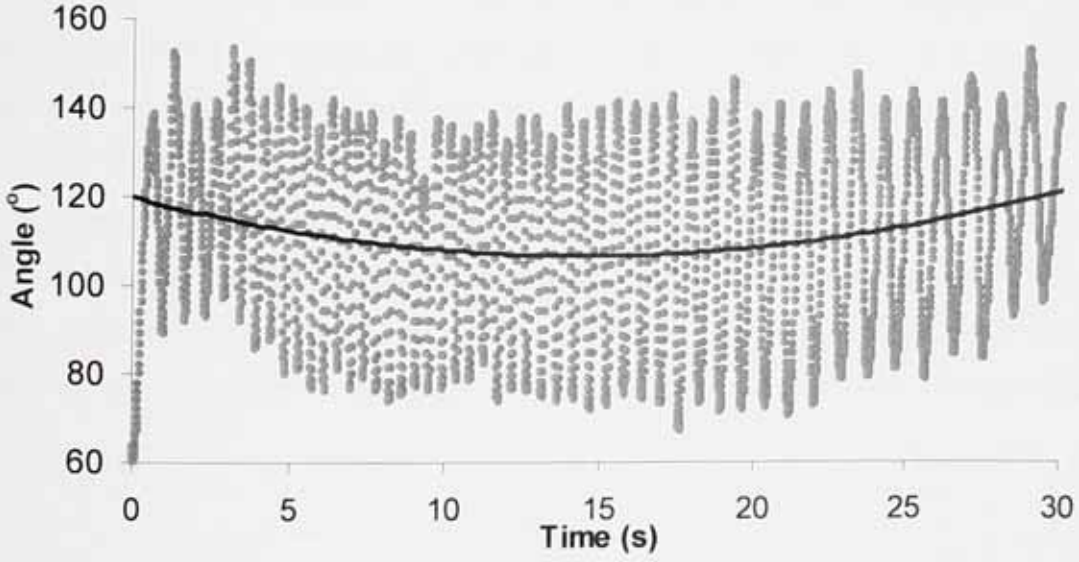


Figure 4.5 Changes in elbow joint angle (°) for a typical participant. Data is shown for a resistive load of 4% body mass with a 2nd order polynomial trendline.

There was a significant difference observed for trunk rotation measured as angular velocity ($°\cdot s^{-1}$) between resistive loadings ($F = 2.856$, $P = 0.040$). Post-hoc testing revealed a significant difference between 3% and 4% resistive loads ($P = 0.029$) with angular velocity being greater for 3% than 4% (282 *vs* 234 $°\cdot s^{-1}$). Figure 4.6 is an example of changes across a single load.

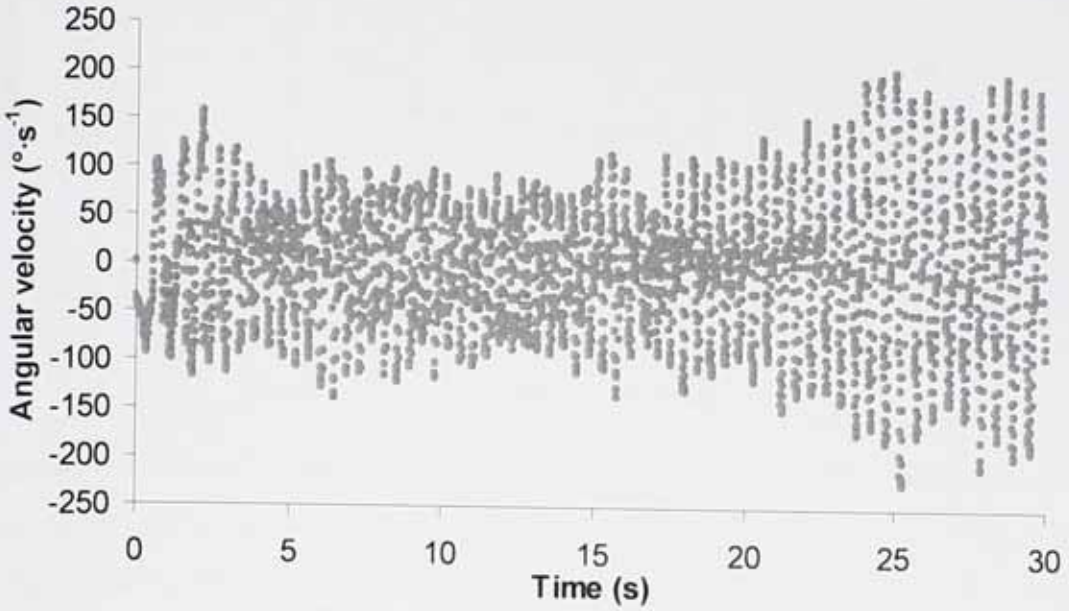


Figure 4.6 Changes in angular velocity between C7 and inner shoulder and seat post for a typical participant. Data is shown for a resistive load of 4% body mass.

There was a significant difference in changes in torso distance (C7; mm) relative to the ACE between resistive loads ($F = 5.135$, $P = 0.002$; Figure 4.7 and 4.8). Post-hoc testing revealed a significant difference between 2% and 4% resistive loads ($P = 0.001$). Additionally, distance at corrected, uncorrected and minimum PO was significantly different ($F = 3.124$, $P = 0.047$). Post-hoc testing revealed a significant difference between minimum PO and uncorrected PPO ($P = 0.047$) with distance (mm) being greater at minimum power output than uncorrected PPO. Therefore, resistive load effects torso distance to the ACE, in general a greater load results in participants moving their torso closer to the ACE.

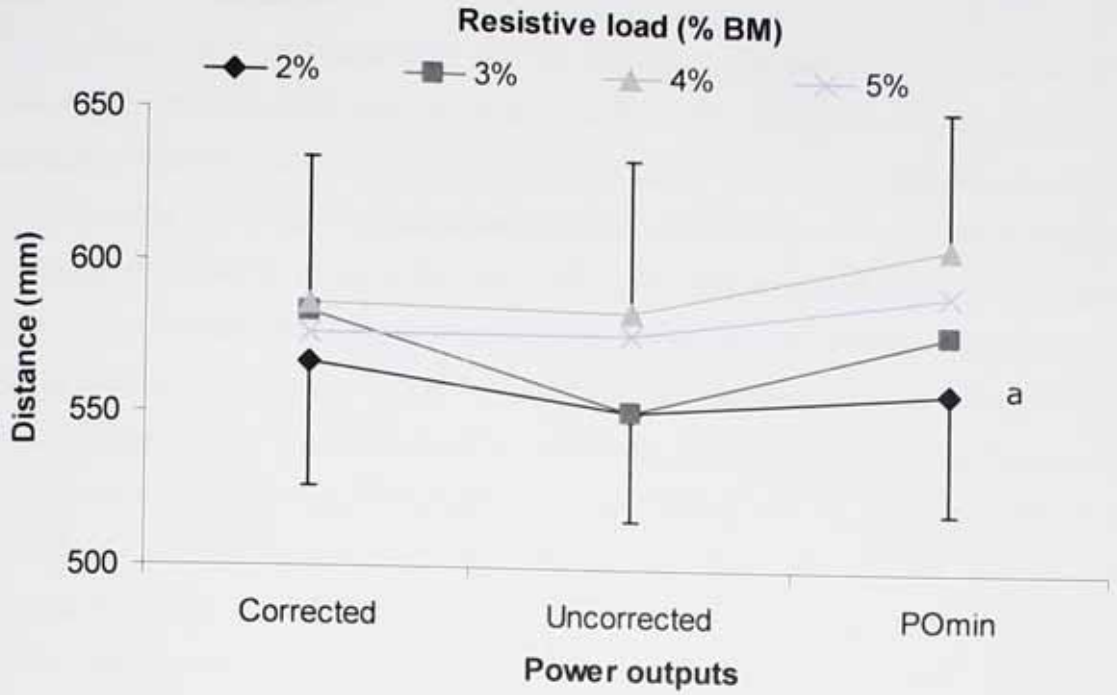


Figure 4.7 Torso distance (mm) relative to the ACE at corrected, uncorrected and minimum power output for 2%, 3%, 4% and 5% body mass resistive loads. [a]. Significantly different from 4% resistive loading.

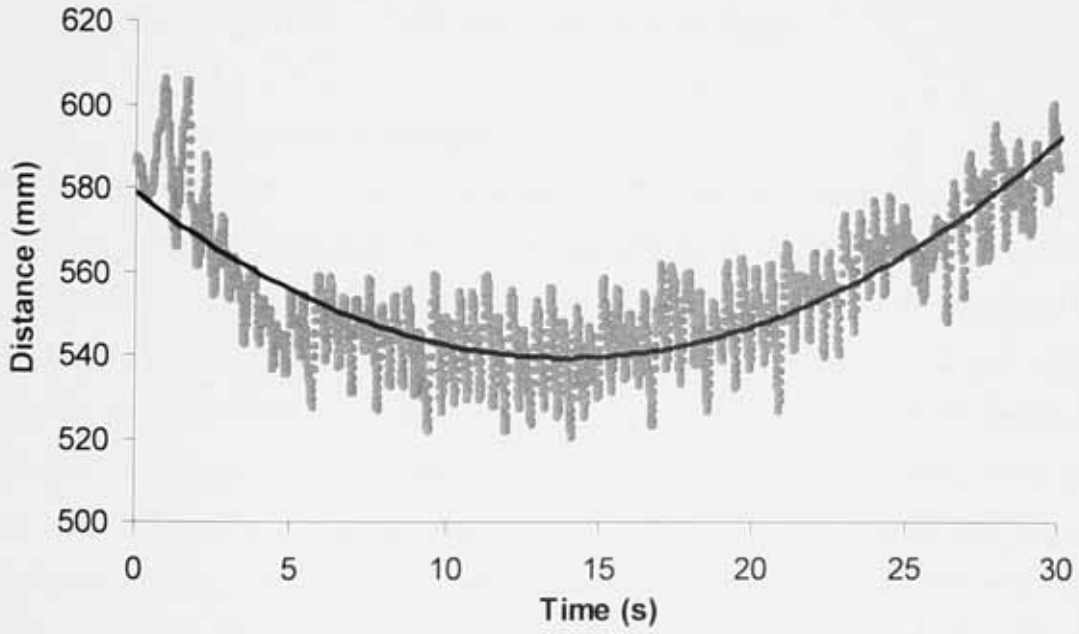


Figure 4.8 Changes in torso distance (mm) relative to the ACE for a typical participant. Data is shown for a resistive load of 4% body mass.

4.4 Discussion

The primary aim of this study was to examine the various parameters that may influence performance and fatigue between physiological and biomechanical variables during maximal, high intensity upper body exercise at varying loads. These loads were manipulated by changes in resistance with participants instructed to performance the test 'all out'. The key findings of the study were that uncorrected PPO increased with resistive load whereas corrected PPO did not differ between resistive loads. Peak cadence decreased with greater resistive load whereas the absolute drop in cadence was similar for all loadings. Results from the EMG analysis demonstrated the biceps brachii to be predominately affected by resistive load at PPO whereas all upper body sites demonstrate an increase in activity at fatigue/minimum power. In comparison to the power and EMG responses, kinematic analysis showed relatively few significant changes. However, there were changes in torso distance and trunk rotational velocity in relation to the ACE and resistive load. Although previous studies have examined resistive loads and corrected PPO during lower body exercise this is the first study to comprehensively examine uncorrected and corrected PPO and mean PO during upper body WAnT with various resistive loads.

4.4.1 Peak power output

The values of corrected and uncorrected PPO were similar to those reported in previous studies (494-629 W) (Mermier, 2000, Kounalakis *et al.*, 2009, Smith *et al.*, 2007b, Smith and Price, 2007) but lower than uncorrected values for javelin throwers (720 W; Bouhlel *et al.*, 2007) and wrestlers (670-732 W; Hubner-Wozniak *et al.*, 2004, Lutoslawska *et al.*, 2003). Values of MPO were also similar to those reported for recreationally active participants (462-466 W; Lovell *et al.*, 2011b, Smith *et al.*, 2007b, Marsh *et al.*, 1999, Arslan, 2005) and high-school wrestlers at age 17 years and above (432 W; Terbizan and Seljevold, 1996) and higher than those reported for climbers (328 W; Mermier, 2000). Therefore, the performance variables were representative of the population studied.

4.4.1.i Uncorrected peak power output

The uncorrected PPO was dependent on the resistive load applied. As resistive load increased uncorrected PPO also increased which is in agreement with previous lower body WAnT studies (Lakomy, 1985, Winter *et al.*, 1996, James *et al.*, 2007b) and lower and upper body ergometry studies (Dotan and Bar-Or,

1983). In the current study the main differences in uncorrected PPO were between the lowest and highest resistive loads (2% *vs* 5%), as well as between the two lowest loads (2% *vs* 3%). Although the uncorrected PPO increased with resistive load, the peak cadence decreased with resistive load. However, the relationship between PPO and cadence (i.e. ratio of peak cadence to peak power) was not linear. This response is in accordance with the force velocity relationship where greater concentric movement speeds elicit lower force (Brooks *et al.*, 2005). Cadence at PPO for the 5% resistive load was 125 rev·min^{-1}, this is the optimal cadence recommended by Neville (2009) for America cup sailors undertaking grinding. Additionally, this cadence is approaching optimal power/velocity relationship of 120 rev·min^{-1} for an even distribution of type I/II muscle fibres in lower body ergometry (Sargeant, 1994). Given the greater proportion of type II fibre in the upper limbs (Mygind, 1995, Savard *et al.*, 1987, Sawka, 1986) it is likely that a 5% resistive load satisfies the optimal force/velocity relationship for uncorrected PPO.

4.4.1.ii Corrected peak power output

Corrected PPO was independent of resistive load, as observed in previous lower body studies (James *et al.*, 2007b, Linossier *et al.*, 1996, Bogdanis *et al.*, 2008). However, a previous study of lower body WAnT's by Lakomy (1985) concluded that lower restive loads produced greater corrected PPO. Although not statistically significant, the results from this study suggest that the 2% and 3% body mass resistive loads produce a slightly greater PPO than the 4% loading of ~32 W, which is greater than the day-to-day variation observed earlier in the thesis (~18 W). Therefore, it is possible that the optimal cadence is faster with the arms (Vanderthommen *et al.*, 1997) and each individual resistive loading may produce an optimal acceleration up until the time where corrected PPO is reached.

4.4.1.iii Time to peak power output

Time to PPO for uncorrected and corrected measures was significantly different. Time to PPO was faster for corrected measures. Furthermore, time to PPO was independent of resistive load applied for both corrected and uncorrected PPO. Studies examining lower body WAnTs have demonstrated comparable results for uncorrected time to PPO but not corrected time to PPO which increased with resistive load (James *et al.*, 2007b). The present study demonstrated that the

time to corrected PPO was reached more quickly than time to uncorrected PPO and corresponds to that reported for lower body WAnTs (James *et al.*, 2007b, Lakomy, 1985, Lakomy, 1986, Vanderthommen *et al.*, 1997). As corrected PO takes into account the inertia of the flywheel and the force required to change the momentum of the flywheel (Bassett, 1989), the initial power phase produces the highest peak values and does not, unlike uncorrected PPO, necessarily occur at peak cadence (Vanderthommen *et al.*, 1997). Uncorrected PPO occurred at the same time (s) across loads, but resulted in lower absolute peak cadences, reflects the greater resistive load applied, which limits peak cadence.

At lower cadences muscle contraction speed is also lower resulting in the generation of large forces and more rapid acceleration of the flywheel (Sargeant *et al.*, 1981). With little difference in time to corrected PPO across resistive loads, it possible that there is a similar pattern and time course of muscle fibre recruitment up to the point of corrected PPO. However, from this point power then decreases at different rates – it becomes more reflective of the resistive load. This could reflect a decrease/shift in optimal energy usage pattern, from PCr degradation. Uncorrected time to PPO is only reached when velocity slows down between 9–11 s (MacIntosh *et al.*, 2003). The reduction in cadence, and therefore power output, for the remainder of the test could be reflective of the point where the maximal rate of glycolysis and ATP turnover begins to decline (Beneke *et al.*, 2002, Bogdanis *et al.*, 2008, Gastin, 2001) and therefore the start of metabolic fatigue.

4.4.1.iv Fatigue index

Fatigue index calculated from uncorrected performance indices increased with resistive load, which has been observed in both arm and leg studies (Dotan and Bar-Or, 1983, James *et al.*, 2007b). In the present study, each resistive load demonstrated a similar absolute decrease in cadence (~40 rev·min^{-1}). Therefore, as a lower resistive load initially enables a greater peak cadence to be produced, the absolute drop in cadence is relatively less than for 5% resistive load where peak cadence is initially less. For uncorrected data the absolute drop in power is therefore greater as the resistive load increases. The significant increase in fatigue index (uncorrected power) from 20% to 32% with the 2% versus 5% resistive loadings, is an indication that the 2% resistive load is in the extreme end of the force-velocity curve to reach a sufficiently high power output;

i.e. it is likely that cadence is approaching a maximal rate of ~180 rev·min^{-1} (optimal speed being 25-30% of maximal speed of contraction; Astrand and Rodahl, 1986). A fatigue index of 20% for a 2% resistive load still indicates that the resistive load is sufficient to elicit fatigue and a drop in power output. Whether there were biomechanical differences as a result of this will be discussed in sections 4.4.2 and 4.4.3.

For corrected performance indices, the PPO was similar across loads; as such, the FI became a function of the minimum power. In the current study minimum PO increased at greater resistive loads presumably as the flywheel slowed down towards end of test due to greater resistance on the flywheel. Therefore, participants were credited with a greater amount of work being done as they are more able to 'resist' the deceleration of the flywheel at greater loads (Bogdanis *et al.*, 2008). With lighter resistive loads, the flywheel presumably 'spins' more and participants are then not credited with as much work being done, i.e. the participants were less able to 'resist' the deceleration of the flywheel. Therefore, corrected FI is almost exclusively dependent on minimum power output. For corrected FI a significant difference was observed between the 2% and 4% resistive loads, although the mean figures indicate a division between 2% and 3% and 4% and 5% body mass loadings. Therefore, although it may be expected that a greater load would result in greater fatigue, the measurement of correct power indicates that when using the standard measurement of FI that fatigue is greater with a lighter load, which has also been observed in leg ergometry (Bogdanis *et al.*, 2008).

Practically the results suggest that application to sports settings may be of benefit in upper body sports. Rowers vary their speed during a race by rapidly increasing boat velocity after the start of the race, decreasing and then increase speed again toward the end of the race (Astrand and Rodahl, 1986). Corrected PPO would be of practical significance to rowers at the start of the race, where the ability to rapidly increase the speed of the boat is required. This may be especially important to 200-m sprint kayaking where the race lasts less than 40-s (van Someren and Palmer, 2003) and the ability to accelerate the kayak rapidly is necessary before the race is finished.

4.4.1.v Resistive load optimisation

The resistive loads eliciting the greatest PPO (i.e. the optimal resistive load) are dependent on whether corrected or uncorrected PO is required. Corrected PPO is independent of resistive load and a lower resistive load (2% or 3%) could then be used. Participants in this study, using a lower resistive load, reported fewer side-effects associated with the WAnT (e.g. nausea and vomiting and dizziness; Inbar *et al.*, 1996, Stickley *et al.*, 2008, Marquardt *et al.*, 1993) which can affect test validity or repeatability and alleviate the need for abbreviating the test duration to 20-s (Smith *et al.*, 2007b, Laurent *et al.*, 2007). Therefore, for uncorrected PPO the greater the resistive load the greater the PPO and MPO.

For uncorrected power, the restive load of 5% produced 12 of the 13 highest PPOs with the remaining highest uncorrected PPO being achieved with the 4% body mass load. Corrected PPO produced some variation in individual PPO between loads of 2%, 3%, 4% and 5% with 23%, 46%, 15%, 15% of highest PPO achieved at each loading, respectively. With uncorrected power, the difference between 4% and 5% BM is 55 W, which is greater than the variation demonstrated earlier in the thesis. Therefore, it is suggested that a 5% BM resistive load is used to elicit maximal power. The variation between corrected power outputs, excluding the 4% BM, is relatively small suggesting (10-15 W) either load may be applied.

Participants were familiarised at 4% BM resistive load as it formed a compromise between the lower loads (2% and 3%) and the higher load of 5%. It would not be practical to familiarise participants at all resistive loads, and the results suggest that participants' performance variables were not preferential over the other BM loads. Despite the order of loading being randomised, there is a slight decrease in corrected PO achieved at 4% resistive loading. Although not investigated in the literature, one consideration could be that as the three warm-up sprints for all resistive loads were completed at 4% BM, and this may have 'over-familiarised' participants with the load. Therefore, it is the 'warm-up' sprints, not the load that over-familiarised participants to a 4% BM resistive load. Another consideration is that although the 4% resistive load was a compromise between resistive loads of 2%, 3% and 5% BM, this may not be beneficial to performance at a 4% resistive load. In future, further consideration may need to be given to the warm-up load, e.g. whether a 3% load could provide

improvement in performance for a 30-s 4% BM load. Finally, it may be that a 4% load produces different responses in biomechanics that result in the resistive load not being optimal for corrected power output and thus may warrant further study.

4.4.2 Electromyography responses

Analysis of normalised surface EMG amplitude can help in performance analysis as it reflects the level of recruitment and general levels of muscle excitation within the area detected by the electrode (Hug and Dorel, 2009). Wingate anaerobic test performance and EMG activity has been examined using lower body tests (Greer *et al.*, 2006, Hunter *et al.*, 2003, Rana, 2006, Stewart *et al.*, 2011, Chtourou *et al.*, 2011). The hamstrings and quadriceps provide the pull and push forces within a lower body WAnT during cycling (Vanderthommen *et al.*, 1997, Marais *et al.*, 2004, Hopman *et al.*, 1995, Zehr and Chua, 2000) the biceps and triceps brachii provide the equivalent pull and push forces respectively for primary power during upper body WAnT performance. This was the first study to examine EMG responses during an upper body WAnT. The power output and FI results discussed above provide an indication of performance in relation to resistive load. However, more detail is required to develop an accurate model of muscle activation and how it may influence performance.

Whether considering corrected or uncorrected power variables the biceps brachii muscle demonstrated an increased activity proportional to resistive load. Biceps brachii activation have been demonstrated to increase with constant load at high intensity ACE (Bernasconi *et al.*, 2006). Therefore, the mean activation levels suggest that biceps brachii activation distinguishes between resistive loads with the level of activation being greater as resistive load increases. The results further suggest that there is greater biceps brachii activation at corrected PPO rather than uncorrected PPO across all four resistive loads. It appears that for both corrected and uncorrected PO that a 2% resistive load for biceps brachii activation is considerably 'easier' than a 5% resistive load. At the end of the WAnT for all resistive loads, with the exception of 3% loading, biceps brachii activity was greater than at PPO either indicating fatigue had occurred and muscle activation was not effective in generating force (Greer *et al.*, 2006, Walker *et al.*, 2012) or that the flywheel had slowed sufficiently to allow greater

force to be applied. As the absolute decrease in cadence was equal across loads it is most likely that the former explanation is more probable. With the exception of the 3% loading, biceps brachii activity was greater at minimum power than at PPO. These data suggest that the biceps brachii are, for the population tested, an important muscle during an upper body WAnT. Whether this applies to a specifically trained population remains unreported.

The triceps brachii demonstrated lower EMG activation at minimum power output with 2% resistive load compared to 3%, 4% and 5%. This response may indicate that biceps brachii may be more important than the triceps brachii to rotate the cranks/flywheel at this point of the test. Lower limb studies indicate that there are changes in EMG amplitude during the WAnT and that some muscle may be more reflective of changes in power output than others (Greer *et al.*, 2006, Rana, 2006). Individual analyses of EMG responses indicate that at peak cadence the EMG activity was lower than at the end of the test. Participants' post-test comments indicated that they were unable to maintain the cadence as the flywheel was moving too fast and the hand grips were being 'pulled away from them'. With a 2% resistive load, at the end of the test participants exhibited a significant reduction in triceps brachii activation which may be an indication that the triceps brachii cannot contract fast enough and the cranks were spinning away from the participant whereas the biceps brachii activation was almost constant. However, as with the biceps brachii a 5% resistive load resulted in a significant increase in activation to limit power loss.

The EMG activation for flexor carpi ulnaris only showed significance differences at the 2% *vs* 5% resistive loads at the minimum PO. The lack of significance between other resistive loads and power outputs, despite a significant level of EMG activity, is suggestive of the need to maintain grip throughout the crank cycles for each test. Flexor carpi ulnaris activation could be linked to the EMG activity of the biceps brachii, as activity of biceps brachii increases so does flexor carpi ulnaris with the increase in pull requiring a stronger flexion of the hand. Greater muscle activity within flexor carpi ulnaris at the 5% resistive load is a new finding. Whether this level of activation is required during more prolonged high intensity upper body exercise (e.g. 100% max to exhaustion) and how it contributes to fatigue remains to be established.

The anterior deltoid indicated significantly greater activity for the 5% resistive load at the end of the test when compared to the other resistive loads. Resistive loads of 2%, 3% and 4% may therefore not be sufficient to require greater muscle activation at this site. A 5% body mass loading appears to be sufficient to increase activation as either a stabiliser or in assisting power production. Such a response has been suggested for infraspinatus during exercise at submaximal resistive loads (Bernasconi *et al.*, 2006) and serves to increase compression for the glenohumeral joint (Ackland and Pandy, 2009). Furthermore, a comparable level of activation across resistive loads and power outputs indicates this muscle acts as a stabiliser throughout all the resistive loads and does not fatigue. The resistive loads examined or the time points may not be sufficient to stimulate the muscle or alter the movement pattern, or it is not an important contributor to the movement pattern.

Similarly to the other EMG sites recorded, the external oblique showed greater activation at corrected PPO for the 5% compared to 2% load. It therefore appears that a 5% resistive load requires greater assistance from the trunk muscles than a 2% resistive load. Furthermore, at minimum PO the external oblique activation was greater at the 4% and 5% resistive loads when compared to 2% resistive load. This pattern of activity has only previously been suggested to occur during sub-maximal arm crank exercise (Mercier *et al.*, 1993, Stamford *et al.*, 1978, Bar-Or and Zwiren, 1975, Bernasconi *et al.*, 2006). However, where rectus abdominis activity has been specifically measured this muscle group has also been demonstrated to contribute to torso stabilisation (Hopman *et al.*, 1995) although this may be due to their greater contribution to trunk stabilisation than the external oblique. The current study is the first investigation to examine torso stabilisation via trunk rotational velocity at a range of WAnT resistive loads and the results show that trunk rotational velocity is important for resistive loads of 5%. For resistive loads of 2% body mass the 'spinning' affect at the end of the test could reduce the need for trunk stabilisation. Whether this activation is similar at submaximal or continuous exercise to exhaustion remains to be examined.

Previous studies have suggested that the lower limbs, in addition to the trunk muscles, aid power production as stabilisers during ACE possibly resulting in disproportionate metabolic cost of exercise (i.e. VO_2 excess) (Smith *et al.*,

2007a, Bar-Or and Zwiren, 1975). However, results of the present study showed no differences in EMG activity at these sites between resistive loads or over the test duration thus indicating that the lower limbs could be activated prior to PPO and were either outside of the time scale analysed, or not activated at all. At the end of the test this suggests that neither muscle stabilised the lower body significantly and that fatigue is unlikely to be a factor in these muscles. With participants 'firmly seated' and correctly positioned the external oblique, rather than the lower limbs, may be assisting trunk positioning and therefore aiding in power production for the upper body.

The increase in EMG seen in a number of muscles, but not all, at the end of the exercise, despite a reduction in power output, suggested this is the result of local muscle fatigue (Greer *et al.*, 2006, Walker *et al.*, 2012). With local muscle fatigue reducing contractile force this may be a result of increased muscle pH (Lovell *et al.*, 2011b, Smith *et al.*, 2002a, Weber *et al.*, 2006), an accumulation of Ca^{2+} (Green, 1997), or increase in inorganic phosphate due to the breakdown of creatine phosphate (Westerblad *et al.*, 2002). However, not all muscles showed changes in EMG which supports the notion of peripheral muscle fatigue - if all muscle showed an increase then fatigue may be central (Greer *et al.*, 2006, Walker *et al.*, 2012) but this is unlikely over a 30-s sprint test. In addition, muscles not showing increases or minimal changes in resistive loading probably indicate their role as stabilisers rather than power producers.

Dependent on the resistive load applied minimum PO resulted in greater EMG activation for a number of muscles. This response contributes to knowledge of submaximal muscle activation where EMG activation increased at two loads (50 and 100 W) (Smith *et al.*, 2008), although interesting for a 30-s WAnT the linear increase the resistive load is only significant at the end of the test. The lack of significant differences in EMG during the various resistive loads, with the exception of biceps brachii, could indicate that these muscles were fully utilised regardless of resistive load. Previous research has shown that during incremental ACE, swim trained leg disabled men showed greater change in muscle activation for the biceps brachii over the triceps brachii (Frauendorf *et al.*, 1989). Whatever the nature of fatigue this study demonstrated a decrease in PO over time with an increase in EMG activity. Such a drop in PO indicates fatigue in terms of reduced power production within the muscles, where there is

increased electrical, but less contraction, indicating a reduction in neuromuscular transmission and/or impaired excitation-contraction coupling (Hautier *et al.*, 2000). The EMG responses may be more easily detected in uncorrected FI compared with corrected FI. The significant difference observed in 2% *vs* 5% resistive load, reflected in the significant changes in EMG response. Uncorrected FI was only significant at 2% *vs* 4%, which was not reflective in EMG responses. It may be that EMG responses were not sampled at the correct time point(s) to reflect corrected FI with the muscles studied.

The above muscle recruitment patterns are the first to be reported for upper body WAnTs. Whether these activation patterns are typical for trained participants is not clear (Smith *et al.*, 2008, Marais *et al.*, 2004, Bernasconi *et al.*, 2006). A further study examining pre and post training changes in EMG could provide information as to whether training changes recruitment patterns and to what extent.

4.4.3 Kinematic analysis

Trunk rotation measured as angular velocity ($°\cdot s^{-1}$) between C7, shoulder inner and top, and centre of seat post pillar was only significant between 3% and 4% resistive loads. The 3% resistive load producing the greatest trunk rotational velocity at corrected, uncorrected and minimum PO. This resistive load could represent a point between the lighter 'spinning' resistive load of 2% and the heavier resistive loads of 4% and 5% that require greater stabilisation. Mean time to corrected PPO was fastest at this resistive loading and it may be that muscles used in creating the trunk rotational velocity are strong enough to overcome the resistive load at 3% but not at 4% and 5% where greater isometric activation is required for stabilisation. With a 2% resistive load requiring less body movement the arms can 'spin' the resistive load and not require any additional assistance from the trunk. The typical participant case study presented (Figure 4.6) indicated that angular velocity increases from about 20-s and trunk rotational velocity may be assisting in power production (EMG data indicating significantly greater activation at minimum PO for both 4% and 5% *vs* 2% resistive loads).

The distance (mm) between C7 and the static point directly vertical to the centre of ergometer bottom bracket was measured in order to determine changes in

distance between the torso and the ACE. The measurement demonstrated significance between a 2% against a 4% resistive load and may help in explaining the significantly greater angular velocity with a 3% resistive load. The distance at uncorrected PPO indicates that for 2% and 3% resistive loads participants were at their closest to the ergometer, whereas at the end of the test they have then to moved back and away from the ergometer. It therefore appears that to generate the cadence required (for the resistive load) to accelerate the flywheel and elicit their corrected PPO participants moved closer to the ergometer. At the end of the test for the 4% and 5% resistive loads (Figure 4.7) resulted in participants moving further away from the ergometer than at either corrected or uncorrected PPO. This movement may suggest why the external oblique EMG activity was significantly greater at these resistive loads when compared to the 2% loading, as moving further away from the ergometer allows the external oblique to assist in either stabilisation and/or power production at point of fatigue and lower cadence compared to faster cadences appear to increase trunk rotation during high intensity ACE (Price *et al.*, 2007). However, unlike (seated) leg ergometry ACE participants are able to increase/decrease elbow joint angle and thereby increasing or decreasing distance between their torso and ACE and therefore elbow joint angle and/or angular velocity to the ergometer. It was not clear from this study if these distances were optimal for the generation of peak cadence as the participants were untrained in ACE.

Although no significant differences were detected for elbow joint ROM across resistive loads, and between peak and minimum power output the case study presented shows that although this joint angle does not change significantly the joint position does (Figure 4.5). Although the elbow marker position has changed, the inner shoulder marker has also changed position and together this results in minimal changes in joint angle. This may explain why these results differ from other fatiguing studies where the significant results were detected for fatigue when the upper limb is more firmly constrained (Voight *et al.*, 1996, Carpenter *et al.*, 1998) and therefore if only one marker is changing position then it may reflect a greater change in ROM. Additionally, the time points used in the present study to assess changes in joint angle may not be those where changes in joint angle occur. The plot of mean joint angle shows an increase in joint angle from corrected to uncorrected power, which may be a factor in the

decrease in distance seen from corrected to uncorrected power. The decrease in mean angle at the end of a 5% resistive load remains unexplained. The figure for a typical participant (Figures 4.5 and 4.8) shows changes in the joint position and the distance variable.

The general lack of significance detected with kinematic analysis could indicate that despite change in power/cadence, kinematics do not change. Another more likely proposition based on the typical participant (Figures 4.5, 4.6 & 4.8) is that kinematic changes do not occur at the time points measured. The case study traces shown indicated that kinematic changes do occur but at the time points either before or after PPOs have occurred.

In conclusion, during an upper body WAnT, there were differences in peak power and time to peak power between corrected and uncorrected PPO, and corrected PPO was independent of resistive load. Although some of these results may have been expected from previous literature this is the first time that EMG and kinematic data has also been reported in conjunction with standard performance indices for the upper body WAnT. Data for EMG activity demonstrated the novel finding of biceps brachii activation in proportion to resistive load at PPO whereas other sites became more active towards the end of the test, possibly in aiding torso rather than lower body stabilisation. Kinematic data demonstrated changes in movement patterns although the results were not as conclusive as for the performance indices and EMG results.

The data presented also demonstrate that it is important to consider the method of power output calculation used and that kinematic and EMG responses differ between resistive loads. Use of corrected/uncorrected PPO may reflect whether the observer is interested in movement speed, or the power output *per se*.

Chapter 5

The physiological and biomechanical responses to exhaustive continuous high intensity upper body exercise

5.1 Introduction

The first study (chapter 4) examined the physiological and biomechanical responses to an all-out 30 s sprint test using the upper body. The key findings were that the electromyographic (EMG) activity of the biceps brachii muscle at corrected peak power distinguished between different resistive loads (percent of body mass). At the end of the test most muscles of the upper body and torso demonstrated greater activity when compared to the initial seconds of the test and with respect to greater resistive loads (i.e. 5% against 2% body mass). These responses were considered to relate to both the increased force requirements with increases in resistive load and muscular effort as fatigue developed throughout the test. These responses were paralleled with changes in movement patterns or exercise technique. Although the Wingate anaerobic test (WAnT) used in study one is a valid and reliable test (Bar-Or *et al.*, 1977, Bar-Or, 1987, Smith *et al.*, 2007b) and therefore the test results accurately reflected the range of movement speeds (i.e. peak cadence) with respect to the resistive load applied and level of fatigue developed during the test. Although there is a significant aerobic component toward the end of the Wingate test (Smith and Hill, 1991, Hill and Smith, 1993), fatigue is predominantly a result of anaerobic processes (Smith and Hill, 1991, Beneke *et al.*, 2002, Medbo *et al.*, 1999, Micklewright *et al.*, 2006). However, there are no reports of the physiological and biomechanical responses to fatigue during exercise at a high intensity that is more aerobic.

The standard test of aerobic fitness is a test of maximal/peak oxygen uptake (Astrand and Rodahl, 1986, Anderson, 1992, McConnell, 1988). Although this test is valid within a clinical setting (Ilias *et al.*, 2009, Martin *et al.*, 1992, Al-Rahamneh *et al.*, 2010) and can provide information regarding athletic potential and/or training status (Forbes and Chilibeck, 2007, Neville *et al.*, 2009), it has little practicality as sport or exercise settings rarely require a participant to steadily increase their work load until they volitionally cease the activity within 10-15 minutes (Smith and Price, 2007, Cooke, 1996, Hopkins *et al.*, 2001). The

majority of sport and exercise activities require the task to be completed over a given distance, which in general requires a more evenly measured power output (Lambert *et al.*, 1995, Grant *et al.*, 1997, Atkinson *et al.*, 2003). To this end a number of researchers have investigated physiological responses completed to exhaustion during exercise intensities at or around maximal/peak oxygen uptake (Billat *et al.*, 1996, Dorel *et al.*, 2009, Lepretre *et al.*, 2004, Hill and Rowell, 1996). Whilst the majority of these investigations have involved lower body exercise, a small number have investigated upper body exercise either on its own or in comparison to lower body exercise (Bressel and Heise, 2004, Bressel *et al.*, 2001, van Drongelen *et al.*, 2009, Dalsgaard *et al.*, 2004). Similar to studies examining submaximal exercise (Bressel and Heise, 2004, Bressel *et al.*, 2001, van Drongelen *et al.*, 2009, Dalsgaard *et al.*, 2004), the comparative studies have indicated that during high intensity upper body ACE the arms work under a greater physiological strain than the legs and the time to exhaustion (T_{lim}) at the same absolute work load as the legs is less (Vokac *et al.*, 1975, Eston and Brodie, 1986, Franklin, 1985). Therefore, if there are differences in physiological response during upper and lower body exercise the physiological responses to upper body exercise at high intensities may also differ from lower body responses and should be considered for further investigation.

A number of reasons for greater fatigue and reduced power output during upper body ACE have been reported such as a relatively smaller skeletal muscle mass (Sawka, 1986), a delayed VO_2 response to exercise (Pendergast, 1989, Koga *et al.*, 1996) and a lower training status (Ahlborg and Jensen-Urstad, 1991, Davis *et al.*, 1976, Koga *et al.*, 1996). Previous research examining ACE at a range of intensities (70%-90% of VO_{2peak}) have shown that there is greater and/or earlier recruitment of type II muscle resulting in a slower VO_2 fast component response and a greater VO_2 slow component compared to leg cycle ergometry (Smith *et al.*, 2006c, Koppo *et al.*, 2002, Schneider *et al.*, 2002, Bernasconi *et al.*, 2006) and may be linked to an additional contribution from the torso and lower limbs and possibly handgrip (Koppo *et al.*, 2002, Bernasconi *et al.*, 2006, Smith *et al.*, 2006c). Additionally, performance at high intensity ACE has been shown to be limited by peripheral rather than central fatigue (a full explanation is given in section 2.3) (Franklin, 1985, Sawka, 1986). Whether this upper body fatigue differs between loads at high intensities has not been comprehensively reported

in the literature and further research is required to establish if there are different physiological responses between exercise intensities.

Biomechanical analysis of incremental ACE via kinematics, indicates that the optimal cadence was 70 ($rev \cdot min^{-1}$) for movement speed and power output, and that at 50 ($rev \cdot min^{-1}$) greater force required greater range of motion (Price *et al.*, 2007); whether manipulating power by load rather than cadence has a similar relationship has not been reported. Incremental ACE studies examining muscle deoxygenation indicate that the biceps brachii had the greatest decrease in muscle oxygenation (Lusina *et al.*, 2008). With EMG studies indicating at sub-maximal loads that the biceps and triceps brachii show increased activation at greater loads (Smith *et al.*, 2008, Frauendorf *et al.*, 1989, Mossberg *et al.*, 1999). A number of such studies have made recommendations for further research regarding contributions/limitations to exercise/fatigue due to torso, back and forearm grip (Bernasconi *et al.*, 2006, Smith *et al.*, 2008, Schneider *et al.*, 2002, Koppo *et al.*, 2002, Stamford *et al.*, 1978, Shiomi *et al.*, 2000, Koga *et al.*, 1996). However, these recommendations remain unreported in the literature and along with the biceps and triceps brachii require further research to establish biomechanical responses at high intensity exercise, as this may have implications for training and testing in sport, exercise and health.

Examining the physiological and biomechanical responses to continuous upper body exercise at fixed exercise intensities and cadence would allow the examination of fatigue from a different perspective than the WAnT. In contrast to the WAnT, where cadence and peak power change during a 30 s period, such continuous exercise tests are open ended with cadence controlled so that power output is maintained, this may help reduce the effect of a pre performance pacing strategy where the participant can control the power output and energy expenditure (Baron *et al.*, 2011, Mauger *et al.*, 2010). The relationships between performance from physiological and biomechanical perspectives might subsequently differ between the WAnT and continuous work test. Therefore, the aim of this study was to examine the relationship between physiological and biomechanical variables in relation to performance during exercise at a range of exercise intensities. To enable direct comparison across exercise intensities, time points of 30 s, 120 s and the time point at exhaustion (T_{lim}) were chosen. The 30 s time point provides data about early responses to the intensities and

the 120 s time point providing data prior to T_{lim} without, hopefully, T_{lim} being achieved. The time point at exhaustion (T_{lim}) provides data at the cessation of the exercise.

5.2 Method

5.2.1 Participants

Fourteen participants (age 21.1 ± 6.1 years, mass = 74.3 ± 12.0 kg, height = 1.77 ± 0.12 m) volunteered to take part in this study. Participants had no previous arm crank ergometry experience and did not regularly participate or train in, or for, upper body sport or exercise. A minimum of 48-h separated experimental tests, which were performed within ± 1-h of the time of day of the initial test. Furthermore, participants were instructed not to conduct new training, or any vigorous training at least 48-h prior to each test. All tests were conducted in the same laboratory with the temperature between 20 ± 1°C. The University's Post Graduate Research Ethics Committee approved all experimental procedures (Appendix 1).

5.2.2 Exercise protocol

Participants completed a VO_{2peak} test to volitional exhaustion (Section 3.3.2). Prior to this test all participants undertook the same exercise protocol for familiarisation (Section 3.3.2). Therefore, all participants completed two VO_{2peak} tests prior to the continuous work tests (CWT). Each participant's peak minute power (PMP) was calculated as described in section 3.2.2.i. Subsequent to the main VO_{2peak} test, participants completed four high intensity continuous work tests to volitional exhaustion on an arm crank ergometer. The four CWTs were conducted at 80%, 90%, 100% and 110% of PMP. To avoid selection bias in testing each test was allocated using a 4 x 4 x 4 Latin square design. Oxygen uptake (VO_2), respiratory exchange ratio (RER), and heart rate (beats·min^{-1}; HR) were continuously recorded for each test (Section 3.4.2). Rating of perceived exertion, local (RPE_L; arms) and cardiorespiratory (RPE_{CR}) were recorded in the last 20 s of the first minute of exercise and the last 20 s thereafter of each incremental stage. Time to exhaustion (T_{lim}) was recorded as the performance outcome measure.

5.2.3 Electromyography

Surface EMG was recorded through active electrodes placed on the following sites: flexor carpi ulnaris (FCU); biceps brachii (BB); triceps brachii lateral (TB); anterior deltoid (AD); infraspinatus (IS); external oblique (EO). A passive reference electrode (Blue sensor M-00-S, Ambu Ltd, Cambs, UK) was placed centrally on the right patella. All sites were cleaned, prior to each test, with isopropyl alcohol to remove oil and dirt. Double-differential (16-3000Hz bandwidth, x300 gain), bipolar, active electrodes (MP-2A, Linton, Norfolk, UK) were firmly taped to the skin surface with the wires also taped down. After the first test each electrode placement was marked on the participant's skin with a surgical marker pen, the electrode was placed on the marked site for subsequent tests (section 3.4.5.i). The mean RMS EMG amplitude was calculated during the warm-up EMG over 10 duty cycles using a load corresponding to 80% of peak minute power. During each test at the time points considered 30's, 120's and T_{lim}, the mean RMS EMG data was taken over three consecutive peaks. Full details of EMG processing and synchronisation are described in section 3.4.5 and 3.6, respectively.

5.2.4 Kinematic analysis

Kinetic data were collected via infrared reflective markers attached using double-sided tape on the following anatomical landmarks; ulnar styloid process (O); lateral epicondyle of the humerous (N); most prominent superior position on the conoid tubercle on the clavical (L), and 7th cervical vertebrae (C7; Figure 5.1). The ACE was also marked with infrared reflective markers at the following static points; directly vertical to centre of bottom bracket (C), and top and centre of seat post pillar (A) (Figure 5.1). Further details of anatomical landmarks and ACE markers are given in Table 3.12. After the first test, the placement of each participant's reflective marker was marked on the participant's skin with a surgical marker pen as for the EMG measurements.

The wrist, elbow and inner shoulder was used to determine the range of movement of the elbow joint (°; ROM) and is comparable to that of the knee joint in cycling (Zehr and Chua, 2000). To determine the change in distance (mm) between the torso and the ACE the distance between C7 and the static point directly vertical to the centre of ergometer bottom bracket ($C7_D$) was recorded throughout each CWT and measured as section 3.5.

To measure velocity and change in direction of the trunk (trunk rotation) in relation to the ergometer, angular velocity ($°\cdot s^{-1}$) between C7, shoulder inner and top centre of seat post pillar was recorded throughout each CWT and measured as section 3.5. The above variables were analysed at the following times 30 s, 120 s and at volitional exhaustion (T_{lim}). Refer to Table 3.12 for a full description of each marker position. For full details of kinematic processing and synchronisation are described in section 3.5.2.

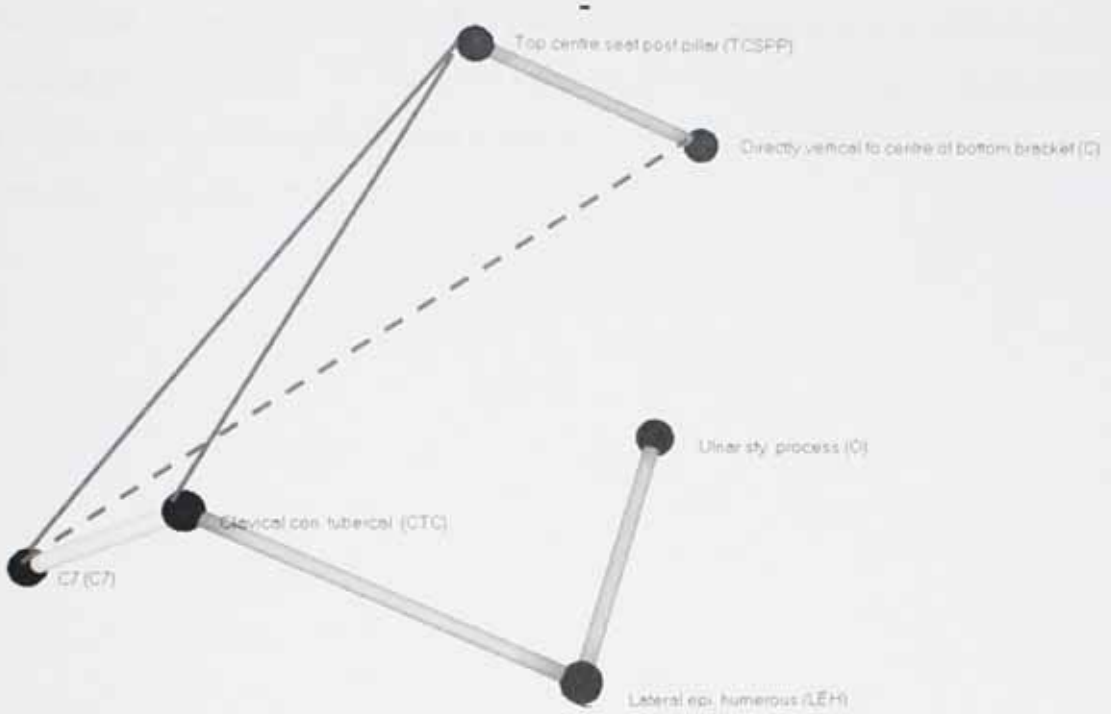


Figure 5.1. Example of kinematic analysis showing anatomical and static markers for analysis. The solid line represents the joined points of trunk rotation. The dashed line represents the joined points of the two markers for distance.

5.2.5 Statistical analysis

The data, including graphs, are presented as the mean ± standard deviation (*s*). For clarity standard deviations are plotted on line graphs for top and bottom lines as the standard deviations were fairly equal across data sets. All analyses were performed using the Statistical Package for Social Sciences (v 17.0; SPSS Inc., Chicago, IL). To assess for differences in T_{lim} between exercise intensities (80%, 90%, 100% and 110% of PMP) time to T_{lim} was analysed across all four resistive loadings using one-way analysis of variance.

Differences in oxygen uptake, RER, HR, RPE, EMG activity for each muscle and kinematic variables corresponding to 30-s, 120-s and volitional exhaustion (T_{lim})

were analysed using separate two-way analysis of variance with repeated measures on both factors (trial × time).

Where SPSS presented *P* values of $P = 0.000$ these are reported as $P < 0.001$. When there were multiple comparisons for performance indices for physiological responses (section 5.3.3), rating of perceived exertion (section 5.3.4), electromyography responses (5.3.5) and kinematic analysis (section 5.3.6) individual differences between means were located using Bonferroni post-hoc correction. To interpret the true meaningfulness of the data, test-retest time to exhaustion for the continuous work test duration (as discussed in section 3.3.3) has been included in the discussion.

5.3 Results

5.3.1 Peak physiological responses

The peak cardio-respiratory responses and performance at volitional exhaustion for the VO_{2peak} test are shown in Table 5.1.

Table 5.1 Cardio-respiratory variables for peak oxygen uptake at T_{lim}.

Variable	Mean ± SD
PMP (Watts)	141 (22)
VO_{2peak} ($l \cdot min^{-1}$)	2.44 (0.48)
Heart rate peak ($beats \cdot min^{-1}$)	179 (12)
RER	1.30 (0.08)

5.3.2 Continuous work tests duration

Significant differences were observed for T_{lim} between exercise intensities (F = 28.9, $P < 0.001$) with values decreasing with resistive load (Figure 5.2) (611 (194), 397 (99), 268 (90) 206 s (67), respectively). Post-hoc analysis revealed that T_{lim} using 80% of PMP exercise intensities was greater than for 90%, 100% and 110% PMP exercise intensities ($P < 0.001$) and 90% *vs* both 100% and 110% PMP exercise intensities ($P = 0.079$, $P = 0.001$). The coefficients of variation for each test were similar (31.9%, 24.9%, 33.6% and 32.5%, respectively). Therefore, as exercise intensity increases, T_{lim} significantly decreases suggesting decreases with higher intensities induced greater fatigue limiting the ability to continue the exercise.

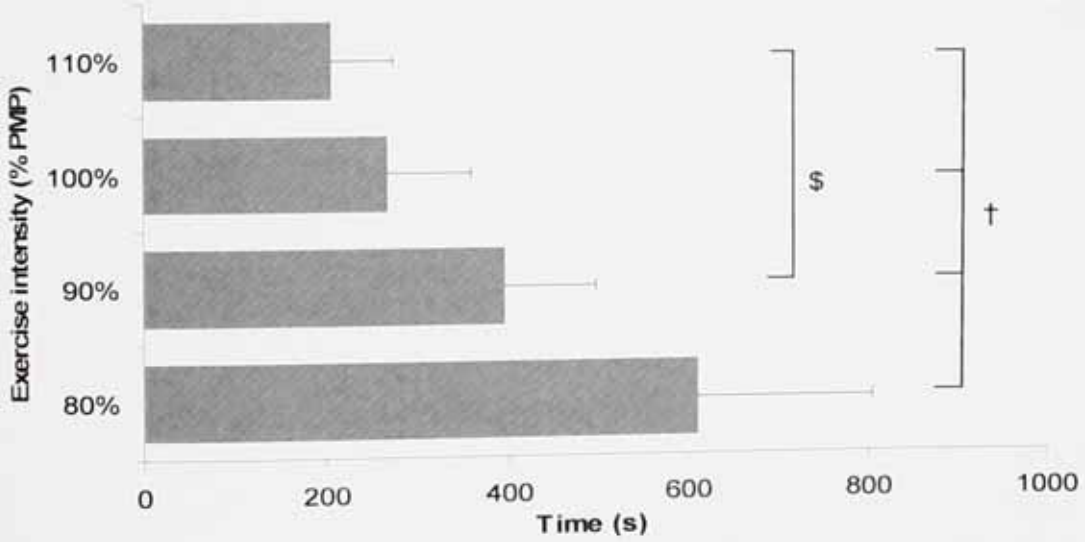


Figure 5.2. Time to exhaustion (T_{lim}) for all four percentage of PMP trials.
($) significant difference between resistive loads $P < 0.01$.
(†) significant difference between resistive loads $P < 0.001$.

5.3.3 Physiological response during the continuous work tests

5.3.3.i Oxygen uptake

There was a significant difference observed for oxygen uptake ($F = 7.524$, $P < 0.001$). Post-hoc analysis revealed that values at 120 s increased with exercise intensity (Table 5.2). Post-hoc analysis revealed that oxygen uptake using 80% of PMP was less than for both the 100% and 110% PMP at 120 s ($P = 0.001$). No differences for oxygen uptake were observed for PMP exercise intensities at 30 s and T_{lim}. There was a significant difference for oxygen uptake and peak oxygen uptake ($F = 4.832$, $P = 0.002$). Post-hoc analysis revealed that oxygen uptake using 80% of PMP was significantly different from peak oxygen uptake ($P = 0.013$).

5.3.3.ii Respiratory exchange ratio

Significant differences were observed between RERs at 120 s ($F = 11.099$, $P < 0.001$) with values increasing with exercise intensities (Table 5.2). Post-hoc analysis revealed that RER using 80% of PMP exercise intensities was less than for both, 100% and 110% PMP ($P = 0.02$, $P < 0.001$) and 90% *vs* 110% PMP ($P = 0.002$). Significant differences between exercise intensities were also observed for T_{lim} ($F = 25.286$, $P < 0.001$) with values increasing with exercise intensities. Significant difference were observed between the exercise intensities at 80% *vs* 90%, 100% and 110% of PMP ($P = 0.007$, $P < 0.001$, $P < 0.001$, respectively) and 90% *vs* both 100% and 110% ($P = 0.013$, $P < 0.001$, respectively). No differences for RER were observed for exercise intensities at 30 s. Therefore, as exercise intensity increases RER also increases suggesting that greater exercise intensities induce a greater RER.

Table 5.2 Mean oxygen consumption, respiratory exchange ratio and heart rate during the each continuous work test (mean ± *SD*).

Variable	PMP	30 s	120 s	Exhaustion
VO_2 ($l \cdot min^{-1}$)	80%	1.04 (0.33)	1.66 (0.23)[a]	2.10 (0.32)
	90%	1.14 (0.17)	1.85 (0.22)	2.29 (0.37)
	100%	1.25 (0.23)	2.07 (0.33)	2.33 (0.49)
	110%	1.28 (0.20)	2.06 (0.28)	2.26 (0.34)
RER	80%	1.07 (0.10)	1.21 (0.07)[a]	1.15 (0.07)[c]
	90%	1.05 (0.10)	1.26 (0.08)[b]	1.26 (0.07)[a]
	100%	0.98 (0.11)	1.33 (0.09)	1.36 (0.10)
	110%	1.03 (0.11)	1.38 (0.09)	1.40 (0.09)
HR ($beats \cdot min^{-1}$)	80%	119 (13)	139 (13)[a]	174 (11)
	90%	115(14)	142 (16)[b]	171 (17)
	100%	118 (11)	152 (12)	174 (11)
	110%	127 (14)	164 (9)	175 (10)

[a]. Significantly different from 100% and 110% peak minute power.

[b]. significantly different from 110% peak minute power.

[c]. Significantly different from 90%, 100% and 110% peak minute power.

5.3.3.iii Heart rate

A significant difference was observed for heart rate ($F = 10.650$, $P < 0.001$). Post–hoc analysis revealed that HR rate increased with exercise intensities at 120 s (Table 5.2). Post-hoc analysis revealed that heart rate using 80% of PMP was less than for both the 100% and 110% exercise intensities ($P = 0.055$, $P < 0.001$ respectively) and 90% *vs* 110% PMP ($P < 0.001$). No differences for HR were observed for exercise intensities at 30 s and T_{lim}. Therefore, the HR response to exercise intensity is the same at 30 s and fatigue at T_{lim}, however the results suggested that exercise intensity affects HR at 120 s with a lower HR at lower intensities which suggested a different HR response to fatigue prior to T_{lim}.

5.3.4 Rating of perceived exertion

5.3.4.i Local fatigue

A significant interaction was observed for RPE_L ($F = 7.767$, $P < 0.001$). Post–hoc analysis revealed that RPE_L increased with exercise intensities at 120 s. Post-hoc analysis revealed that RPE_L using 80% of PMP was less than for both 100% and

110% PMP ($P = 0.75$, $P < 0.001$ respectively) and 90% *vs* 110% PMP ($P = 0.010$). No differences for RPE_L were observed for exercise intensities at 30 s and T_{lim}.

5.3.4.ii Cardiorespiratory fatigue

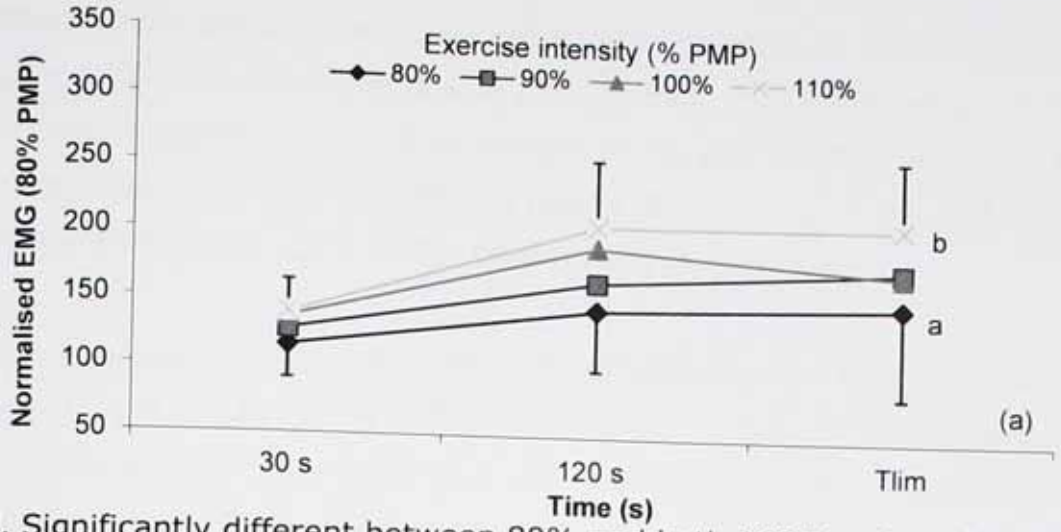
A significant interaction was observed for RPE_{CR} ($F = 5.682$, $P = 0.002$). Post-hoc analysis revealed that RPE_{CR} increased with exercise intensities at 120 s. Post-hoc analysis revealed that RPE_{CR} at 80% of PMP was less than for 110% PMP ($P = 0.002$) and 90% *vs* 110% PMP ($P = 0.025$). No differences for RPE_{CR} were observed for PMP exercise intensities at 30 s and T_{lim}. Therefore, RPE_L and RPE_{CR} reflect the different exercise intensities at 120 s but not at 30 s and T_{lim}, this suggested that greater exercise intensities induced greater RPEs at 120 s.

5.3.5 Electromyography responses

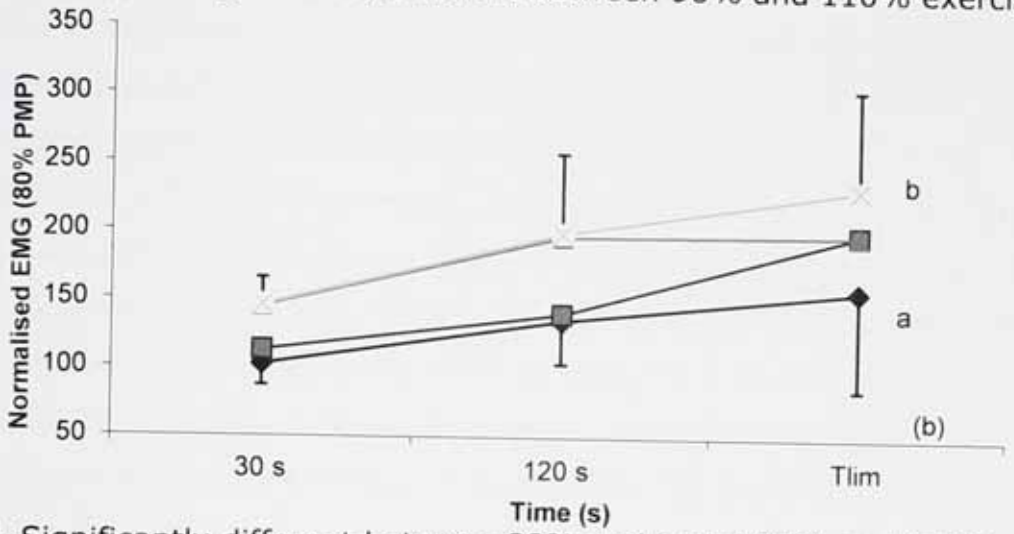
Electromyographic activation was measured for each muscle at 30 s, 120 s and T_{lim}. For biceps brachii there were differences in peak EMG activity between PMP exercise intensities ($F = 8.276$, $P < 0.001$) and time ($F = 20.808$, $P < 0.001$). Post-hoc testing for PMP exercise intensities revealed significantly less activation between 80% *vs* both 100% and 110% ($P = 0.016$, $P < 0.001$, respectively) and 90% *vs* 110% ($P = 0.027$) (Figure 5.3a). Differences in time were observed between 30 s and both 120 s and T_{lim} ($P < 0.001$).

For triceps brachii there were differences between PMP exercise intensities ($F = 10.135$, $P < 0.001$) and time ($F = 23.205$, $P < 0.001$). Post-hoc testing for PMP exercise intensities revealed significant differences between 80% and both 100% and 110% ($P = 0.001$, $P < 0.001$, respectively) and 90% *vs* 110% ($P = 0.006$) (Figure 5.3b). Differences in time were observed between time at 30 s *vs* both 120 s and T_{lim} ($P < 0.001$ all) and 120 s *vs* T_{lim} ($P = 0.019$).

There was a significant difference for flexor carpi ulnaris between PMP exercise intensities ($F = 6.099$, $P = 0.001$) and time ($F = 15.273$, $P < 0.001$), with post-hoc testing revealing a significance between PMP exercise intensities of 80% *vs* both 100% and 110% ($P = 0.004$, $P = 0.0053$, respectively), 90% *vs* 100% ($P = 0.008$) and approaching significance for 90% *vs* 110% ($P = 0.092$) (Figure 5.3c). Time was significant at 30 s *vs* both 120 s and T_{lim} ($P = 0.002$, $P < 0.001$, respectively).



[a]. Significantly different between 80% and both 100% and 110% exercise intensity. [b]. Significantly different between 90% and 110% exercise intensity



[a]. Significantly different between 80% and both 100% and 110% exercise intensity. [b]. Significantly different between 90% and 110% exercise intensity.

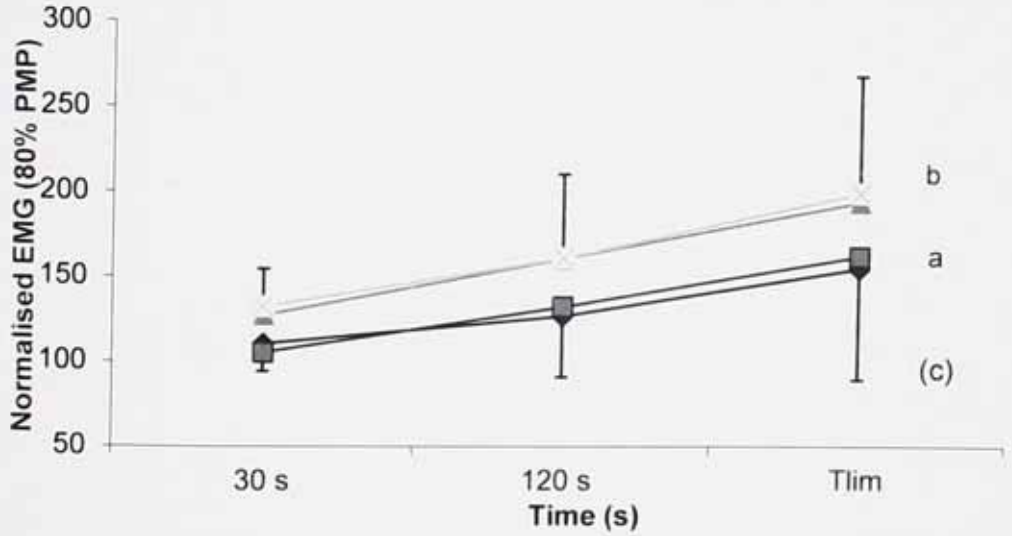
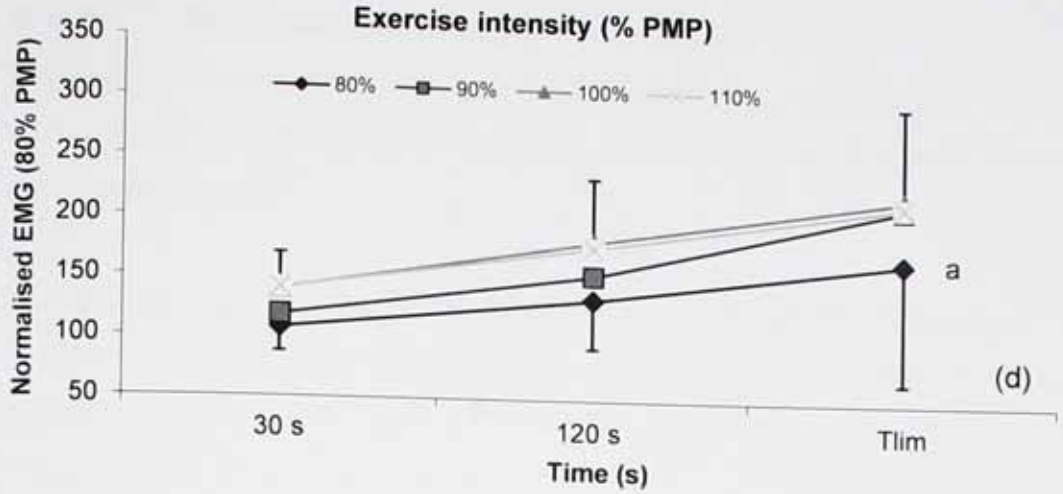


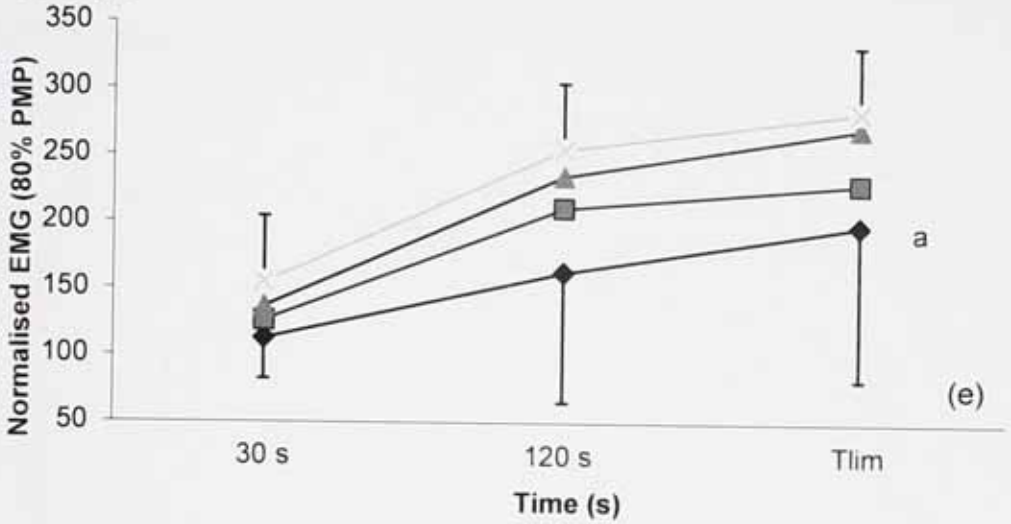
Figure 5.3. Normalised EMG (80% PMP) against exercise intensities (% PMP) and time (s). (a) Biceps brachii. (b) Triceps brachii. (c) Flexor carpi ulnaris. [a]. Significantly different between 80% and both 100% and 110% exercise intensity. [b]. Significantly different between 90% and 100% exercise intensity

The anterior deltoid was significantly different between PMP exercise intensities ($F = 4.210$, $P < 0.001$) and time ($F = 20.892$, $P < 0.001$) with post-hoc testing indicating significance at 80% *vs* both 100% and 110% ($P = 0.011$, $P = 0.022$, respectively), and time at 30 s *vs* both 120 s and T_{lim} ($P = 0.012$ and $P < 0.001$ respectively) and 120 s *vs* T_{lim} ($P = 0.002$) (Figure 5.4d).

There was a significant difference for infraspinatus for PMP exercise intensities ($F = 5.437$, $P = 0.001$) and time ($F = 24.144$, $P < 0.001$), with post-hoc testing indicating significance at 80% *vs* both 100% and 110% ($P = 0.007$, $P = 0.004$, respectively). Time was significant at 30 s *vs* both 120 s and T_{lim} ($P < 0.001$) and approaching significance at 120 s *vs* T_{lim} ($P = 0.078$) (Figure 5.4e).
The external oblique demonstrated a significant difference for PMP exercise intensities ($F = 14.871$, $P < 0.001$) and time ($F = 20.508$, $P < 0.001$). Post-hoc testing indicated significance at 80% *vs* 90%, 100% and 110% ($P = 0.005$, $P = 0.017$, $P < 0.001$ respectively), 90% *vs* 110% ($P = 0.009$) and 100% *vs* 110% ($P = 0.002$). Time was also was also significant at 30 s *vs* both 120 s and T_{lim} ($P < 0.001$) (Figure 5.4f). Therefore, regardless of the exercise intensity as the exercise duration increased EMG activation also increases suggesting that duration increases fatigue. In addition, differences observed at 80% and 90% against 100% and 110% exercise intensity demonstrated that greater exercise intensities induce greater EMG activity.



[a]. Significantly different between 80% and both 100% and 110% exercise intensity.



[a]. Significantly different between 80% and both 100% and 110% exercise intensity.

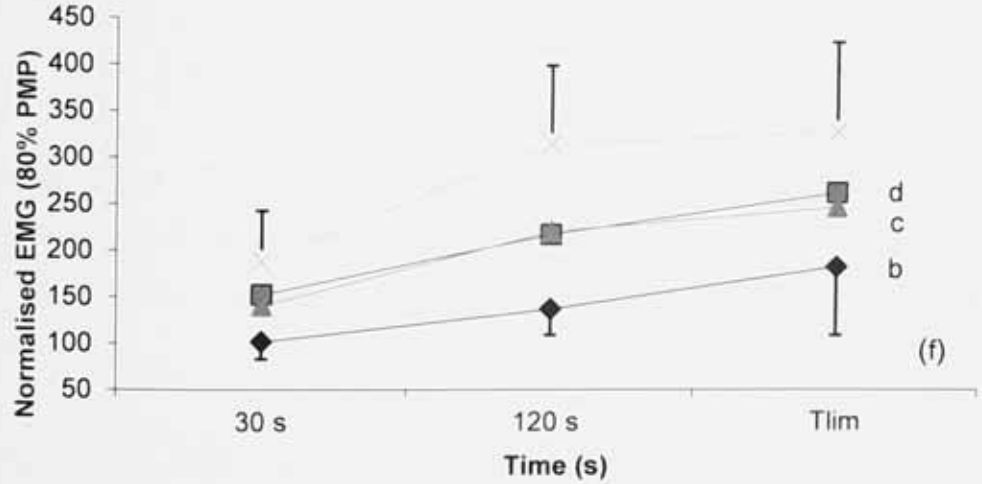


Figure 5.4. Normalised EMG (80% PMP) against exercise intensities (% PMP) and time (s). (d) Anterior deltoid. (e) Infraspinatus. (f) External oblique. [b]. Significantly different between 80% and both 90%, 100% and 110% exercise intensity. [c]. Significantly different between 90% and 110% exercise intensity. [d]. Significantly different between 100% and 110% exercise intensity.

5.3.6 Kinematic analysis

No interactions between time and trial were observed for any of the kinematic variables measured. Main effects for both time and trial were observed for time and resistive load.

There were significant differences for elbow joint ROM (°) between time (F = 6.149, P = 0.003) with values decreasing over time. Post-hoc testing indicating significance at 30 s *vs* both 120 s and T_{lim} (P = 0.032, P = 0.003, respectively) (Figure 5.5). There were no significant differences for elbow ROM (°) between PMP exercise intensities. Therefore, the results suggest that exercise duration affects elbow ROM at 120 s. However, at T_{lim} elbow ROM is similarity affects by fatigue regardless of the duration or intensity.

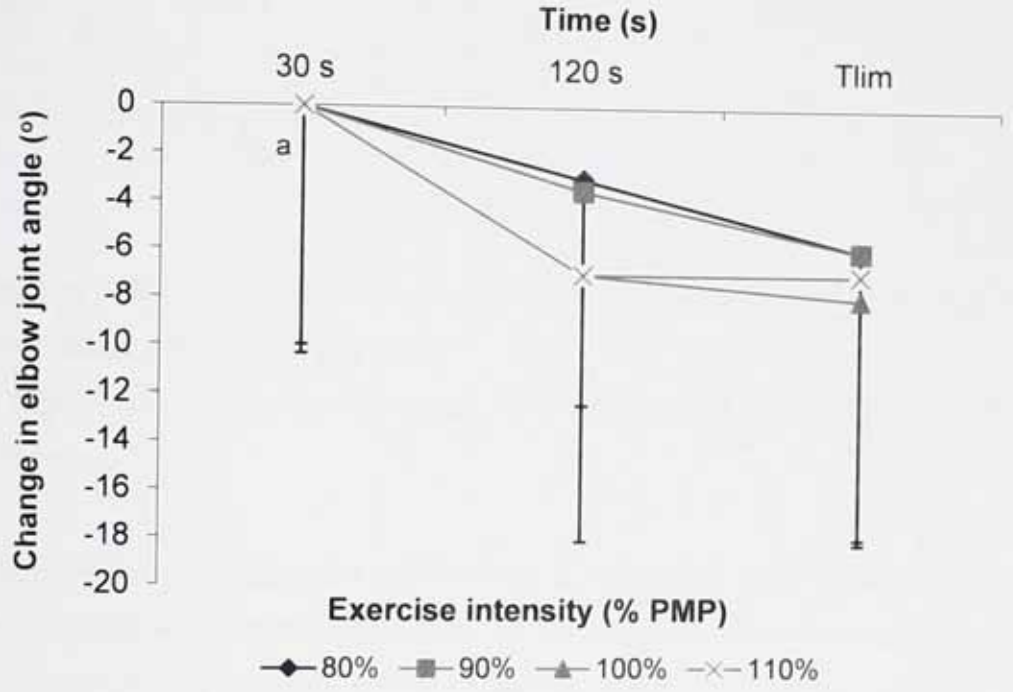


Figure 5.5. Changes in elbow joint angle (°) at all four exercise intensities (% PMP) from 30 s.

[a]. Significantly different between 30 s and both 120 s and T_{lim} 110% exercise intensity.

There was a significant difference observed for trunk rotation measured as angular velocity ($°\cdot s^{-1}$) between PMP exercise intensities (F = 5.217, P < 0.001). Post-hoc testing revealed significance was approached between 80% *vs* 90%, 100% and 110% PMP exercise intensities (P = 0.054, P = 0.055, P = 0.010, respectively). Time was significant between 30 s *vs* both 120 s and T_{lim} (P < 0.001 all) and approaching significance for 120 s *vs* T_{lim} (P = 0.070) with trunk

rotational velocity increasing over time (Figure 5.6). Therefore, the results suggested that the exercise intensity affects trunk rotational velocity.

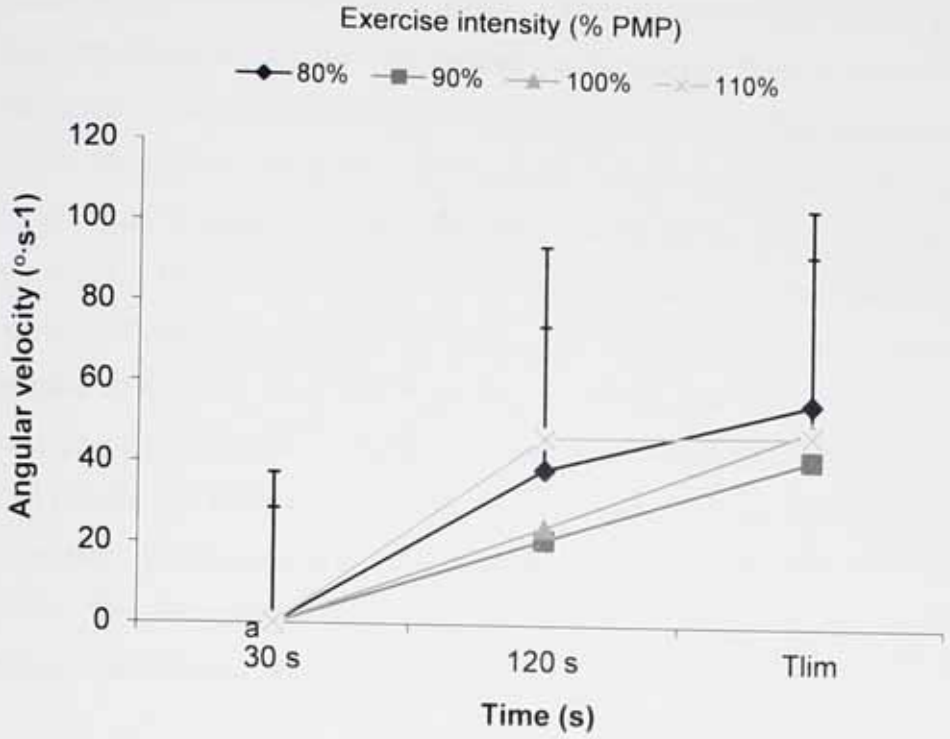


Figure 5.6. Changes in trunk rotational velocity ($^{\circ}\cdot s^{-1}$) at all four exercise intensities (% PMP) from 30 s.

[a]. Significantly different between 30 s and both 120 s and T_{lim} 110% exercise intensity.

There were no significant differences in distance (C7) relative to the ACE between PMP exercise intensities or for comparisons over time, although there were variations across all four resistive loads.

5.4 Discussion

The primary aim of this study was to examine the interaction between physiological and biomechanical parameters during continuous high intensity upper body exercise to exhaustion. The study aimed to add to the findings from the first study by exploring similar parameters (EMG and kinematics) along with the addition of respiratory measure to provide further analysis of ACE over a longer duration. As participants were required to maintain a constant cadence of 70 rev·min^{-1}, power output (W) was manipulated by load which was kept constant. Therefore, unlike the Wingate anaerobic test participants are only in control of the duration of the exercise. The main findings were that oxygen uptake and heart rate were similar at exhaustion whereas the respiratory exchange ratio data suggested different contributions of anaerobic metabolism and therefore different underlying physiological responses. The EMG activity increased over time and was greater for the 100% and 110% VO_{2peak} intensity trials. The kinematic data suggested that trunk rotation velocity rather than trunk stabilisation occurred throughout each exercise test.

5.4.1 Peak oxygen uptake tests and peak heart rates

Values of peak oxygen uptake (l·min^{-1}) were similar to the range reported in the literature for non-active or recreationally active participants (1.58-2.89 l·min^{-1}) (Davis *et al.*, 1976; Jensen-Urstad *et al.*, 1993; Kang *et al.*, 2004; Magel *et al.*, 1978; Sawka *et al.*, 1983; Washburn and Seals, 1984; Yasuda *et al.*, 2006) (Ahlborg and Jensen-Urstad, 1991; Kang *et al.*, 1997; Koga *et al.*, 1996; Lusina *et al.*, 2008; Swensen *et al.*, 1993; Warren *et al.*, 1990). However, values were lower than for peak oxygen uptake for physically active or trained participants (2.92-3.36 l·min^{-1}) (Jensen-Urstad, 1992; Price *et al.*, 2007; Smith *et al.*, 2006; Warren *et al.*, 1990). Peak heart rates were within the median range reported in the literature HR 166-184 (Castro *et al.*, 2010; Davis *et al.*, 1976; Jensen-Urstad *et al.*, 1993; Kang *et al.*, 1997; Price and Campbell, 1997; Smith *et al.*, 2001). Interestingly it appears that HR_{max} for the peak oxygen uptake test equals 200 – age. It is possible that a criteria maker of the achievement of peak oxygen uptake, in untrained participants, during incremental ACE to volitional exhaustion is 200 (beats·min^{-1}) minus age. The mean HR reported at peak oxygen uptake for ACE in Table 2.1 is 180 (6) which also indicated that 200 – age is appropriate, although for lower limb ergometry the mean values reported are 187 (4).

5.4.2 Continuous work tests duration

The T_{lim} at 100% PMP is within the values found for kayakers (239-289 s; Billat *et al.*, 1996, Leveque *et al.*, 2002) and swimmers (243-286 s; Billat *et al.*, 1996, Fernandes *et al.*, 2008a) at 100% of PMP. The T_{lim} at 110% PMP was greater than values found for arm cranking at this intensity (114 (SD 12); (Weissland *et al.*, 1999), however participant were not fully rested prior to this test. There was a decreased in T_{lim} across exercise intensities with particular differences between 80% and 90% and both 100% and 110% VO_{2peak} intensities. The T_{lim} for the different intensities are all much greater than the daily biological variation of 12 s for 110% PMP and 41 s for 80% PMP (section 3.3.3) and therefore are a good indication of performance duration. There was a clear response of duration and intensity as to be expected in lower body exercise (Morton and Hodgson, 1996, Hill *et al.*, 2002). In addition, the selection of the load (kg) applied needs to be accurate as the difference between each load is only 14 W which could easy affect the duration of the exercise. Despite the significant difference in T_{lim} across loads, from 611.2 ± 194.6 s for 80% to 206.4 ± 66.6 s for 110% PMP, oxygen uptake across all four PMP exercise intensities was not significant.

5.4.3 Physiological response during the continuous work tests

5.4.3.i Oxygen uptake

Although there was no significant difference between oxygen uptake at PMP when compared to VO_{2peak}, the significant difference between 80% PMP and VO_{2peak} (90% of VO_{2peak}) indicates that this exercise intensity is not sufficient to achieve VO_{2peak} before fatigue results in the cessation of exercise and likewise the 110% PMP intensity (94% of VO_{2peak}) was too intense to achieve VO_{2peak}. Additionally, test-retest reliability data from section 3.3.2.iii indicated that oxygen uptake at 80% and 110% PMP was lower than the expected variation (0.07 $l \cdot min^{-1}$) between tests. Further analysis of percentage of PMP compared to VO_{2peak} indicates that 90% and 100% PMP (both 97% of VO_{2peak}) was within the 3% variance in VO_2 to indicate that VO_{2peak} was achieved (Bird and Davison, 1997). The exercise intensities used were within the severe exercise domain seen in leg cycling (Caputo and Denadai, 2008) and for 110% PMP it is likely that the intensity resulted in cessation of exercise (T_{lim}; 206 s) before there could be a sufficient increase in VO_2 and may be too short for the slow component of VO_2 to have a maximal effect (Hill and Rowell, 1996, Gastin, 2001, Xu and Rhodes, 1999). The 80% PMP intensity may be sufficient for the attainment of VO_{2peak}

but before this can be achieved fatigue factors such as perceptions of fatigue (Kang *et al.*, 1998, Taylor *et al.*, 2000) stop the exercise. Research indicates that 92% PMP below 100% takes longer to achieve VO_{2peak} than exercise at 100% PMP (491 against 299 s) (Hill *et al.*, 1997). These results suggest that the optimal exercise intensity for continuous high intensity exercise to volitional exhaustion was between 90% and 100% of peak minute power.

The significant difference detected at 80% PMP compared with 100% and 110% PMP at 120 s is also in agreement with previous research indicating that phase 2 VO_2 kinetics occur at around 120 s (Jensen-Urstad *et al.*, 1993; Koga *et al.*, 1996). This is the first time that this has been reported for a continuous high intensity exercise study i.e. that phase 2 VO_2 kinetics occurred at the time point of 120 s. Although 120 s still indicated that this was the point of phase 2 oxygen kinetics, additionally this may represent faster and slower kinetics for each exercise intensity as the exponential increase is meant to be the same in each person regardless of the intensity. The kinetic response therefore is dependent on exercise intensity. However, lower intensities simply take longer to get to VO_{2peak} via the slow component.

5.4.3.ii Respiratory exchange ratio

The RER values at VO_{2peak} were similar to those previously reported at 1.31-1.35 (Castro *et al.*, 2010) although they were slightly higher than reported by other authors (1.12-1.23) who have examined prolonged aerobic exercise (Kang *et al.*, 1997; Price and Campbell, 1997; Price *et al.*, 2007; Smith *et al.*, 2001; Washburn and Seals, 1983; Yasuda *et al.*, 2006). Values of RER differed significantly between trials at 120 s and T_{lim} between lower exercise intensities (80% and 90% PMP) and higher exercise intensities (100% and 110% PMP). The higher RER values would indicate that participants were working more anaerobically and utilising a greater proportion of carbohydrate metabolism (Jensen-Urstad *et al.*, 1993; Jensen-Urstad, 1992) than at lower exercise intensities. The RER can reflect changes in muscle pH and greater bicarbonate buffering of lactate acid (Casaburi *et al.*, 1992), and the greater recruitment of type II muscle fibres (Schneider *et al.*, 2002) which would also be consistent with a greater reliance on carbohydrate metabolism (Ahlborg and Jensen-Urstad, 1991). During the 80% trial there was a decrease in RER from 120 s to T_{lim} (1.21 to 1.15 respectively). The reduction in RER could be linked to muscle

reoxygenation (following deoxygenation) seen during the later stage ACE exercise of 15 min total duration (Jensen-Urstad *et al.*, 1995) although the mechanism for this change remains unexplained (Bhambhani, 2004). Whatever the mechanism the final RER value was still indicative of anaerobic metabolism. Therefore, although at T_{lim} all the exercise intensities, indicated by the RER, have an increased activation of the anaerobic metabolism, the greater exercise intensities (100% and 110% PMP) appear to have a greater anaerobic component. This could be due to the greater resistance which results in an increased activation of type II fibres types (Koppo *et al.*, 2002, Bernasconi *et al.*, 2006), which would work more anaerobically than type I fibres and may be less efficient (Coyle *et al.*, 1992). The type II anaerobic glycolysis could utilise more carbohydrate through greater use of muscle glycogen resulting in a higher lactate acid release (Ahlborg and Jensen-Urstad, 1991) and the buffering of the by-product (CO_2) results in an increased RER and as oxygen uptake was relatively similar at T_{lim} it may be that the anaerobic metabolism was limiting the exercise duration and not oxygen uptake.

5.4.4 Electromyography responses

The measurements of EMG activation indicated that there were significant increases in EMG amplitude for all muscles from 30 s to both 120 s and volitional exhaustion. The load corresponding to 110% PMP distinguished between the other loads, with the greatest amount of activation for all sites. The EMG values increased over time for all exercise intensities, given that power output was constant, then the greater muscular activity/effort for the same workload is likely the result of fatigue (Kamen and Gabriel, 2010). This is in contrast to the EMG response found in study one where the EMG values increased with a decrease in power output. Therefore, changes in movement patterns might change activity to maintain power, with changes in joint angle additionally affecting EMG activity (Kamen and Gabriel, 2010).

A constant increase in EMG activity for the biceps brachii, triceps brachii, anterior deltoid and infraspinatus during constant but high intensity exercise (40% between ventilatory threshold and VO_{2peak}) has previously been shown to increase with duration of the exercise (Bernasconi *et al.*, 2006). This response suggested that an increase in muscle fibre recruitment is required to maintain power output. Previously unreported is the finding that this recruitment is

dependent on load, and that activation at 100% and 110% of PMP was significantly greater than activation at 80% and 90% of PMP. This increase in EMG activation was seen in the significant increases in RER at 120 s and T_{lim} for exercise intensities at 100% and 110%. Whether this recruitment pattern changes with training or is typical for all types of participants needs further investigation (Bernasconi *et al.*, 2006). Data from study one were indicative that for the Wingate anaerobic test (WAnT) the bicep brachii muscle is an important contributor to power out reflect an increase in EMG activating as resistive load (% BM) increased while other muscles indicated limited increases in activity across resistive loads. For the exercise intensities examined in the present study the biceps and triceps brachii showed an equal amount of activation. These results supported previous research at a variety of exercise intensities: 50 W and 100 W (Smith *et al.*, 2008) and 30 W, 60 W, 90 W and maximal exercise (Hopman *et al.*, 1995). Therefore, the current results add to previous work in that this pattern of activation is similar across a range of submaximal to maximal exercise intensities which have not been previously reported.

Flexor carpi ulnaris demonstrated significant differences in activation between exercise intensities of 80% and 90% when compared to 100% and 110% of PMP, with no significance between 100% and 110% of PMP this could indicate that the limitation of forearm muscle activity and grip endurance has been reached. Previous research has suggested flexor carpi ulnaris muscle activation increased with load at submaximal intensities (Frauendorf *et al.*, 1986) and that handgrip was an important component during heavy-intensity ACE (Smith *et al.*, 2006c). It has been suggested that increased forearm grip contributes to fatigue during ACE by reducing skeletal muscle pump activity and venous return (Koga *et al.*, 1996, Sawka, 1986) although it does not appear to affect performance during incremental ACE to exhaustion (Hooker and Wells, 1991). The RPE_L values being higher than RPE_{CR} suggest that exercise duration is limited by peripheral rather than cardiorespiratory fatigue, with EMG analysis substantiating anecdotal evidence from a number of participants that fatigue of forearms was one of the reasons for stopping the exercise. A number of studies have suggested that local rather than peripheral fatigue limits ACE performance (Sawka, 1986, Franklin, 1985) and gripping during ACE may increase perceptions of fatigue (Hooker and Wells, 1991).

One of the key findings was that the external oblique muscle had significant increases in activation across all loads. Whether the increase in activation was linked to the reduction in efficiency associated with using the trunk muscles at higher loads (Shiomi *et al.*, 2000) is not clear. The greater power output required for the higher loads could result in a greater trunk rotational velocity and activation of the trunk muscle which has been associated with power production required with a low cadence (50 rev·min^{-1}) (Price *et al.*, 2007). Study one results also indicated that the external oblique contribute to power production and also may fatigue at the end of the exercise, although the level of activation was not the same across all loads as found in this study. Additionally, increased torso activation may affect breathing frequency, with incremental ACE studies suggesting that breathing frequency increases with load (Eston and Brodie, 1986) and is greater for 90 compared to 50 rev·min^{-1} for the same load (Price *et al.*, 2007); whether there are changes in breathing frequency from a constant cadence but variable load and its possible effect on RER would require further investigation.

5.4.5 Kinematic analysis

Elbow ROM decreased across time for all trials suggesting that time rather than exercise intensity has an effect on elbow ROM. At T_{lim} there were no differences in elbow ROM which indicates that fatigue, regardless of intensity, is the main factor in changes in elbow joint angle. This is the opposite of study 1 where elbow range of motion for the three greatest loads was greater at the end of exercise than the start and that elbow ROM may increase, decrease and increase again at the end of exercise (Figure 4.4). The reduction in elbow joint angle might still be within the limit found for maximal power production for the bicep brachii as isometric studies indicate that the joint angle was within the limit of maximal power production (Doheny *et al.*, 2008). Interestingly this study also showed that the elbow joint angle is outside the ideal ROM for maximal power production for triceps brachii, and it may be that similar to study one as T_{lim} approaches the biceps brachii contributed more to power production. Changes in joint angle (Figure 5.7) occurred at 120 s, with a distinction between the two lower (-4° both) and higher loads (-7° both). At 120 s the two higher loads were 50% of T_{lim}, and might indicate that the changes in elbow ROM of motion occur at a time percentage of T_{lim} between 50% and 30% (the T_{lim} percentage for 120 s for the 90% PMP). This was the first study to examine changes in elbow ROM

across a variety of high intensity exercise loads, with previous studies only examining fixed joint angles (participants were restricted in their body movement) (van Drongelen *et al.*, 2009, Miller *et al.*, 2004). This showed that a fixed elbow joint angle may not be optimal for power production through the full duration of the exercise i.e. that fatigue changes joint angle. Therefore, for upper body ACE testing participants could improve performance by changing their elbow ROM during the exercise rather than adopting a 'fixed' position which would be more like leg ergometry. Whether elbow ROM differs between trained and untrained participants is unreported.

Similar to study one there were no significant differences detected for torso distance $C7_D$ relative to the ACE. This may be due to a prior familiarisation session and being correctly positioned relative to the ergometer as indicated by the literature (Sawka *et al.*, 1983, Sawka, 1986, Washburn and Seals, 1984, Miller *et al.*, 2004). A further consideration is that changes in $C7_D$ had a significant effect on another parameter in the kinematic chain, such as angular velocity (discussed below). The angular velocity ($°\cdot s^{-1}$) measured as trunk rotation between C7, shoulder inner and top and centre of seat post pillar was significantly lower between 80% and 90%, 100% and 110% of PMP (114 *vs* 137, 137, 143 $°\cdot sec^{-1}$, respectively). The 80% PMP exercise intensity may be a threshold between this and the higher exercise intensities. The greater loads representing greater trunk rotational velocity to compensate for fatigue and therefore activating the external oblique as seen in EMG analysis (section 5.4.3). This is a novel finding as the general research consensus is that torso stabilisation contributes to the VO_2 excess found during high intensity ACE and not trunk rotational velocity (Casaburi *et al.*, 1992; Franklin, 1985; Miles *et al.*, 1989; Stenberg *et al.*, 1967; Vokac *et al.*, 1975). Overall, in order to maintain the required power as the time to T_{lim} approaches, elbow joint angle is reduced by increasing trunk rotational velocity and torso distance from the ACE. Therefore, trunk rotational velocity may be an important component in extending exercise duration in ACE at the intensities studied.

In conclusion, this study found a number of novel findings not previously reported in the literature. The cardiorespiratory measures indicate that oxygen uptake at exhaustion was the same/similar regardless of the exercise intensity. However, test-retest data considering biological variation indicated that 90% and

100% PMP were optimal for oxygen uptake. However significant differences in RER demonstrated that there were changes in metabolic responses, probably linked to power output requirements which effects local muscle recruitment and metabolism, indicating that as power output increases there is a greater reliance on anaerobic metabolism. The EMG responses showed that the biceps and triceps brachii provide a similar but increasing level of activation with increases in load, unlike during the WAnT where the biceps brachii was an important muscle in power production. Flexor carpi ulnaris distinguished between the two lower and two upper exercise intensities, and could represent changes in grip required for the high loads. The role of the external oblique coupled with changes in angular velocity indicated that they contribute to trunk rotation, rather than stabilisation that previous studies have suggested. Kinematic data has, as described, above aided in movement and EMG analysis, especially for the trunk.

All participants reached their functional cardio respiratory maximum (VO_2 and HR). Prior to this, maximum changes in movement pattern and EMG activation occurred. The results suggested that participants were changing their body movement to maintain power output and after a certain time point no further body movement can be made or muscle recruitment achieved (e.g. increase elbow ROM, external oblique activation). The changes in body movement may by driven by the increase in RPE_L which in untrained participants is limiting exercise capacity compared to RPE_{CR} (RPE_L is greater than RPE_{CR}). A further study to analyse these fatigue parameters in trained participants would help to answer a number of outstanding questions.

Chapter 6

The effects of a 6-week arm crank training programme on physiological and biomechanical responses to high intensity upper body exercise

6.1 Introduction

A number of studies have described the physiological attributes of elite athletes performing upper body sports (Mygind, 1995, Kounalakis *et al.*, 2008, Lutoslawska *et al.*, 2003, Aziz *et al.*, 2002) or sports with a significant upper body component (Neville *et al.*, 2009). These reports have identified a number of factors consistent with a higher level of performance, such as a high peak/maximal oxygen uptake (Neville *et al.*, 2009), peak anaerobic power output (Horswill *et al.*, 1992, Neville *et al.*, 2009, Kounalakis *et al.*, 2008) and lactate threshold (Holmberg *et al.*, 2007, Jemini *et al.*, 2006, Volianitis *et al.*, 2004a). In addition, studies examining lower body based sports performance have indicated differences in kinematic responses and EMG recruitment patterns (Chapman *et al.*, 2007, Chapman *et al.*, 2008) in elite athletes when compared to less well trained participants (Stoggl and Muller, 2009, Sandbakk *et al.*, 2010). As these studies have examined populations that were already well trained it is not clear whether it is the training or the individuals 'inherent' ability to perform at a higher level that is of key importance (Timmons *et al.*, 2005).

Few studies have sought to examine the relationship between physiological and biomechanical responses during upper body exercise. Lower body exercise is generally reported to be limited centrally by maximal cardiac output (Savard *et al.*, 1987, Warren *et al.*, 1990). In contrast upper body exercise by more local factors such as regional muscle blood flow (Sawka, 1986), greater use of fast twitch muscle fibres (Sawka, 1986, Ahlborg and Jensen-Urstad, 1991), greater isometric component (Stenberg *et al.*, 1967, Marais *et al.*, 2002a), lower work efficiency (Marais *et al.*, 2002a, Eston and Brodie, 1986, Blasio *et al.*, 2009) and a compromised respiratory response (Ramonatxo, 1996, Martin *et al.*, 1991, Romagnoli *et al.*, 2006) differences in the relationship between physiology and biomechanics might be expected. Despite a number of recommendations from previous studies (Bernasconi *et al.*, 2006, Smith *et al.*, 2008, Yasuda *et al.*, 2002), the effects of upper body exercise (arm crank) training on both

physiological and biomechanical responses to high intensity upper body exercise have not been reported.

The first two studies of this thesis examined the physiological and biomechanical responses to upper body exercise across a range of exercise intensities (i.e. Wingate tests at a variety of resistive loads and constant load exercise to volitional exhaustion in the severe exercise domain (80%-110% VO_{2peak}). The main findings were that during the Wingate anaerobic test (WAnT) the electromyographic (EMG) activity at peak power output and at the end of the test increased with load. Most of the muscles demonstrated greater EMG activity at the end of the test which could be linked to the fatigue observed during each test (Hautier *et al.*, 2000). Additionally, for the performance trials in study 2 (Chapter 5) EMG activity was greatest for the 100% and 110% PMP exercise intensity tests across all time points. The EMG responses were also reflected in alterations in kinematic responses suggesting that trunk rotational velocity increased with fatigue and was not activated to aid stabilisation. Therefore, if training can offset fatigue and improve performance, as would be expected, such improvements may also be reflected in biomechanical responses.

Arm crank ergometry training has shown increased peak oxygen uptake suggesting that performance increases were related to both local and central adaptations (Loftin *et al.*, 1988, Magel *et al.*, 1978, Clausen *et al.*, 1973, Tordi *et al.*, 2001) or specific local adaptations (Stamford *et al.*, 1978, Magel *et al.*, 1978, Bhambhani *et al.*, 1991). Additionally, 4-weeks (12 sessions) of upper body weight training also increased peak oxygen uptake (Swensen *et al.*, 1993) with the authors suggesting that the mechanism, other than improvements in muscle strength, was important but not clear. However, these studies did not examine whether biomechanical responses were related to improvements in performance. Sports that require a high level of upper body involvement such as handball have suggested that training may change maximal angular velocity as measured by internal shoulder rotation during a throwing action (Roland van den and Mario, 2011). Furthermore, changes in kinematics and EMG (biceps and triceps brachii) have been linked to improvements in an elbow flexion task (Gabriel, 2002). For studies examining the lower body, elite cyclists showed a more consistent pattern of muscle recruitment and a smaller variation in kinematics which accounted for a higher level of performance in comparison to novice cyclists

(Chapman *et al.*, 2009). Additionally, coactivation of the antagonistic muscles has been shown to be reduced following training (Carolan and Cafarelli, 1992, Aagaard, 2003, Duchateau *et al.*, 2006) resulting in improved performance. However, a number of studies involving runners have indicated that despite improvements in running performance there were no changes in kinematics after training (Collins *et al.*, 2000, Lake and Cavanagh, 1996). Despite evidence for improvements in upper body exercise performance through biomechanical changes, these have not been specifically examined during high intensity ACE.

Investigating the physiological and biomechanical responses to a Wingate test and a continuous work test to exhaustion before and after training would provide a unique investigation as to how the relationship between physiology and biomechanics may change in a previously untrained population. Therefore, the aim of this study was to examine the changes in physiological and biomechanical variables in relation to performance and fatigue after a 6-week upper body exercise-training programme.

6.2 Method

6.2.1 Participants

Twelve participants with no previous arm crank ergometry experience volunteered to take part in this study (age = 20.7 ± 4.1 years, mass = 72.0 ± 11.9 kg, height = 1.80 ± 0.07 m). Participants did not regularly participate or train for upper body sport or exercise. Each participant undertook three preliminary performance tests (30-s Wingate, VO_{2peak} and T_{lim}) prior to undertaking a 6 week arm crank training programme. The performance tests were then repeated. A minimum of 48-h separated each experimental test, with participants reporting to the laboratory for testing within one hour of the initial test. Participants were instructed not to conduct new training, or any vigorous training prior to each test. All training and tests were conducted in the same laboratory with the temperature between 20 ± 1°C. All experimental procedures were approved by the University's Post Graduate Research Ethics Committee (Appendix 1).

6.2.2 Anthropometry and body composition

Each participant's body mass (kg), left and right hand grip strength (kg·N), girth (right upper arm flexed and tensed and forearm relaxed; mm); skinfold (right

arm biceps and triceps brachii; mm) were measured prior to the beginning of the training programme and after completing the training programme.

6.2.3 Exercise protocol

6.2.3.i Wingate test

After a full familiarisation session for the WAnT and the VO_{2peak} test participants completed a seated upper body WAnT, as outlined in section 3.3.1 using Cranlea Wingate software (v.4.00; Cranlea & Company, Birmingham, UK). A resistive load of 4% body mass was used as in study 1 (Chapter 4) and as used by previous researchers (Aschenbach *et al.*, 2000, Biggerstaff *et al.*, 1997, Hubner-Wozniak *et al.*, 2004, Weber *et al.*, 2006). Body mass recorded at the familiarisation session was used as the subsequent reference mass for all WAnT's. Peak power output (PPO; over 1 s duration) corrected and uncorrected, mean power output (MPO; over 29 s duration) and minimum power output (PO_{min}), peak, mean and final cadence ($rev \cdot min^{-1}$) were recorded. Time to peak power output (PPO_{time}; 1 s) for corrected and uncorrected peak power were also recorded.

6.2.3.ii Peak oxygen uptake and peak minute power test

After completing the WAnT familiarisation test, participants completed a VO_{2peak} test to volitional exhaustion (Section 3.2.2) with each participant's peak minute power (PMP) calculated (Section 3.2.2.i). A T_{lim} continuous work test (CWT) at 100% of PMP exercise intensity ($Pre_{100\%}$) was the undertaken. During each test oxygen uptake (VO_2), respiratory exchange ratio (RER), and heart rate ($beats \cdot min^{-1}$; HR) were continuously recorded as described in section 3.4.2. In the last 20 s of the first minute of exercise, the last 20 s of each subsequent 2 min and at exercise cessation a ratings of perceived exertion, local (RPE_L; arms) and cardiorespiratory (RPE_{CR}) were recorded. Time to exhaustion (T_{lim}) was recorded as the performance outcome measure.

6.2.3.iii Training programme

After completing the pre-training tests each participant completed the same number of training sessions (18 in total) over 6 weeks. Participants were required to complete a minimum of three and a maximum of four training sessions each week (Table 6.1) with no constraint on the time of day for training and were free to continue with, but not increase, any regular training during the

training programme. A training diary was completed for each participant during the period of study. The 50% and 100% PMP exercise intensity sessions undertaken were specifically prescribed to each participant based on their preliminary tests. Each week participants completed three training sessions including; repeated 10 s sprints, exercise at 100% PMP and a submaximal aerobic sessions for 30 minutes at 50% PMP.

6.2.4.iv Repeated sprint sessions

The sprint-training resistive load was 4% body mass for each participant with all the 10 s sprints completed with maximal effort. The recovery load between repetitions was 30 W for 1 min. Prior to the start of each sprint session a warm-up was conducted at 60 rev·min^{-1} (30 W) for 3 min. The number of 10-s sprints increased every other week, starting at six, then eight and finally ten sprints in the last two week of training (Table 6.1).

6.2.4.v Exercise at 100% PMP session

The 100% PMP exercise sessions were completed for a duration equal to 50% of the duration of the CWT achieved in the preliminary tests (section 3.3.3). The recovery load between repetitions was 35 W for twice the duration of the interval. Prior to the start of the 100% PMP session a warm-up was conducted at 70 rev·min^{-1} (35 W) for 3 min. The number of 100% PMP tests increased every other week, starting at three, then four and finally five in the final two weeks of training (Table 6.1).

6.2.4.vi Submaximal aerobic exercise sessions

The aerobic training was completed at 50% PMP for the first 3-weeks and then 60% of PMP exercise intensity for the remaining 3-weeks (Table 6.1). The duration of all sessions was 30 min. Each aerobic session was completed at a cadence of 70 rev·min^{-1}. Heart rate was continuously recorded during each session using a telemetric chest strap and watch (Polar, Electro Oy, Finland).

Table 6.1 Development of the training programme over the 6-week training programme.

Type of training	Number of repetitions and sessions (session numbers are bracketed)			Total sessions
	Weeks 1-2	Weeks 3-4	Weeks 5-6	
Sprint	(2) 6 x 10 s	(2) 8 x 10 s	(2) 10 x 10 s	6
PMP 100%	(2) 3 x 100% PMP	(2) 4 x 100% PMP	(2) 5 x 100% PMP	6
Aerobic	(2) 1 x 50% PMP	(1) 1 x 50% PMP (1) 1 x 60% PMP	(2) 1 x 60% PMP	6
Total	6	6	6	18

Note: Total number of sessions is 18 and the aerobic training intensity increased after the third week.

6.2.5 Electromyography

Surface EMG activity was recorded using active electrodes at the following sites: flexor carpi ulnaris (FCU); biceps brachii (BB); triceps brachii lateral (TB); anterior deltoid (AD); infraspinatus (IS); external oblique (EO). A passive reference electrode (Blue sensor M-00-S, Ambu Ltd, Cambs, UK) was placed centrally on the right patella. Before each test all sites were cleaned with isopropyl alcohol to remove oil and dirt. The bipolar double-differential (16-3000Hz bandwidth, x300 gain) active electrodes (MP-2A, Linton, Norfolk, UK) were firmly taped to the skin surface with the wires also taped down. Once the first test was complete each electrode placement was marked on the participant's skin with a surgical marker pen, the electrode was placed on the marked site for subsequent tests (section 3.4.5.i). Full details of EMG processing and synchronisation are described in section 3.4 and 3.6 respectively.

6.2.6 Kinematic analysis

Kinetic data were collected via infrared reflective markers attached using double-sided tape on the following anatomical landmarks; ulnar styloid process (USP); lateral epicondyle of the humerous (LEH); most prominent superior position on the conoid tubercle on the clavical (CTC), and 7th cervical vertebrae C7 (C7) (Figure 5.1). The ACE was also marked with infrared reflective markers at the following static points, directly vertical to centre of bottom bracket (CBB), and top and centre of seat post pillar (TCSP) (Figure 5.1). For further details of

anatomical landmarks and ACE markers refer to Table 3.11. After the first test, the placement of each participant's reflective marker was marked on the participant's skin with a surgical marker pen as for the EMG measurements. The wrist, elbow and inner shoulder was used to determine the range of movement of the elbow joint (°; ROM) and is comparable to that of the knee joint in cycling (Zehr and Chua, 2000). To determine the change in distance (mm) between the torso and the ACE the distance between C7 and the static point directly vertical to the centre of ergometer bottom bracket ($C7_D$) was recorded throughout each CWT and measured as section 3.5.1. To measure velocity and change in direction of the trunk rotation velocity in relation to the ergometer, angular velocity ($°\cdot s^{-1}$) between C7, CTC and TCSP was recorded throughout each CWT and measured as section 3.3.3. The above variables were analysed at the following times 30 s, 120 s and at T_{lim}. Each marker position is presented in Table 3.11 for a full description of each marker position. Full details of kinematic processing and synchronisation are described in section 3.5 and 3.6 respectively.

6.2.7 Post-training tests

The tests in section 6.2.3 were replicated at the end of the training programme in the following order, WAnT, a CWT to exhaustion at the pre-training 100% PMP exercise intensity ($Post_{ABS}$), VO_{2peak} test and a further CWT test at 100% PMP based on the post-training PMP ($Post_{100\%}$). Recovery between tests, the time of testing and laboratory conditions were as section 6.2.1.

6.2.8 Statistical analysis

All data are presented as the mean ± standard deviations (s). All analyses were performed using the Statistical Package for Social Sciences (v 17.0; SPSS Inc., Chicago, IL). Differences in VO_2, RER, HR, PMP, T_{lim}, RPE, EMG activity for each muscle and kinematic variables corresponding to 30-s, 120-s and volitional exhaustion (T_{lim}) were analysed using separate two-way analysis of variance with repeated measures on training. For the WAnT and VO_{2peak} test variables analysis was undertaken using a paired t-test. Where SPSS presented P values of P = 0.000 these are reported as $P < 0.001$. Individual differences between means were located using Bonferroni post-hoc correction when there were multiple comparisons for data sets for, metabolic responses, T_{lim}, peak minute power, EMG, and kinematics. A two-tailed level of significance was set at $P \leq 0.05$ for

all tests unless otherwise specified. In the discussion, to interpret the true meaningfulness of the data, test-retest power output for the WAnT (as discussed in section 3.3.1.i) and physiological response data (as discussed in section 3.3.2ii & 3.3.3.ii) has been included.

6.3 Results

6.3.1 Interval and constant load exercise training sessions

Peak power output (W) for the WAnT for the first and penultimate sprint ($Sprint_{PEN}$) of each training session is shown in Table 6.2. Uncorrected PPO, for the first sprint, decreased by 12 W from the start to the end of the training programme whereas the PPO for the penultimate repetition increased by 19 W. End HR for the penultimate 10 s sprint increased by 7 beats·min^{-1} by the final sprint training session compared to the end HR during the initial sprint training sessions.

Table 6.2 Uncorrected PPO (W) and end HR response during WAnT interval training (mean ± *SD*).

	Repeated sprints sessions					
	Week 1-2		Week 2-3		Week 3-4	
	First sprint	$Sprint_{PEN}$	First sprint	$Sprint_{PEN}$	First sprint	$Sprint_{PEN}$
Uncorrected PPO (W)	429 (95)	354 (89)	407 (92)	339 (84)	417 (99)	373 (75)
End HR		155 (8)		162 (12)		162 (12)

* means are for a two week block of training.

Mean heart rate for the first and last interval of the interval training session and mean HR over the 30 min submaximal training sessions are presented in Table 6.3. The results from the interval training sessions indicate that although the number of repetitions increased every two weeks there was a gradual reduction in both peak and mean HR from session 1 to session 6. The reduction in heart rate is mirrored by a reduction in RPE_L and RPE_{CR} (Figure 6.1). The 30 min training mean HR indicates that there was a slight increase in heart rate after the increase in the PMP exercise intensity from 50% to 60%. However, RPE_L and RPE_{CR} remained the same (Figure 6.2).

Table 6.3 Interval & 30 min heart rate response during training (mean ± *SD*).

Session no.	Interval training peak HR		Training 30 min
	First rep	End rep	Mean HR
1	155 (15)	173 (14)	127 (15)
2	151 (19)	172 (12)	127 (15)
3	146 (16)	168 (13)	120 (19)
4	146 (13)	169 (11)	129 (17)
5	145 (15)	164 (14)	132 (18)
6	143 (19)	164 (16)	135 (17)

Ratings of perceived exertion (local and central) at the end of the interval and 30 min training sessions are shown in Figure 6.1 and 6.2. Note: for the 30 min training, sessions 1-3 where completed at 50% of PMP and sessions 4-6 at 60% PMP.

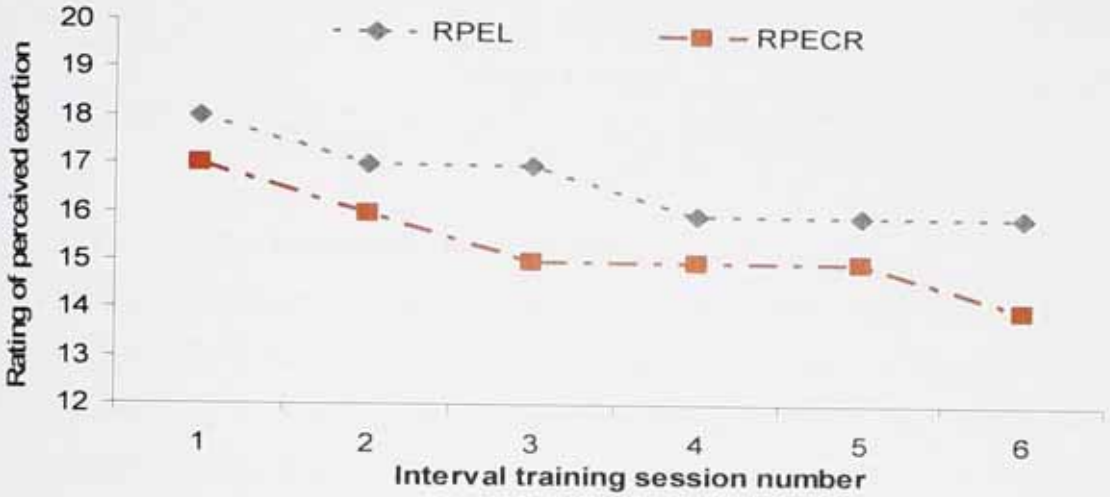


Figure 6.1 Rating of perceived exertion (RPE_L and RPE_{CR}) after each interval training session.

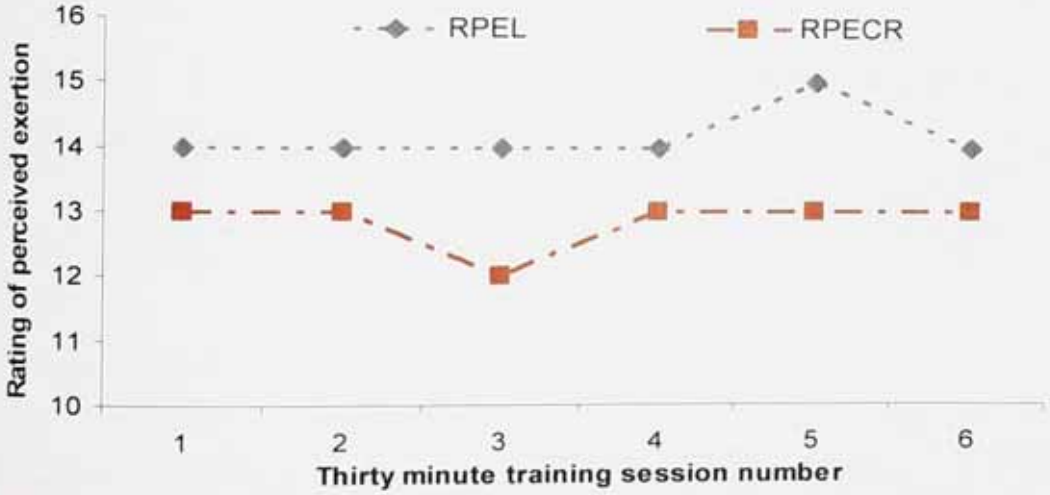


Figure 6.2 Rating of perceived exertion (RPE_L and RPE_{CR}) after each 30 min training session.

6.3.2 Anthropometry and body composition

There were no significant differences for body mass, grip strength arm girth and arm skinfold following training (Table 6.4). However, there was a significant increase for relaxed upper arm girth post-training ($P = 0.001$).

Table 6.4 Anthropometric measures pre and post-training (mean ± *SD*).

Measure		Pre	Post
Body mass (kg)		72.0 (11.9)	70.8 (10.7)
Grip strength (kg·N)	Left	42.0 (6.9)	41.3 (6.3)
	Right	43.3 (8.8)	44.3 (7.9)
Arm girth (cm)	Relaxed	30.3 (3.2)	30.9 (3.2)$
	Flexed & tensed	32.6 (3.0)	32.9 (3.0)
	Forearm relaxed	27.3 (3.0)	27.1 (1.9)
Skinfold (mm)	Biceps brachii	4.1 (0.8)	4.2 (1.0)
	Triceps brachii	11.3 (3.8)	11.2 (3.6)

$ Significant difference ($P < 0.01$) from pre-training value.

6.3.3 Incremental exercise test

The peak physiological responses to the incremental exercise test for VO_{2peak} are presented in Table 6.5. No significant differences were observed before and after training for VO_{2peak}, respiratory exchange ratio or peak heart rate. However, a significant increase was observed post training for PMP ($P < 0.001$) suggesting that the increase in PMP was not the result of changes in peak oxygen uptake, RER or HR_{max}.

Table 6.5 Incremental exercise test pre and post-training peak physiological responses (mean ± *SD*).

	Pre-training	Post-training
VO_{2peak} ($l \cdot min^{-1}$)	2.33 (0.67)	2.29 (0.48)
RER	1.37 (0.08)	1.35 (0.09)
HR_{peak} ($beats \cdot min^{-1}$)	177 (19)	179 (13)
PMP (W)	127 (27)	145 (26)†

† Significant difference ($P < 0.01$) from pre-training value.

6.3.3.i Representative participant data during the incremental exercise tests

The peak physiological response ($l \cdot min^{-1}$) to the incremental exercise test for VO_{2peak} (pre and post training) are presented in Figure 6.3 for two typical participants (marked participant 1 and participant 2). Both graphs indicated that a similar VO_{2peak} was reached pre and post training. However, it took longer for VO_{2peak} to be reached post training for both participants. Therefore, post training both participants, after the initial early stages of the test, were exercising at the same work load with a lower oxygen uptake.

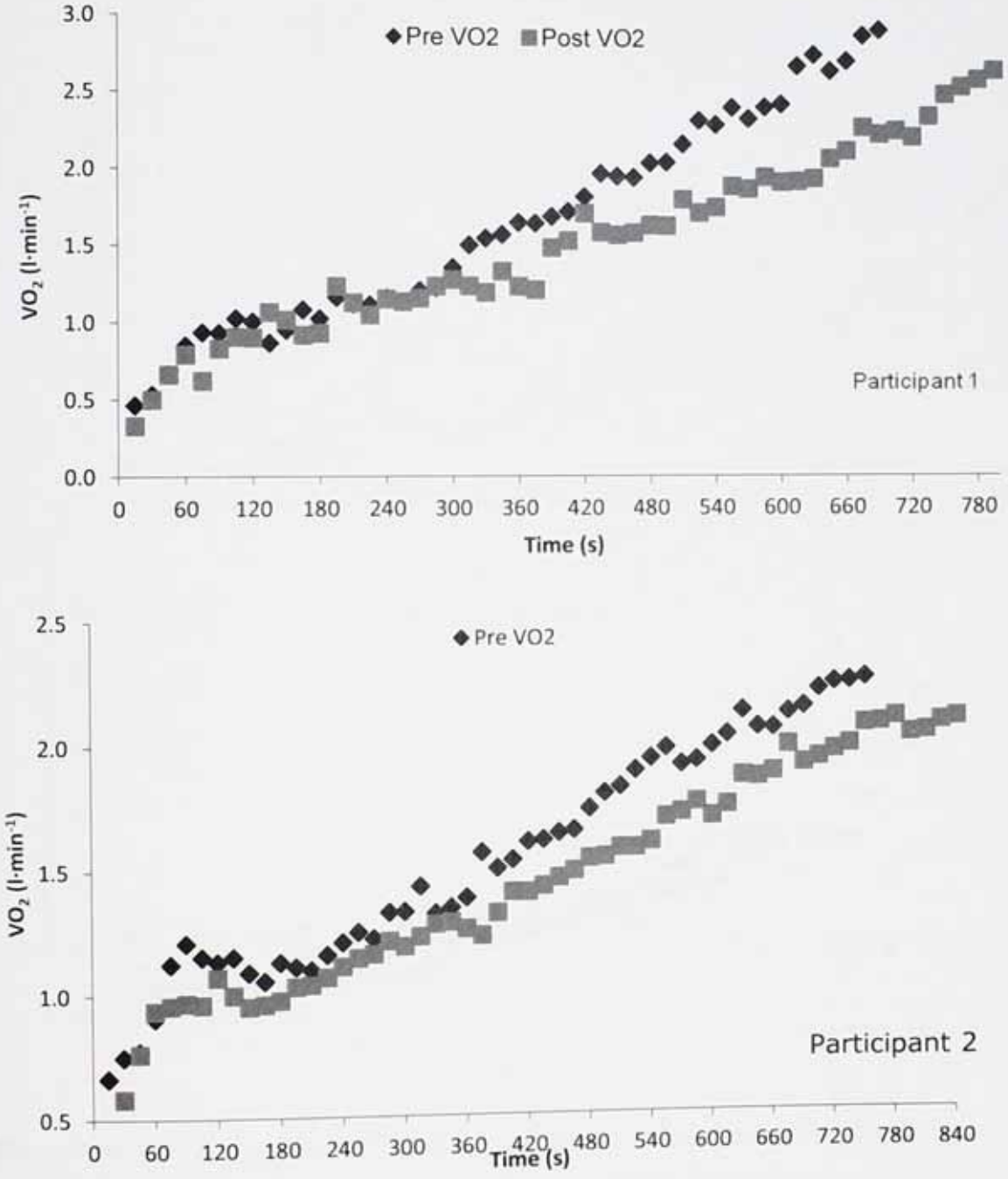


Figure 6.3. Typical response during incremental exercise test measured as VO_2 ($l \cdot min^{-1}$) to exhaustion (pre and post training) for typical participant 1 and typical participant 2.

The breathing frequency (1/min) to the incremental exercise test for VO_{2peak} (pre and post training) are presented in Figure 6.4 for two typical participants (marked participant 1 and participant 2). Both graphs indicated that despite a similar breathing frequency being reached at the end of the test pre and post training it took longer for the same breathing frequency to be reached post training for both participants. Therefore, post training both participants, after the initial early stages of the test, were exercising at the same work load with a lower breathing frequency.

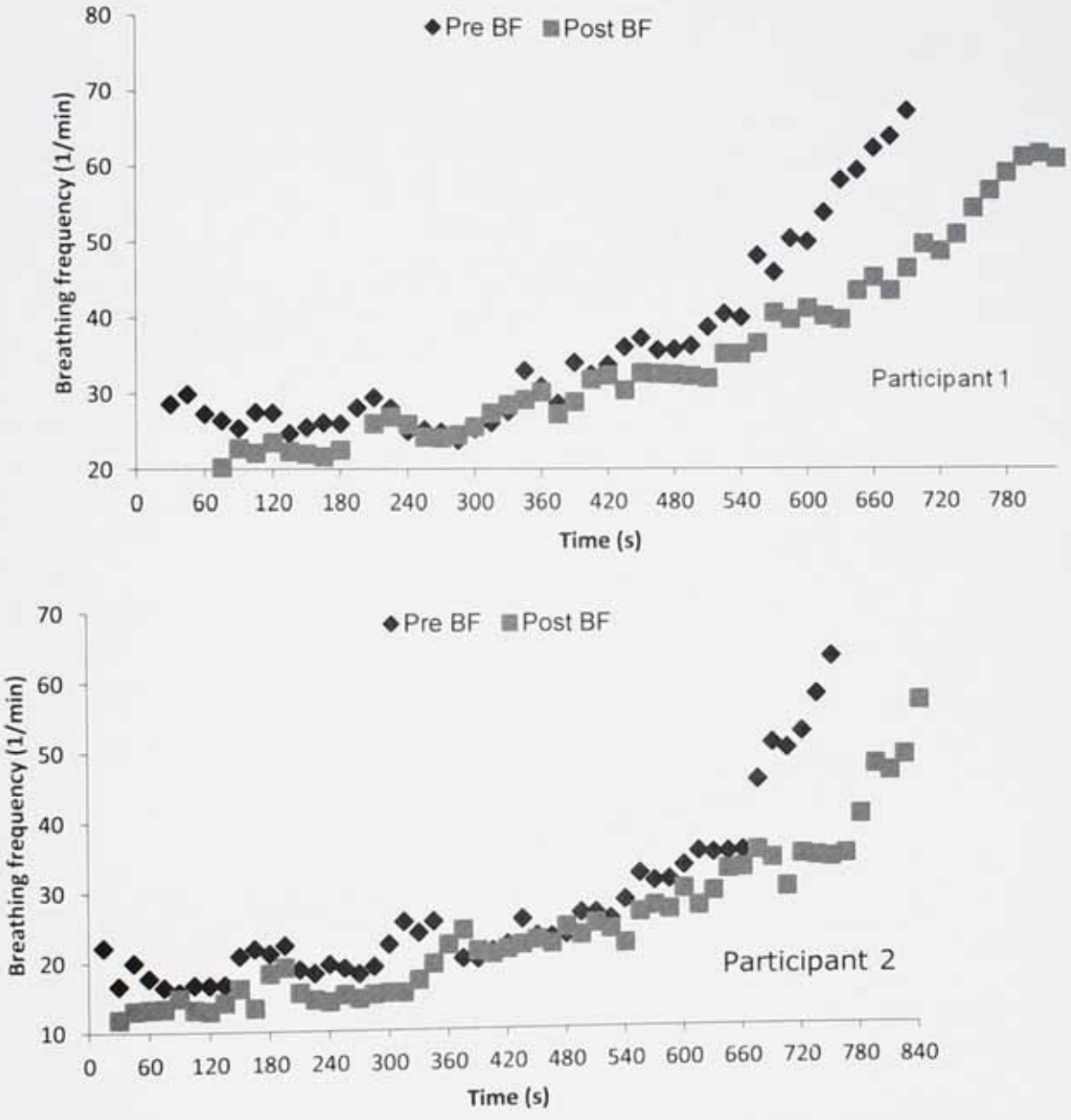


Figure 6.4. Typical response to an incremental exercise test measured as breathing frequency (1/min) pre and post training for typical participant 1 and typical participant 2.

The ventilatory equivalent for oxygen, measured as $V'E/V'O_2$ ($l \cdot min^{-1}$), response to the incremental exercise test for VO_{2peak} (pre and post training) are presented in Figure 6.5 for two typical participants (marked participant 1 and participant 2). Both graphs indicated that a slightly greater $V'E/V'O_2$ was reach post training. Initially towards the later half of the test the $V'E/V'O_2$ response was lower post training for both participants. Therefore, post training both participants, after the initial early stages of the test, were exercising at the same work load with a lower $V'E/V'O_2$.

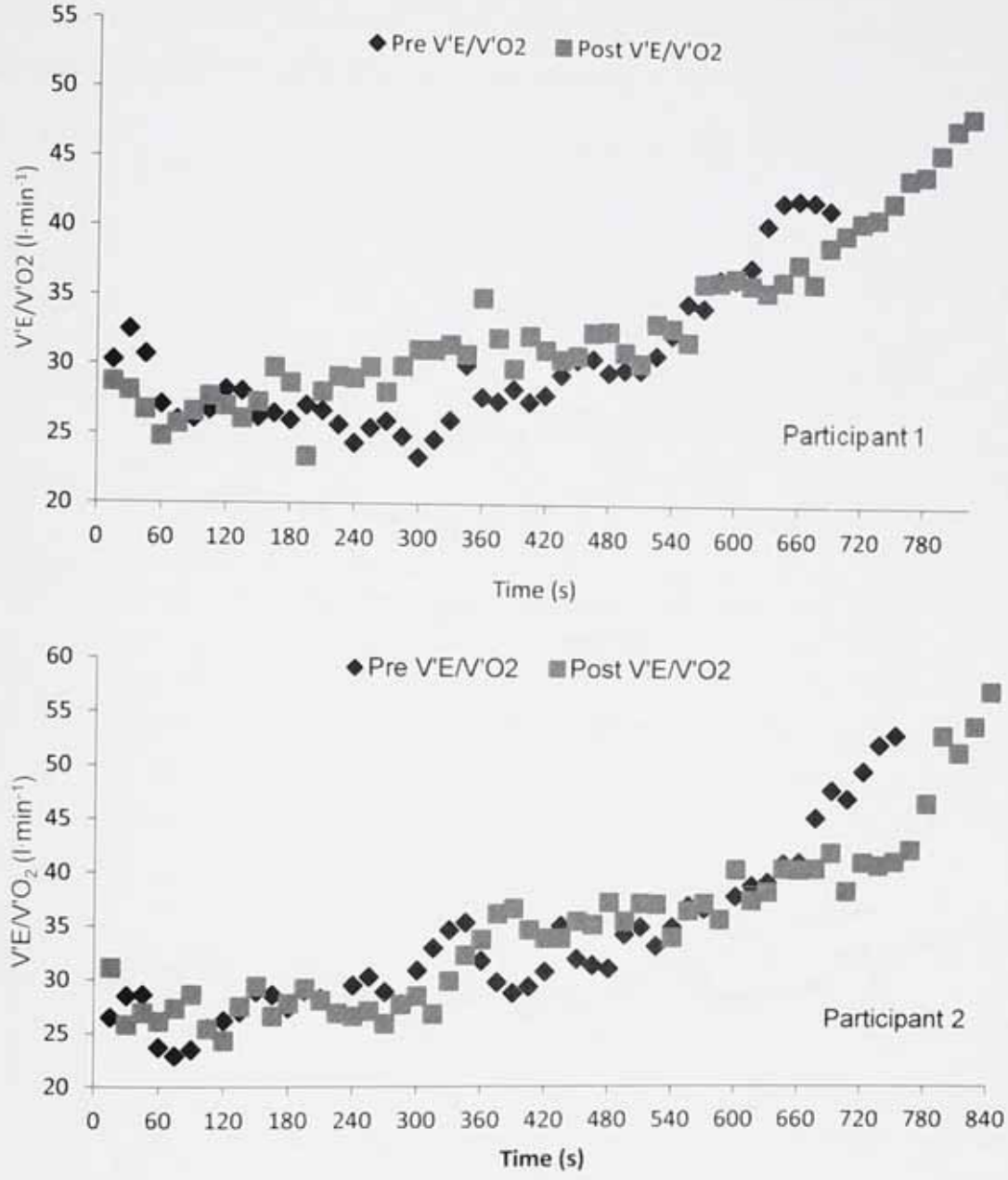


Figure 6.5. Typical response to an incremental exercise test measured as $V'E/V'O_2$ ($l \cdot min^{-1}$) pre and post training for typical participant 1 and typical participant 2.

The ventilatory equivalent for carbon dioxide, measured as $V'E/V'CO_2$ ($l \cdot min^{-1}$), response to the incremental exercise test for VO_{2peak} (pre and post training) are presented in Figure 6.6 for two typical participants (marked participant 1 and participant 2). For participant 1 a greater $V'E/V'CO_2$ was reach post training and for participant 2 a similar $V'E/V'CO_2$ was reached post training. Participant 2 maintained a greater $V'E/V'CO_2$ post training and was able to achieve greater power output with a greater $V'E/V'CO_2$. Initially towards the later half of the test the $V'E/V'CO_2$ response was lower post training for both participant 2. Therefore, post training participant 2, after the initial early stages of the test, was exercising at the same work load with a lower $V'E/V'CO_2$.

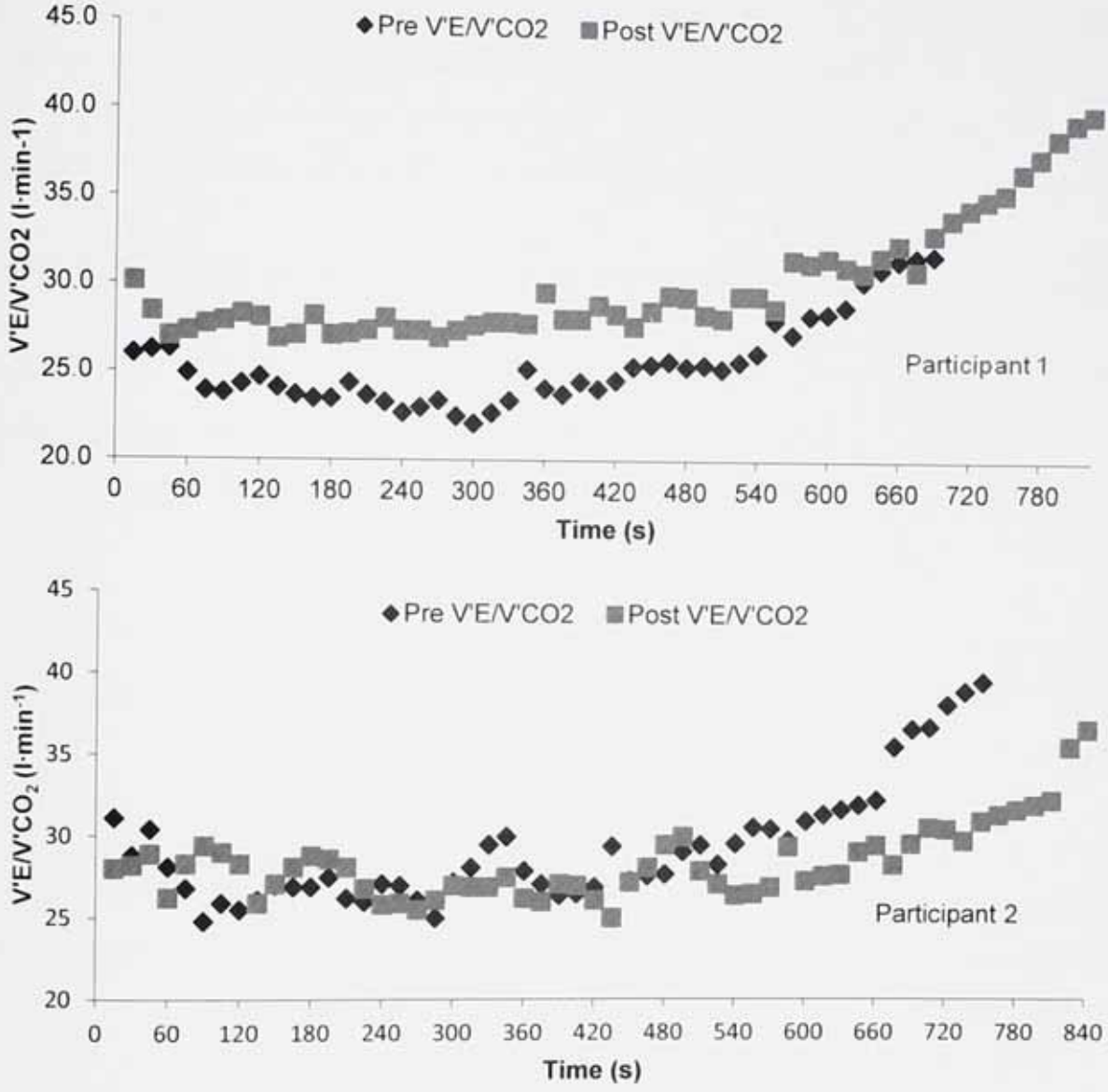


Figure 6.6. Typical response to an incremental exercise test measured as $V'E/V'CO_2$ ($l \cdot min^{-1}$) pre and post training for typical participant 1 and typical participant 2.

6.3.4 Wingate anaerobic test

6.3.4.i Performance measures

The WAnT performance variables are presented in Table 6.6. A significantly greater PPO was observed post training for both corrected and uncorrected PPO (t = 5.369, t = 6.630, respectively, $P < 0.001$) (Table 6.6). Significant increases were also observed after training for: corrected and uncorrected MPO (t = -5.156, t = 6.630, respectively, $P < 0.001$), peak and mean cadence (t = -7.059, t = -5.300, respectively, $P < 0.001$) and uncorrected and corrected FI (t = -2.332, t = 2.399 and P = 0.035, P = 0.040, respectively). No significant differences were observed for corrected or uncorrected PO_{min}, final cadence and both uncorrected and corrected PPO_{time} ($P > 0.05$). Increases in peak and mean cadence coupled with no differences in minimum PO indicate an increased PPO and a greater rate of fatigue post-training. Additionally, although training significantly increased PPO it did not appear to influence time to peak power.

Table 6.6 Performance variables for the WAnT pre and post-training (mean ± *SD*).

		Pre	Post	Change (±)
Uncorrected	PPO (W)	373 (83)	429 (80)	+ 56 (22)†
	MPO (W)	326 (66)	364 (63)	+ 38 (25) †
	PO_{min} (W)	272 (52)	287 (52)	+ 15 (38)
	PPO_{time} (s)	9 (2)	9 (1)	0 (2)
	FI (%)	27 (10)	33 (7)	+ 6 (9)*
Corrected	PPO (W)	503 (103)	636 (65)	+ 133 (86) †
	MPO (W)	346 (67)	384 (65)	+ 38 (25) †
	PO_{min} (W)	254 (57)	247 (92)	- 7 (94)
	PPO_{time} (s)	4 (2)	5 (2)	+ 1 (3)
	FI (%)	50 (11)	61 (14)	+ 11 (16)*
Cadence	Peak ($rev \cdot min^{-1}$)	133 (16)	152 (13)	+ 19 (9) †
	Mean ($rev \cdot min^{-1}$)	117 (12)	129 (9)	+ 12 (8) †
	Final ($rev \cdot min^{-1}$)	98 (15)	102 (11)	+ 4 (13)

† Significant difference ($P < 0.001$) from pre-training value.

* Significant difference ($P \leq 0.05$) from pre-training value.

6.3.4.ii Electromyography responses

Electromyographic activation was measured for each muscle at corrected PPO, uncorrected PPO and minimum power output. There were no significant differences observed at corrected PPO, uncorrected PPO and PO_{min} for flexor carpi ulnaris, biceps brachii, triceps brachii and external oblique post-training. At uncorrected PPO there was a significant decrease in peak EMG activity following training for the anterior deltoid ($P = 0.048$) with infraspinatus approaching significance ($P = 0.062$). No significant differences for these muscles were observed at corrected PPO and PO_{min}. Therefore, increases in uncorrected PPO were accompanied by reductions in EMG activity for the anterior deltoid and possibly the infraspinatus.

6.3.4.iii Kinematic analysis

There were no significant differences in $C7_D$ relative to the ACE pre and post training. At uncorrected PPO elbow ROM decreased following training (57° ± *vs* 54°, respectively) and approached significance ($t = 1.897$, $P = 0.084$). A significant increase in trunk rotation velocity was observed post-training at corrected PPO ($t = -3.429$, $P = 0.006$) (Figure 6.7). Therefore, as corrected PPO and uncorrected PPO increased post-training trunk rotational velocity also increased suggesting that post-training trunk rotational velocity, PO and cadence were linked for the WAnT. The correlation coefficient between peak cadence and trunk rotational velocity at uncorrected PPO was not significant ($r = .473$, $P > 0.05$). However, when compared to pre-training values were $r = .122$ ($P > 0.05$) which indicates changes occurred in the relationship between trunk rotational velocity and peak cadence following training.

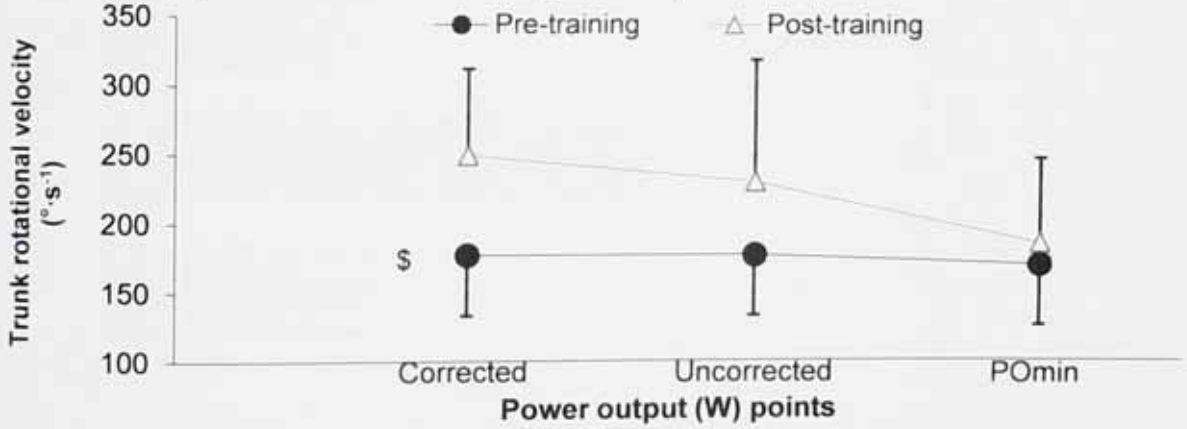


Figure 6.7 Changes in trunk rotational velocity ($^{\circ}\cdot s^{-1}$) before, and after training for the WAnT for corrected and uncorrected PPO and PO_{min}. $ Significant difference ($P < 0.01$) between correct & uncorrected power output.

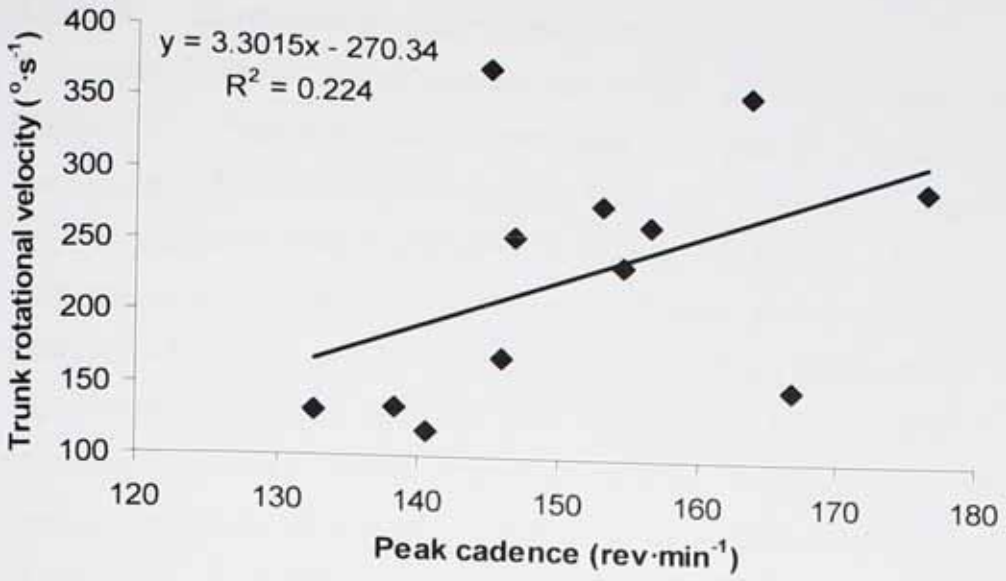


Figure 6.8. Relationship after training between trunk rotational velocity and peak cadence (r = .473, P > 0.05).

6.3.5 Continuous work tests

6.3.5.i Time to exhaustion

A significant difference was observed for time to exhaustion achieved in the continuous work tests (F = 16.604, P = 0.001). Post-hoc analysis revealed a significant increase in time to exhaustion for $Post_{ABS}$ when compared to $Pre_{100\%}$ (506 (207) and 244 (82) s, respectively, P = 0.002) and $Post_{100\%}$ (255 (45) s, P = 0.008) (Figure 6.9). Therefore, after training time to exhaustion significantly increased. Additionally, training did not change time to exhaustion at the new level of 100% PMP but significantly increased the absolute load (W) accomplished before fatigue limits the ability to continue the exercise.

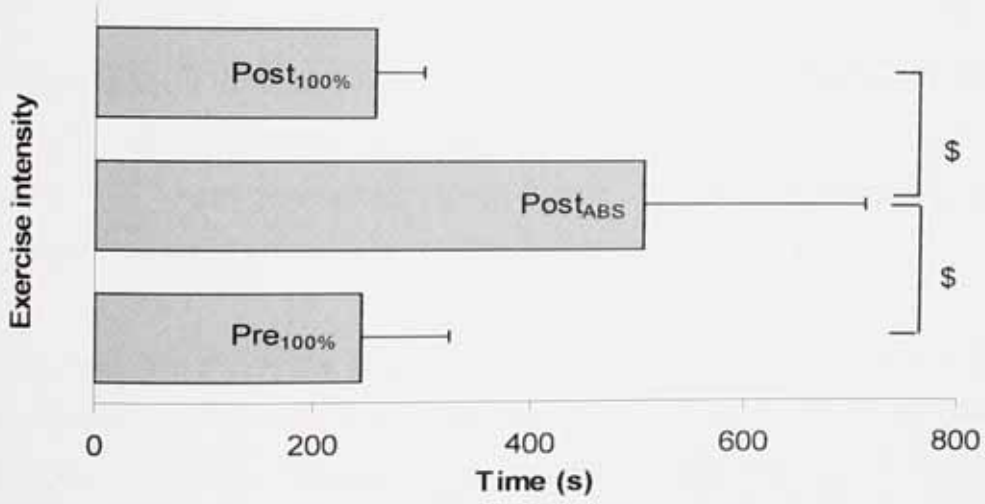


Figure 6.9. Time to exhaustion (T_{lim}) before and after training.
$ Significant difference (P < 0.01) between resistive loads.

6.3.5.ii Cardiorespiratory response

There was a significant difference for oxygen uptake between time and trial (Table 6.7). Post-hoc analysis revealed a greater VO_2 for both $Pre_{100\%}$ and $Post_{100\%}$ at 120 s when compared to $Post_{ABS}$. There was a significant interaction for RER between time and trial ($F = 7.253$, $P = 0.003$). Post-hoc analysis revealed a greater RER at 120 s for the $Pre_{100\%}$ *vs* $Post_{ABS}$ trials and a lower $Post_{ABS}$ *vs* both $Pre_{100\%}$ and $Post_{100\%}$ at exhaustion (Table 6.7). There was a significant interaction between time and trial for HR ($F = 5.117$, $P = 0.002$). Post-hoc analysis revealed a greater HR at 30 s $Post_{ABS}$ when compared to $Post_{100\%}$ and a greater heart rate $Post_{ABS}$ compared to $Pre_{100\%}$ at exhaustion (Table 6.7). The results suggest that the significant changes in oxygen uptake, RER and HR increased T_{lim} for $Post_{ABS}$.

Table 6.7 Cardiorespiratory response at 30 s, 120 s and T_{lim} before and after training (mean ± *SD*).

Variable	PMP	30 s	120 s	Exhaustion
VO_2	$Pre_{100\%}$	1.21 (0.22)	1.95 (0.42)	2.31 (0.59)
	$Post_{ABS}$	1.13 (0.21)	1.75 (0.42)[c]	2.33 (0.49)
	$Post_{100\%}$	1.14 (0.21)	1.94 (0.37)	2.20 (0.43)
RER	$Pre_{100\%}$	0.97 (0.09)	1.39 (0.11)	1.39 (0.15)
	$Post_{ABS}$	0.97 (0.12)	1.30 (0.07)[a]	1.27 (0.10)[c]
	$Post_{100\%}$	0.91 (0.07)	1.33 (0.07)	1.38 (0.08)
HR	$Pre_{100\%}$	126 (15)	155 (15)	172 (13)
	$Post_{ABS}$	129 (18)[b]	150 (17)	179 (12)[a]
	$Post_{100\%}$	123 (13)	155 (11)	174 (14)

[a] Significantly different from $Pre_{100\%}$. [b] Significantly different from $Post_{100\%}$.
[c] Significantly different from $Pre_{100\%}$ and $Post_{100\%}$.
Note: VO_2 = oxygen consumption ($l \cdot min^{-1}$), RER = respiratory exchange ratio, HR = heart rate ($beats \cdot min^{-1}$), PMP = peak minute power.

The peak physiological responses ($l \cdot min^{-1}$) to the PMP (pre and post training) are presented in Figure 6.10 for two typical participants (marked participant 1 and participant 2). Participant 1 was able to the same power output ($Post_{ABS}$) with a lower VO_2 and the $Post_{100\%}$ PMP was able to maintain a greater power output with a lower VO_2. Participant 2 was able to maintain the same power output ($Post_{ABS}$)

with a slightly lower VO_2 and the $Post_{100\%}PMP$ was able to maintain a greater power output a similar VO_2 as pre training.

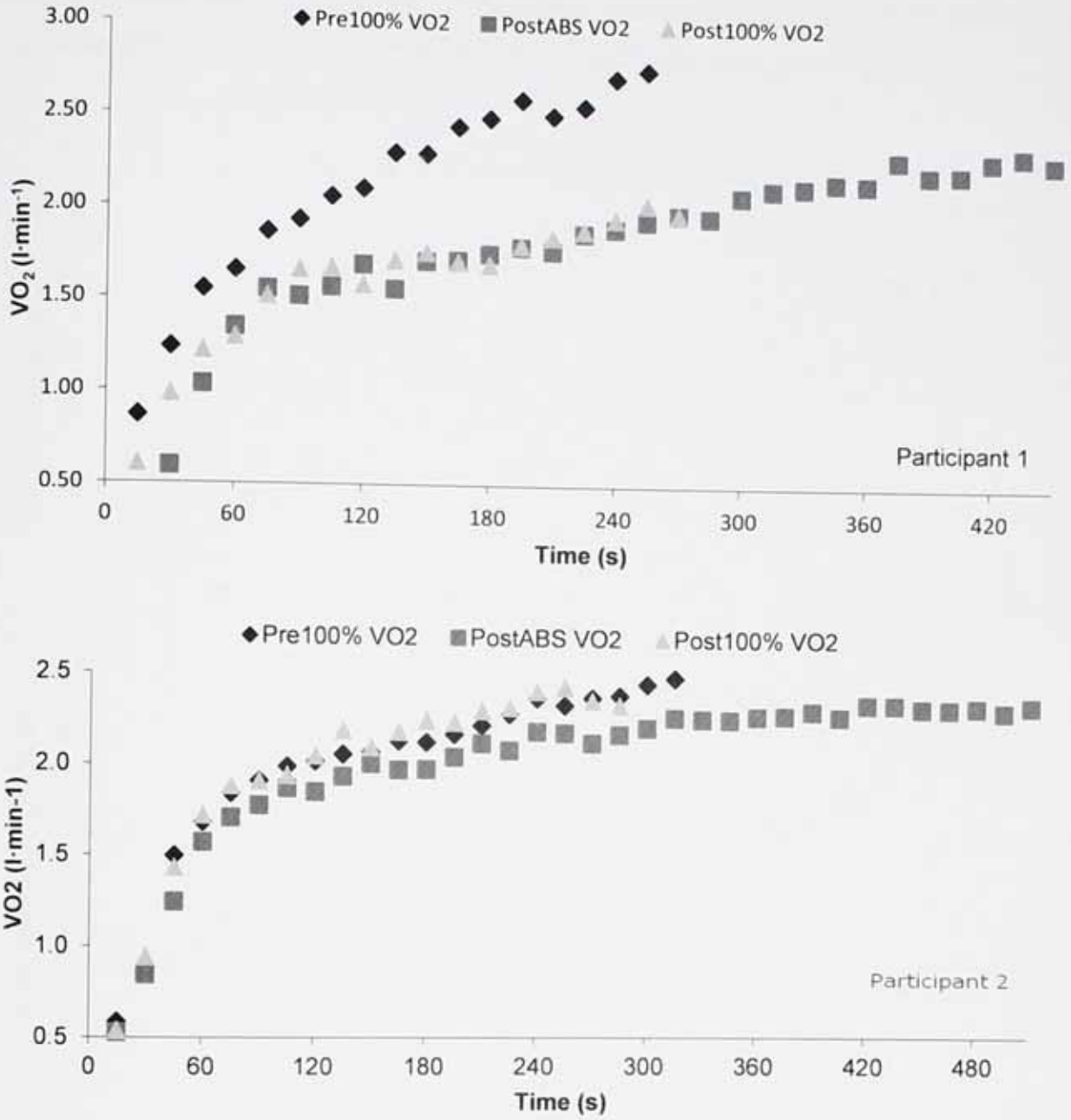


Figure 6.10 Typical response during PMP trails measured as VO_2 ($l \cdot min^{-1}$) (pre and post training) for typical participant 1 and typical participant 2.

The breathing frequency (1/min) to the PMP test (pre and post training) are presented in Figure 6.11 for two typical participants (marked participant 1 and participant 2). Both graphs indicate that despite a similar breathing frequency being reached at the end of the test $Post_{ABS}$ as $Pre_{100\%}$ it took longer for the same breathing frequency to be reached post training for both participants while maintaining the same power output for a grater duration. Therefore, post

training both participants, after the initial stages of the test, were exercising at the same work load with a lower breathing frequency. For the $Post_{100\%}$ exercise intensity participants had a similar breathing frequency but with a greater work load than the $Pre_{100\%}$ work load. Therefore, the participant breathing frequency may be affected by relative and absolute work load post training.

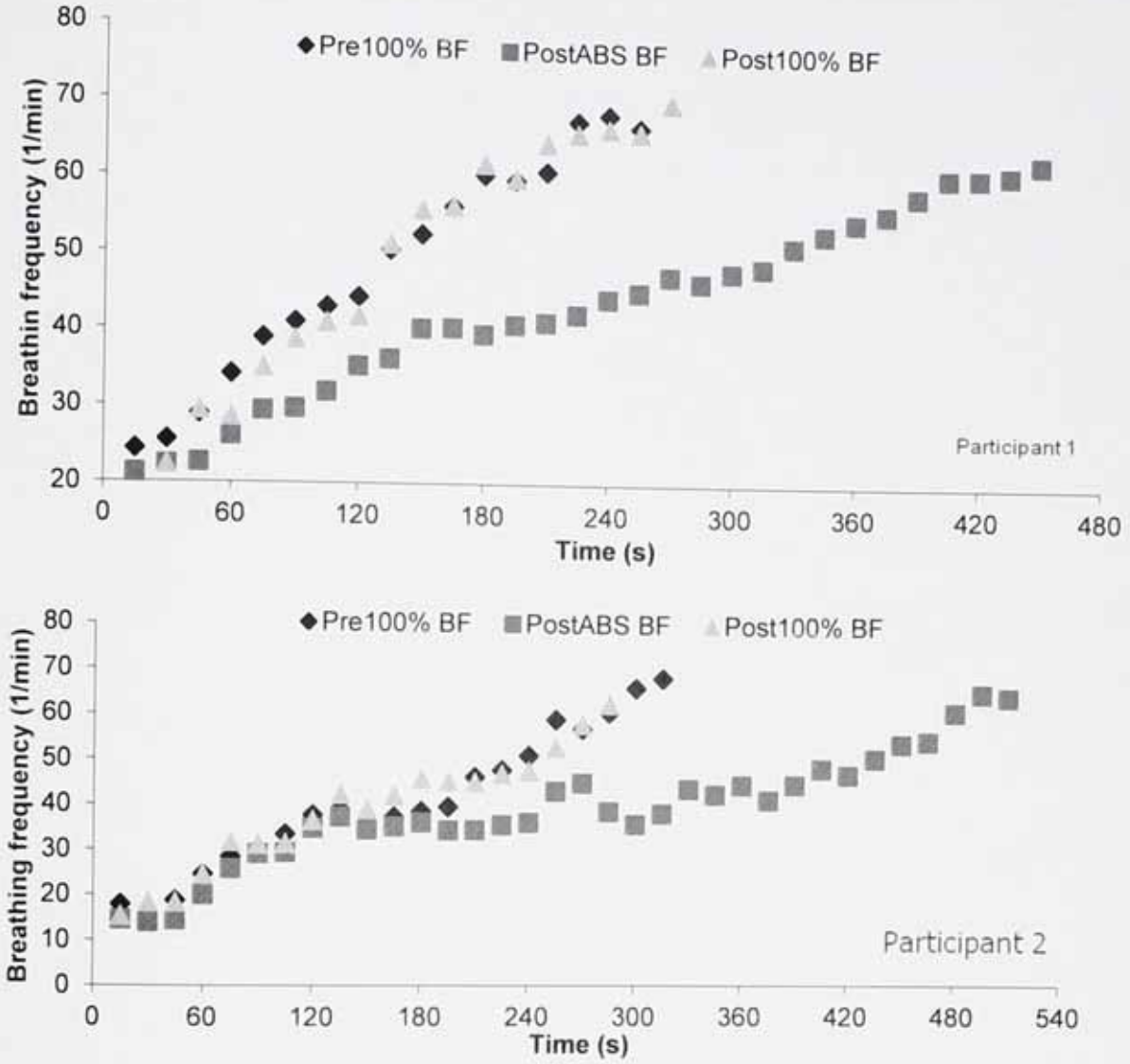


Figure 6.11. Typical response during PMP trails measured as BF (1/min) (pre and post training) for typical participant 1 and typical participant 2.

The ventilatory equivalent for oxygen measured as $V'E/V'O_2$ ($l \cdot min^{-1}$) to the PMP trials (pre and post training) are presented in Figure 6.12 for two typical participants (marked participant 1 and participant 2). Both graphs indicated that a slightly greater $V'E/V'O_2$ was reach post training for the $Post_{ABS}$ workload. Initially towards the later half of the test the $V'E/V'O_2$ response was lower post training for both participants. Therefore, post training both participants, after

the initial early stages of the test, were exercising at the same work load with a lower $V'E/V'O_2$. The response to the $Post_{100\%}$ compared to the $Pre_{100\%}$ work load indicates that during the first half of the trial both participants had a similar $V'E/V'O_2$ response despite a greater work load. However during the second half of the test participant 1 increased their $V'E/V'O_2$ response above the $Pre_{100\%}$ response, whilst participant 2 had a very similar response as their $Pre_{100\%}$ response.

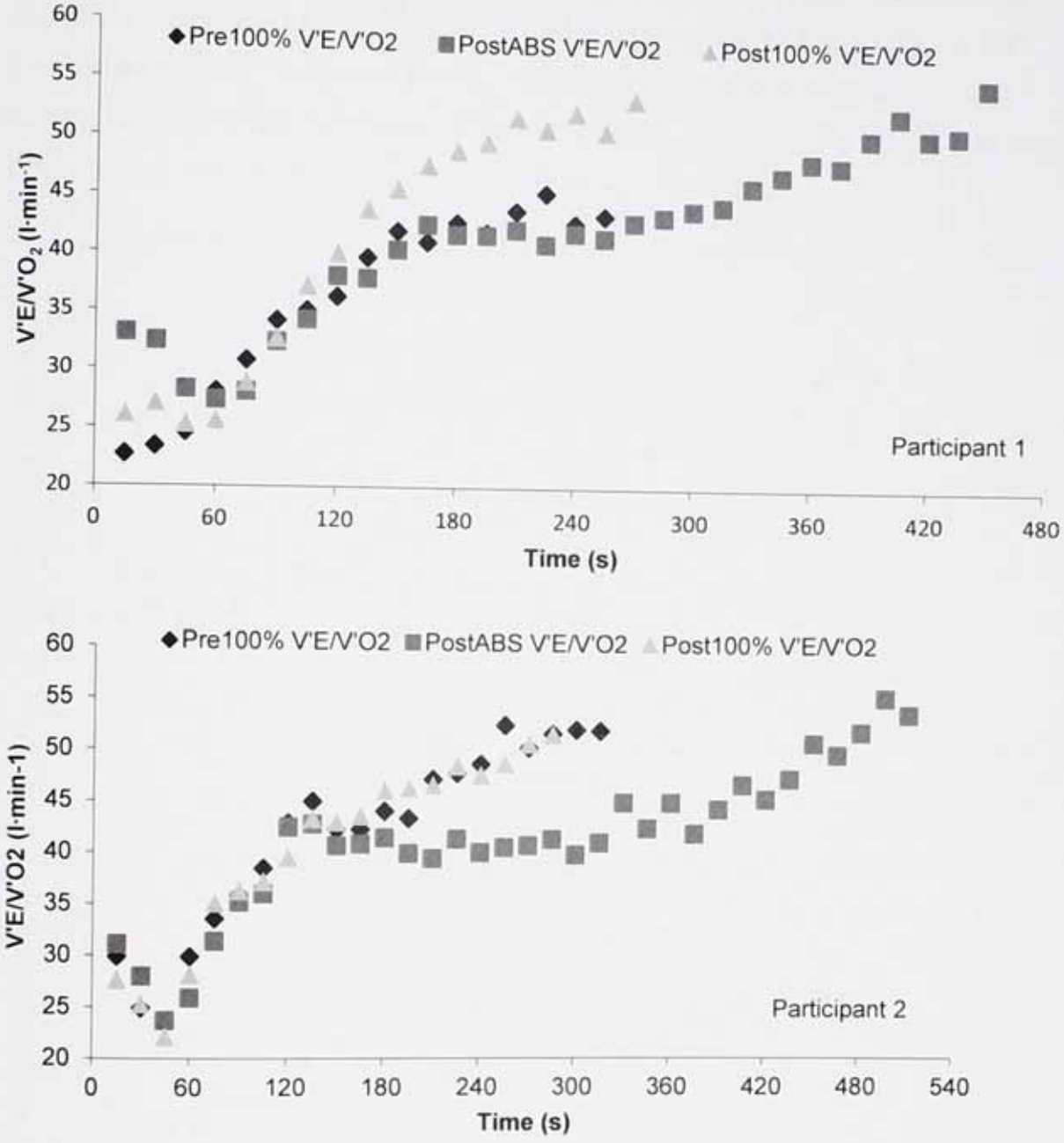


Figure 6.12 Typical response during PMP trials measured as $V'E/V'O_2$ ($l \cdot min^{-1}$) pre and post training for typical participant 1 and typical participant 2.

The ventilatory equivalent for carbon dioxide measured as $V'E/V'CO_2$ ($l \cdot min^{-1}$) to the PMP trials (pre and post training) are presented in Figure 6.13 for two typical

participants (marked participant 1 and participant 2). Both graphs indicated that a slightly greater $V'E/V'CO_2$ was reach post training for the $Post_{ABS}$ workload. Initially towards the later half of the test the $V'E/V'CO_2$ response was lower post training for both participants. Therefore, post training both participants, after the initial early stages of the test, were exercising at the same work load with a lower $V'E/V'CO_2$. The response to the $Post_{100\%}$ compared to the $Pre_{100\%}$ work load indicates that during the first half of the trial participant 1 had a slightly greater $V'E/V'CO_2$ response and participant 2 had a slightly lower $V'E/V'CO_2$ to the greater work load. However during the second half of the test participant 1 increased their $V'E/V'O_2$ response above the $Pre_{100\%}$ response, whilst participant 2 had a very similar response as their $Pre_{100\%}$ response.

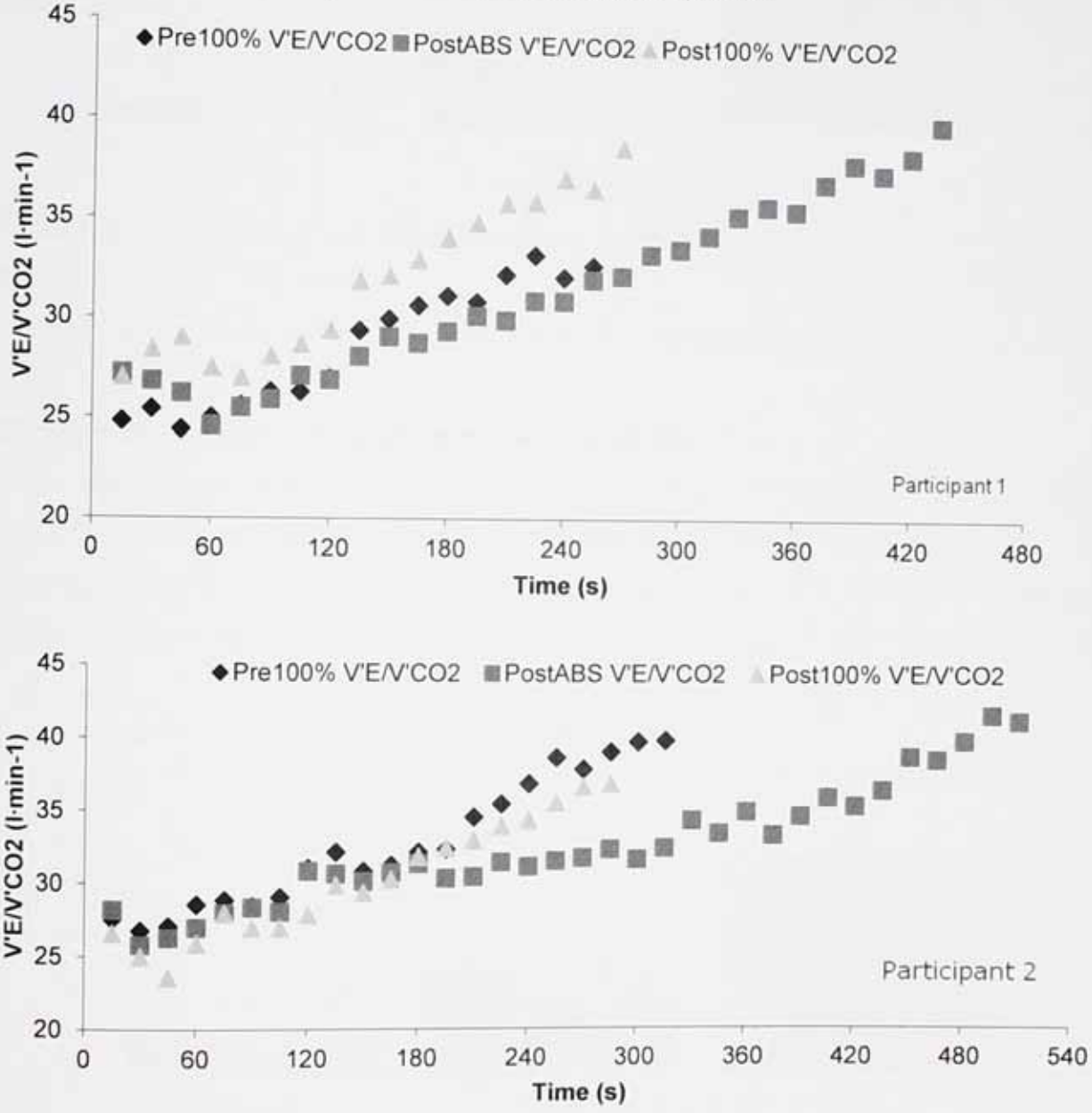


Figure 6.13. Typical response during PMP trails measured as $V'E/V'CO_2$ ($l \cdot min^{-1}$) pre and post training for typical participant 1 and typical participant 2.

6.3.5.iv Ratings of perceived exertion

Local rating of perceived exertion

There was a significant interaction for RPE_L between time and trial ($F = 22.444$, $P < 0.001$). Post-hoc analysis revealed differences between $Pre_{100\%}$ and $Post_{100\%}$ *vs* $Post_{ABS}$ at 30 s and 120 s with a significant reduction $Post_{ABS}$. Additionally $Pre_{100\%}$ was significantly lower than both $Post_{ABS}$ and $Post_{100\%}$ at T_{lim} (Fig. 6.14).

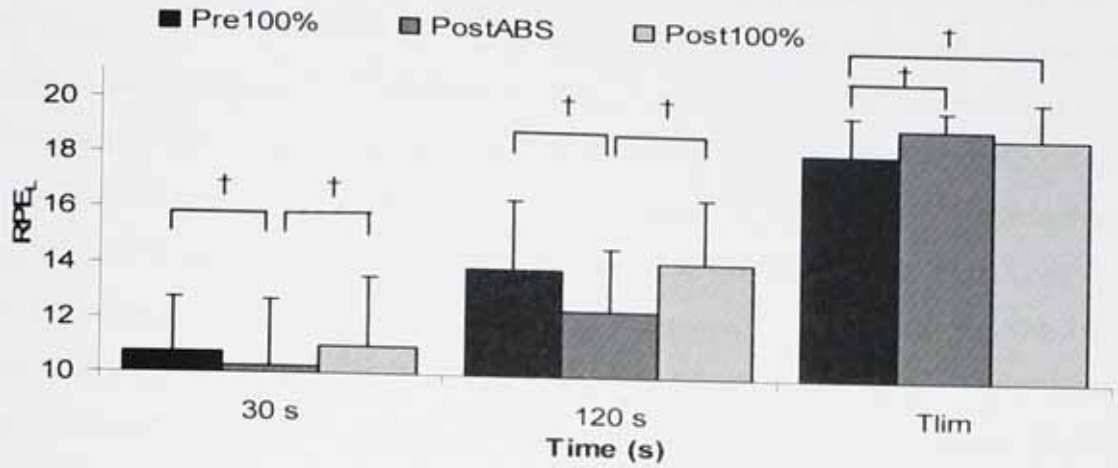


Figure 6.14 Rating of perceived exertion (RPE_L) during the 100% PMP trials before and after training.

† Significant difference ($P < 0.001$) between variables.

Cardiorespiratory rating of perceived exertion

There was a significant interaction for RPE_{CR} between time and trial ($F = 2.784$, $P = 0.050$). Post-hoc analysis revealed differences at 30 s and 120 s with a significant reduction $Post_{ABS}$ *vs* both $Pre_{100\%}$ and $Post_{100\%}$ and additionally $Post_{ABS}$ being significantly greater than both $Pre_{100\%}$ and $Post_{100\%}$ (Figure 6.15).

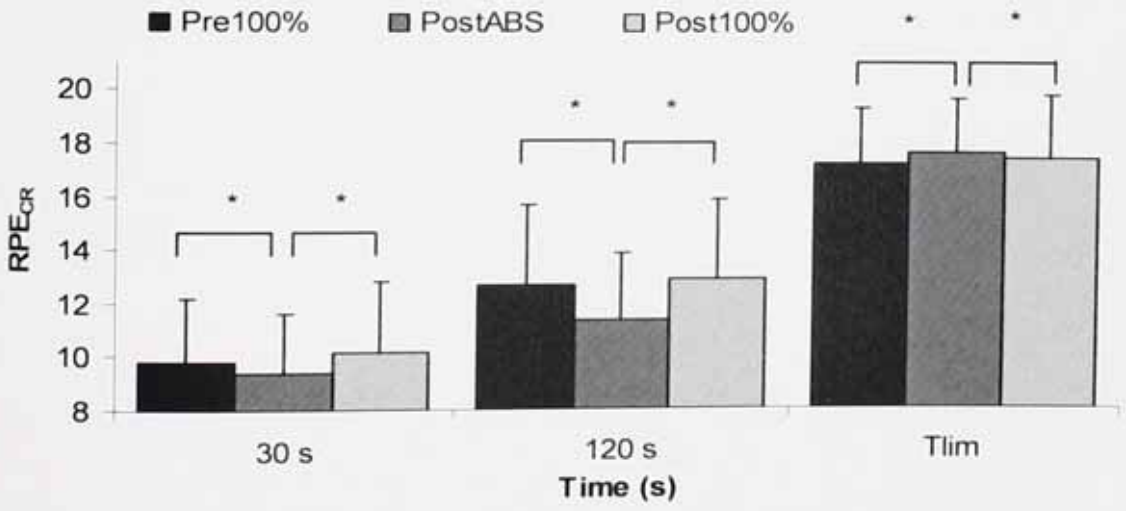


Figure 6.15 Rating of perceived exertion (RPE_{CR}) during the 100% PMP trials before and after training. * Significant difference ($P \leq 0.05$) between variables.

6.3.5.v Electromyography responses

Electromyographic activation was measured for each muscle at 30 s, 120 s and T_{lim}. There were no significant interactions for flexor carpi ulnaris, biceps brachii, triceps brachii, anterior deltoid and infraspinatus EMG. However, there was a significant main effect for time for all muscles (F = 10.944, P = 0.001; F = 13.426, $P < 0.001$; F = 8.223, P = 0.008; F = 21.57, $P < 0.001$; F = 32.146, $P < 0.001$, respectively) with EMG activity increasing over time. Post-hoc analysis for time showed significant differences for all muscles between $Pre_{100\%}$ *vs* both $Post_{ABS}$ and $Post_{100\%}$ and between $Post_{ABS}$ and $Post_{100\%}$ for all muscles except triceps brachii. There was a significant main effect for trial for triceps brachii (F = 5.283, $P < 0.013$). Post-hoc analysis revealed differences approached significance between $Pre_{100\%}$ and $Post_{ABS}$ (P = 0.060; Figure 6.16 (a)) and $Pre_{100\%}$ and $Post_{100\%}$ (P = 0.057). There was a significant interaction for the external oblique (F = 39.805, $P < 0.001$). Post-hoc analysis revealed a significant reduction in EMG activity at 120 s between $Post_{ABS}$ *vs* both $Pre_{100\%}$ and $Post_{100\%}$ ($P < 0.001$; Figure 6.16 (b)). Therefore, the results suggest that training has reduced triceps brachii activation at both exercise intensities, and reduced activation for the external oblique at 120 s for both exercise intensities following training. Additionally, external oblique activation could be related (R^2 = 0.239; figure not shown) to RPE_{CR} as both were reduced following training for $Post_{ABS}$. Training does not appear to influence activation of the flexor carpi ulnaris, biceps brachii, anterior deltoid and infraspinatus.

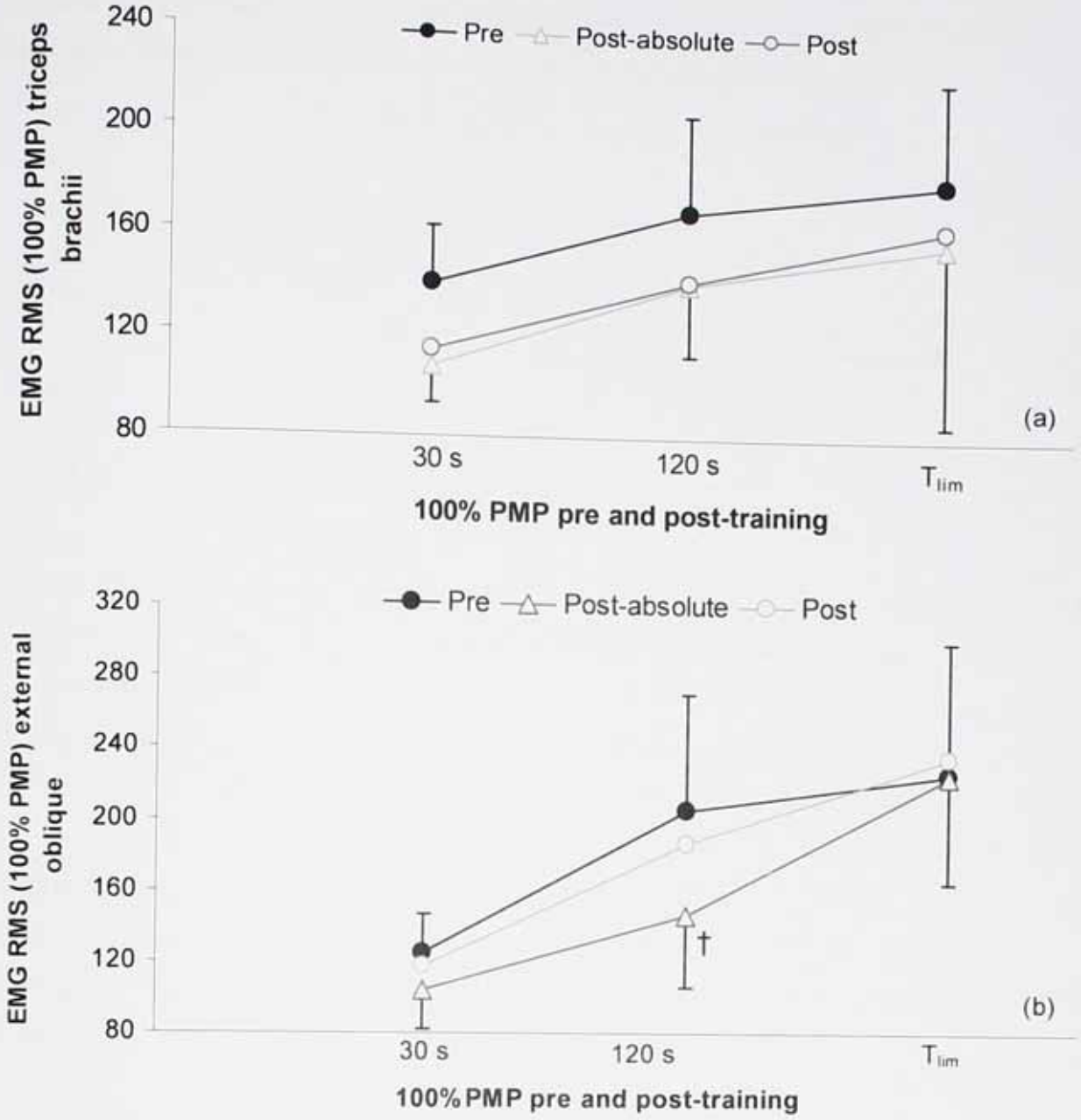


Figure 6.16 Normalised EMG (100% PMP) against exercise intensities (% PMP) before and after training against time (s). (a) Triceps brachii. (b) External oblique.

† Significant difference ($P < 0.001$) between $Pre_{100\%}$ and both $Post_{ABS}$ and $Post_{100\%}$.

6.3.5.vi Kinematic analysis

There was significant interaction between time and trial for elbow joint angle ($F = 6.561$, $P < 0.001$). Post-hoc analysis revealed a significant decrease in elbow ROM at 30 s between $Post_{100\%}$ *vs* both $Pre_{100\%}$ and $Post_{ABS}$. Elbow angle was significantly lower at 120 s for $Pre_{100\%}$ than both $Post_{ABS}$ and $Post_{100\%}$. Additionally, at time to exhaustion $Pre_{100\%}$ was significantly lower than $Post_{100\%}$

(Figure 6.17). Post-hoc analysis also revealed greater elbow joint angles between 30 s and both 120 s and T_{lim} ($P < 0.001$).

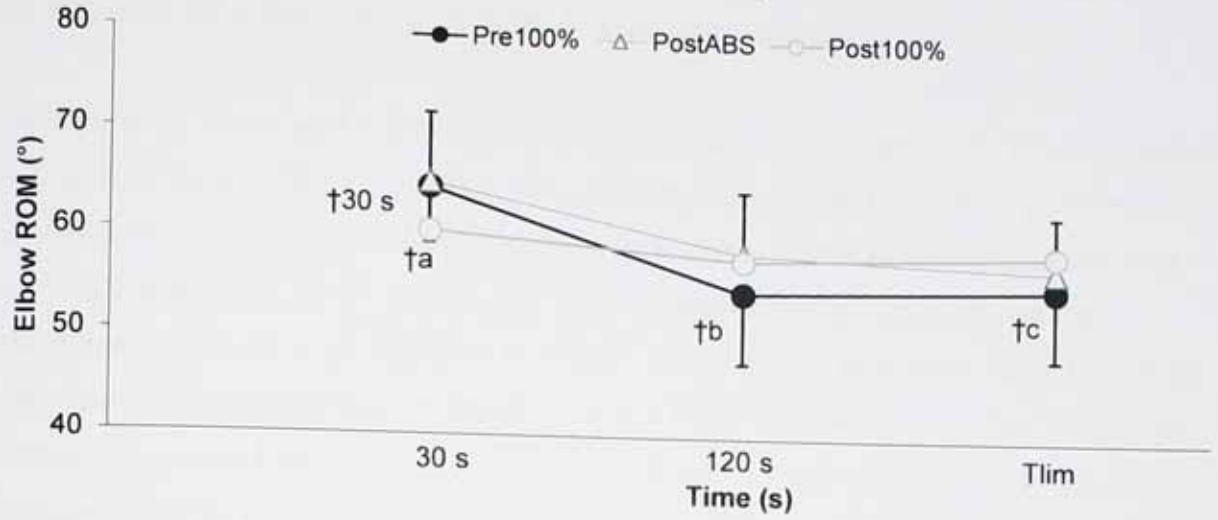


Figure 6.17 Changes in elbow ROM (°) before & after training against time (s).
$^{\dagger a}$. Significant difference ($P < 0.001$) from both $Pre_{100\%}$ and $Post_{ABS}$.
$^{\dagger b}$. Significant difference ($P < 0.001$) from both $Pre_{100\%}$ and $Post_{ABS}$.
$^{\dagger c}$. Significant difference ($P < 0.001$) from $Post_{100\%}$.
$^{\dagger 30\ s}$. Significant difference ($P < 0.001$) at 30 s from both 120 s and T_{lim}.

There was a significant interaction for $C7_D$ ($F = 3.990$, $P = 0.003$). Post-hoc analysis revealed a significant decrease in $C7_D$ at 120 s between $Post_{ABS}$ and $Post_{100\%}$ and a significant increase between $Post_{ABS}$ and $Post_{100\%}$ at T_{lim} (Figure 6.18).

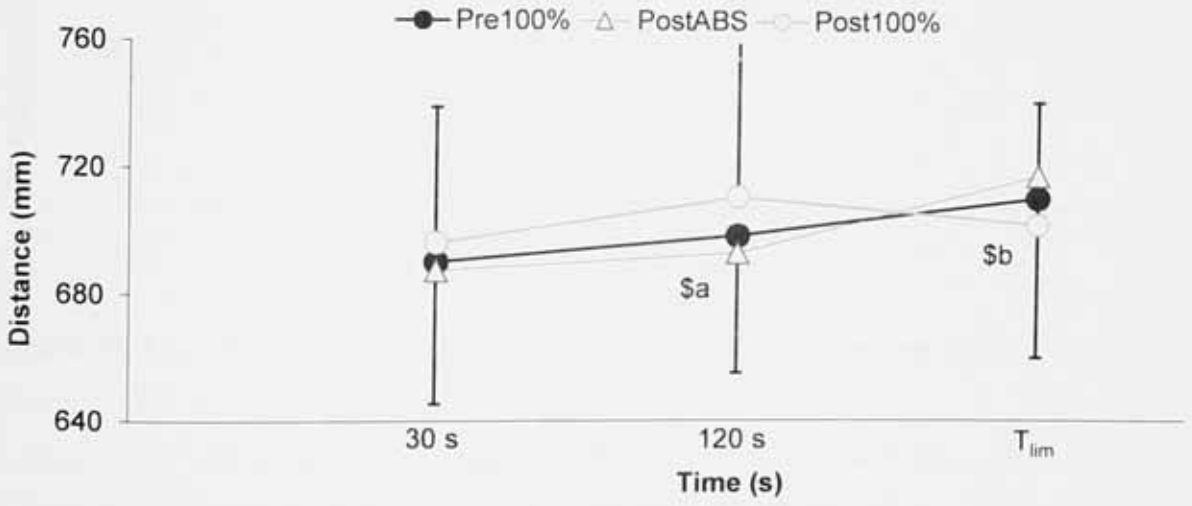


Figure 6.18 Changes in $C7_D$ before and after training against time (s).
$^{\$a}$. Significant difference ($P < 0.01$) from $Post_{100\%}$.
$^{\$b}$. Significant difference ($P < 0.01$) from $Post_{ABS}$.

There was significant interaction between time and trial for trunk rotational velocity ($F = 16.884$, $P < 0.001$). Post-hoc analysis revealed a significant decrease at 30 s for $Pre_{100\%}$ *vs* both $Post_{ABS}$ and $Post_{100\%}$.

Additionally, there was a significant main effect for time and trial ($F = 16.158$, $P < 0.001$; $F = 13.158$, $P < 0.001$, respectively; Figure 6.19). With post-hoc analysis revealing a significant increase in trunk rotational velocity at 30 s vs both 120 s and T_{lim} ($P = 0.001$, $P = 0.024$, respectively) and a significant decrease from 120 s *vs* T_{lim} ($P = 0.046$). Additionally, post-hoc testing of trial also revealed a significant increase in trunk rotational velocity from $Pre_{100\%}$ *vs* both $Post_{ABS}$ and $Post_{100\%}$ ($P = 0.007$, $P = 0.016$, respectively). The results suggest that as a response to both the critical work tests following training trunk rotational velocity was significantly greater early (30 s) in the exercise period. However, by T_{lim} the values converged and therefore training does not influence trunk rotational velocity at T_{lim} despite increases in time to exhaustion ($Post_{ABS}$) and exercise intensity ($Post_{100\%}$).

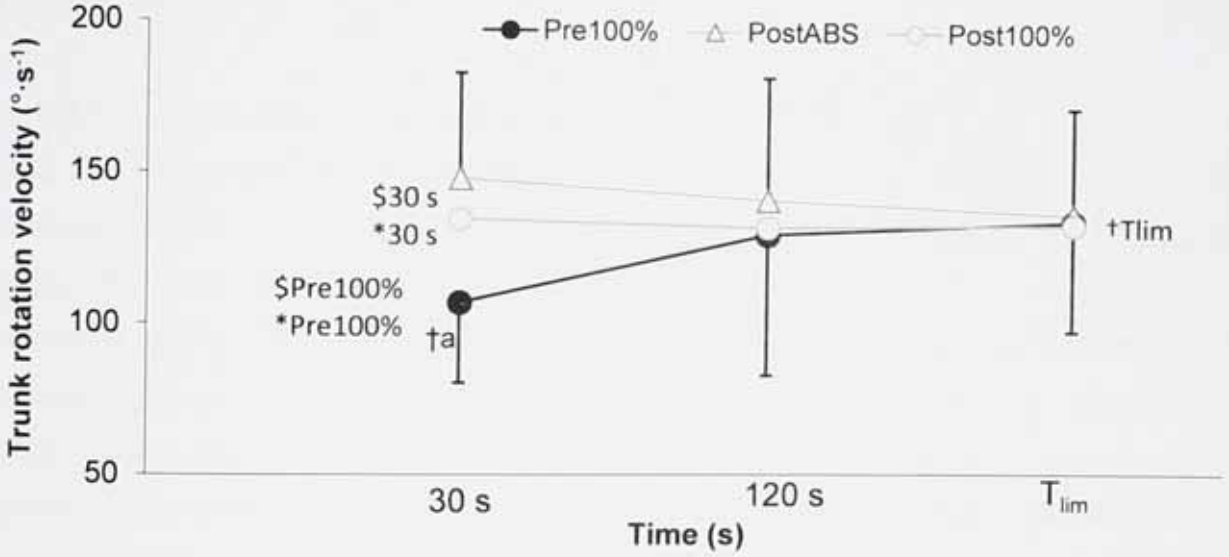


Figure 6.19. Changes in trunk rotation velocity ($°·s^{-1}$) before and after training against time (s).

^{†a}. Significant difference ($P < 0.001$) from both Pre_{ABS} and $Post_{ABS}$ 30 s.

$^{\$30\ s}$. Significant difference ($P < 0.01$) between 30 s and 120 s.

$^{*30\ s}$. Significant difference ($P < 0.05$) between 30 s and T_{lim}.

^{*Tlim}. Significant difference ($P < 0.05$) between 120 s and T_{lim}.

$^{\$Pre100\%}$. Significant difference ($P < 0.01$) between $Pre_{100\%}$ and $Post_{ABS}$.

$^{*Pre100\%}$. Significant difference ($P < 0.05$) between $Pre_{100\%\ and}$ $Post_{100\%}$.

6.4 Discussion

The primary aim of this study was to examine the effects of 6 weeks arm crank training on the physiological and biomechanical responses during high intensity upper body exercise. The main findings were that the training programme increased Wingate PPO, mean PO and cadence but not minimum PO. Changes in EMG during the WAnT were only observed for infraspinatus and anterior deltoid with a reduction in activation following training suggesting this may result in a concomitant increases in trunk rotational velocity at both corrected and uncorrected PPO. Therefore, peak power output improvements may be the result of changes in technique rather than muscle recruitment as there were few changes in EMG after training. Furthermore, peak aerobic power (PMP) improved despite no change in peak oxygen consumption. After training, time to exhaustion during the CWT increased and there was a reduced activation for triceps brachii at $Post_{abs}$ and $Post_{100\%}$. Additionally, there was a reduction in external oblique activation for the $Post_{ABS}$ intensity. Kinematic analysis indicated that during $Post_{100\%}$ the elbow joint angle, trunk distance to the ACE and trunk rotational velocity was held constant for the duration of the test compared to $Pre_{100\%}$ and $Post_{ABS}$.

6.4.1 Wingate anaerobic test

6.4.1.i Performance variables

The significant increases in peak power output (15% uncorrected PPO and 11.5% mean PO) were greater than reported for lower limb studies, which have reported increases of 7.0–12.0% post training involving purely sprint interval training (Burgomaster *et al.*, 2005, Barnett *et al.*, 2004, Hazell *et al.*, 2010). The performance training increases were greater than the daily variation observed in power output data (3%) for the WAnT as discussed in Section 3.3.1.i. Therefore, the results represented a meaningful increase in power output. The greater improvements observed in the present study suggest that the arms may be susceptible to greater training improvements than the legs possibly due to the relative inactivity of the upper body compared to the lower body in a young population (Marsh *et al.*, 1999, Casaburi *et al.*, 1992). In addition, training for this study was more varied than in the above studies. Subsequently, a cross-over effect from the interval and endurance training could have improved the aerobic component during the WAnT. Improvements in WAnT performance in previous studies have been partly attributed to increases in the

aerobic contribution to ATP resynthesis (Burgomaster *et al.*, 2008, Barnett *et al.*, 2004). For example, high intensity interval training has been shown to increase WAnT mean power output and time to peak power output (Ziemann *et al.*, 2011) by increasing the aerobic contribution to PO, and repeated sprints have been shown to increase mechanical efficiency by stimulating slow-twitch muscle fibres (Bangsbo, 1996). This response may also be enhanced in the upper body due to lower initial aerobic capacity.

Despite improvements in peak PO there were no increases in minimum PO resulting in the significant increases in the FI for uncorrected and corrected power. Previous lower body studies have shown either no change in FI (Hazell *et al.*, 2010, Ziemann *et al.*, 2011) or a reduced FI (Burgomaster *et al.*, 2006). This may be due to the training in these two studies utilising a short work effort of 10-30 s work with longer recoveries (2-4 min) (Burgomaster *et al.*, 2006, Hazell *et al.*, 2010) and the third 90 s work with 180 s recovery (Ziemann *et al.*, 2011). The training in the present study utilised shorter recovery periods for the interval training which may have enhanced PPO output but not the ability to sustain a sufficiently high power output for 30 s. Therefore, durations longer than 10 s and/or recovery for WAnT training may be required to significantly decrease the fatigue index.

The increase in uncorrected PPO is also reflected in an increase in peak cadence which was achieved in the same time as the pre training value. No increases in muscle girth (hypertrophy) were observed in the present study and the increase may be due to changes in neural function (section 6.4.1.ii). Although it is acknowledged that there is a link between contraction time and percentage of type II fibres (Mannion *et al.*, 1995) the training is unlikely, due to the 6-week duration, to have induced a change in fibre type distribution (Barnett *et al.*, 2004) or the activity of fast twitch (FT) fibre metabolism in enhancing performance (Ziemann *et al.*, 2011, Burgomaster *et al.*, 2005, Hazell *et al.*, 2010). However, the high intensity intervals may have increased glycogen depletion and enhanced FT recruitment (Krustrup *et al.*, 2004) seen as an increase in peak cadence within the same time as the pre training value; time to peak cadence was not reported by Burgomaster *et al.*, (2005), Hazell *et al.*, (2010) and Ziemann *et al.*, (2011) .

6.4.1.ii Electromyography responses

The lack of statistical significance within the EMG data indicated that there were no changes in muscle recruitment patterns, other than for a decrease in EMG activity of anterior deltoid and infraspinatus relating to peak cadence and therefore uncorrected PPO. This result may indicate that the improvement in uncorrected PPO observed in the present study was brought about by a change in muscle recruitment and/or technique. The significant reduction in EMG activity following training for the anterior deltoid, which assists in shoulder flexion (Mossberg *et al.*, 1999), could potentially have assisted the biceps brachii in increasing power generation by placing the biceps brachii in a more optimal position for power production (Murray *et al.*, 2000). Additionally, reduced activation of the anterior deltoid may have improved shoulder stabilisation (Ackland and Pandy, 2009) resulting in a change in technique to improve uncorrected PPO. The reduction in infraspinatus may also be connected to the reduction in EMG activity of the anterior deltoid as their activity is closely linked (Bressel and Heise, 2004). Whether these changes in activation for anterior deltoid and infraspinatus resulted in a more favourable position for activation of the biceps brachii and therefore increase flywheel cadence is not clear, although it is documented that muscle force changes due to its ROM and its relative position (Murray *et al.*, 2000). The EMG data were the first to indicate that upper body WAnT performance following training may be more affected by muscles of the shoulder than the muscles that contribute to power production the biceps and triceps brachii.

6.4.1.iii Kinematic analysis

The kinematic analysis of the WAnT indicates changes in technique post training. The elbow ROM at uncorrected PPO approached significance suggesting that reducing elbow joint angle may contribute to improvements in peak cadence (and therefore uncorrected PPO) potentially allowing for a faster push and pull phase of each crank revolution. Joint position had an effect on force production (Leedham and Dowling, 1995, Doheny *et al.*, 2008) and the change in joint angle may have contributed to an increase in PPO. The significant increase in trunk rotation velocity at corrected PPO suggested that faster trunk rotational velocity contributed to an increased acceleration of the flywheel, which is an important change in technique. Following training, trunk rotation velocity at PO_{min} (Figure 6.19) was the same as pre-training which indicates that the kinematic fatigue

point was the same. The EMG data showed a reduction in activity at uncorrected PPO, which along with kinematic analysis of elbow joint angle and trunk rotational velocity indicates that improvements in performance can be attributed to changes in technique and probable neurological improvements in muscle recruitment rather than change in muscle strength.

6.4.2 Incremental tests for peak oxygen uptake

Peak oxygen uptake values and peak heart rate pre training were similar to those reported in section 5.4.1. Contrary to the majority of previous studies that have reported increases in oxygen uptake post training in arm crank training (Pogliaghi *et al.*, 2006, Clausen *et al.*, 1973, Magel *et al.*, 1978, Gates *et al.*, 2003) and leg ergometry (Rosler *et al.*, 1985a) the present study observed no significant change in VO_{2peak} following training. Additionally, the change in VO_{2peak} was within the test-retest reliability observed in the general methods (Section 3.3.2.iii). Therefore, the Bonferroni correction is unlikely to be too conservative and a Type II error is unlikely to have occurred. However, this is most likely due to the high intensity/anaerobic nature of the training programme which did not affect aerobic enzymes, oxygen delivery or utilisation found in previous training studies. For example, following sprint interval training no changes were observed in VO_{2max} despite an increase in time to exhaustion at 80% VO_{2max} (Burgomaster *et al.*, 2005). As suggested in section 6.4.1.i there may have been a change in oxygen uptake kinetics leading to an improved aerobic contribution to the WAnT performance and therefore improved the WAnT performance and may be independent of changes in peak oxygen uptake (Invernizzi *et al.*, 2008). The intensity of the training programme may have been sufficient to cause changes in the fast phase of oxygen kinetics. Gas analysis during the WAnT would be needed to confirm if these changes were due to changes in oxygen uptake.

The increase in PMP post training was greater than the daily variation of ~10 W observed in the general methods (Section 3.3.2.iii). The significant increase in PMP (18 W) which may indicate greater mechanical efficiency/reduced energy cost of work and therefore a re-direction of cardiac output from auxiliary musculature reducing the VO_2 slow component which indicated that economy has improved (i.e. same VO_2 but at greater power output). Volianitis *et al.*, (2004b) observed that trained rowers had local changes in anaerobic/aerobic

metabolism when compared to untrained participants. Similarly, Rasmussen *et al.*, (1975) found arm crank training improved exercise performance and that local adaptations (e.g. variations in arterial blood such as reduced venous blood lactate content) were the main reason for a reduced ventilatory equivalent . Additionally, the sprint interval training is likely to have increased acidosis during training resulting in improvements in the anaerobic lactic metabolism (Billat, 2001a, Linossier *et al.*, 2011) and increasing tolerance to high intensity exercise.

There may have been an increase in psychological factors contributing (Lindsay *et al.*, 1996) to the improvement in incremental exercise test performance such that individuals have a greater tolerance to pain as a result of high plasma lactate levels and ischaemia (Katch and Henry, 1972, Westerblad *et al.*, 2002, Billat, 2001b). The subjective responses (RPE_L and RPE_{CR}) during interval training show that despite an increase in repetitions subjective responses were lower at training session 6 compared to session 1. This is most likely a training adaptation, training adaptations suggest a reduced HR with the same load (Rasmussen *et al.*, 1975, Franklin, 1985), but consideration should also be given that the participants have a greater tolerance of the discomfort of the activity having trained at their peak work capacity for 6-weeks (Westerblad *et al.*, 2002). The results indicate that VO_{2peak} and therefore oxygen delivery and utilisation were not necessarily limitations to performance in ACE i.e. that a higher $VO2_{peak}$ is not required for improvements in peak work capacity (Balady *et al.*, 1990). Future, studies examining ACE training should include an additional functional test other than a VO_{2peak} test such as a CWT as improvements in performance due to training may not always be reflected in a greater VO_{2peak}.

Typical representative data from two participants indicated that despite similar VO_{2peak} pre and post training there were differences in ventilatory and metabolic responses post training which are likely to have contributed to the increases in PMP despite no significant increase in VO_{2peak}. The VO_2 and breathing frequency responses showed a reduction for the same work load in the later half of the test (Figure 6.3 and Figure 6.4). This reduction was also for the same work load was also evident in the $V'E/V'O_2$ and $V'E/V'CO_2$ response (Figure 6.5 and Figure 6.6). Further analysis (not shown) indicates that the ventilatory threshold (calculated using the V-slope method) occurred later in the exercise period i.e. shifted to the right. Therefore, the metabolic exercise response in terms of oxygen uptake has

been reduced, at higher intensities, post training which has been suggested by previous researcher and suggests an improvement in economy (Edwards *et al.*, 2003, Jones, 1998, Burgomaster *et al.*, 2005).

6.4.3 Continuous work tests duration

6.4.3.i Time to exhaustion

The time to exhaustion at 100% PMP exercise intensity before and after training were within the values as reported in section 5.4.2. The peak heart rate and oxygen uptake indicate that the severe exercise domain occurred for all three loads (Caputo and Denadai, 2008, Xu and Rhodes, 1999). Following training the $Post_{ABS}$ workload showed a significantly increased time to exhaustion of 262 s and was considerably greater than the daily variation of 12 s at 80% PMP and 21 s at 110% PMP observed in the general methods (Section 3.3.3.ii). Even though one participant had a much greater T_{lim} increase than the other participant for $Post_{ABS}$ trial (1099 s) removal of this value still almost doubles the time to exhaustion during this trial. A similar response was observed by Burgomaster *et al* (2005) which also occurred at 80% of VO_{2peak} from sprint interval training. The exercise intensity for the $Post_{ABS}$ constant workload test represents 87.5% of the initial peak minute power achieved. The subsequent time to exhaustion is consistent with being between the 80 and 90% PMP exercise intensity observed in Chapter 5. However, the time to exhaustion following training at the new PMP was not significantly different from the pre training value and was less than the biological variation of 12 s observed for 110% PMP in the general methods (Section 3.3.3.ii). Therefore, it is unlikely the Bonferroni correction was too conservative and did not result in a type II error. As the same time to exhaustion was achieved for both 100% trials there may be a consistent time limit for performance at 100% PMP. This is the first ACE training study to report the changes in T_{lim} following training and is greater than T_{lim} increases of 32% at VO_{2max} found in runners (Esfarjani and Laursen, 2007) and 55% at VO_{2max} found in cyclists (McKay *et al.*, 2009).

6.4.3.ii Physiological responses

There were no significant differences observed in the $Pre_{100\%}$ and $Post_{100\%}$ oxygen uptake responses following training despite the $Post_{100\%}$ test being completed at a greater work load (18 W). Therefore, the $Post_{100\%}$ was completed at a greater workload but with the same oxygen uptake and a greater work load which

suggested an improved functional capacity (Figure 6.11). However, the lower VO_2 at 120 s for the $Post_{ABS}$ trial indicates a reduced cardiovascular load as HR was lower, as represented by the 87.5% PMP exercise intensity. There was a significant reduction in RER at 120 s during the $Post_{ABS}$ trial (now 87.5% of PMP). This response indicated that despite a high anaerobic component to exercise at this intensity there was a possible decrease in lactate acid production requiring less buffering and therefore reduced CO_2 production which was reflected in the reduced RER. Furthermore, RER was also lower during $Post_{ABS}$ at exhaustion in comparison to both $Pre_{100\%}$ and $Post_{100\%}$. This result confirmed a previous study observing a reduction in RER post-training following sprint cycle training (Burgomaster *et al.*, 2005) and indicated that training adaptations can improve submaximal and maximal PMP but not time to exhaustion at maximal PMP although PMP was greater.

Representative data from two typical participants indicated that despite a similar VO_{2peak} pre and post training for the PMP exercise intensities there were differences in ventilatory and metabolic responses post training. During the $Post_{ABS}$ test both participants exhibited a reduced VO_2 for the same absolute workload (Figure 6.10). There was also a marked reduction in breathing frequency during the early stages of exercise at this intensity. Together with $V'E/V'O_2$ (Figure 6.12) and $V'E/V'CO_2$ (Figure 6.13) data there was a marked shifted to the right the metabolic and ventilatory threshold (not shown). As previously noted this is a classic training response despite no increase in VO_{2peak} which is likely to have contributed to an increase the economy of exercise (Burgomaster *et al.*, 2005, Jones, 1998, Edwards *et al.*, 2003). In comparison, comparing $Pre_{100\%}$ and $Post_{100\%}$ (Figure 6.10) indicates that for participant 1 there was a reduction in VO_2 for a greater workload and for participant 2 there was a similar VO_2 response for the greater workload, and breathing frequency responses showed a similar response in both participants (Figure 6.11). The $V'E/V'O_2$ (Figure 6.12) and $V'E/V'CO_2$ (Figure 6.13) appear similar pre and post training for the $Pre_{100\%}$ and $Post_{100\%}$ exercise intensity. Therefore, ventilatory threshold (not shown) occurred at a similar time point but at a greater exercise intensity post training. The responses shown could be a function of the type of training undertaken by the participants as two-thirds of the training was completed at higher workloads which require a greater ATP turnover. The 'aerobic' training was completed more as a recover sessions than an 'aerobic'

training session. Therefore, the power output response could be greater as a response to the nature of the training programme.

The significant increase in $Post_{ABS}$ heart rate at exhaustion suggested that 6-weeks training may allow for sufficient time for HR to increase before a fatiguing end point is reached. As noted earlier, following training the original 100% PMP now represents 87.5% of PMP and elicited a similar time to exhaustion to the 90% PMP exercise intensity reported in section 5.3.2. This demonstrates the improved functional capacity and improved high intensity exercise performance following high intensity ACE training. The significant reduction in oxygen uptake at 120 s during the $Post_{ABS}$ test linked with a reduced RER would therefore indicate an increased aerobic component at this intensity. With no increased VO_{2peak} and changes in RER suggested an improvement in the economy of movement. Arm crank ergometry training can increase performance independent of an increased VO_{2peak}. Following training changes in $Post_{ABS}$ oxygen uptake, RER and HR reflect an improved functional capacity. Whilst $Post_{100\%}$ can be achieved with the same cardiorespiratory responses as before training but with a greater power output. Therefore, improved functional capacity and increased economy following training have improved ACE performance.

6.4.3.iii Electromyography responses

The EMG data presented here were the first to be reported in relation to performance tests before and after a period of ACE training In general muscular activity during the 100% PMP test to exhaustion did not change before and after training suggesting that training had a minimal effect on flexor carpi ulnaris, biceps brachii, infraspinatus and anterior deltoid activity patterns. In the present study local muscular fatigue was detected at T_{lim} for all muscles apart from the significant reduction following training for triceps brachii. Research by Hautier (2000) comparing trained to untrained cyclists completing a series of short (5-s) fatiguing sprints indicated that the trained cyclists reduced activation of their antagonist muscles to improve effective transfer of power, and cycle training was also found to decrease biceps femoris activity with no change in the rectus femoris (prime mover) (Ziemba *et al.*, 2003). The present study suggested that the biceps brachii has become more important in power production in this type of ACE, as suggested by (Smith *et al.*, 2008) with greater torque produced from

biceps brachii rather than triceps brachii. This is the first report of this pattern of activity following ACE training and although torque production was not measured it may answer questions raised by Smith *et al.*, (2008) and Bernasconi (2006) in that trained participants change their pattern of muscle activation and therefore torque production following training.

The significant difference for the external oblique at 120-s post training of the absolute load trial indicated the importance of this muscle to ACE performance. The longer exercise duration for $Post_{ABS}$ trial suggested reduced muscle activity and was an expected training adaptation. Support for this training effect is that there was almost identical EMG activation at 90% and 100% of PMP as shown in Chapter 5. The 90% PMP exercise intensity being close to the 87.5% of PMP exercise intensity that the $Post_{ABS}$ trial represents. Reduced activation of the external oblique at 120-s $Post_{ABS}$ may allow for a reduction in respiratory stress as indicated by a lower RPE_{CR} at this time point. There was a weak correlation between these two variables ($R^2 = 0.239$). A number of studies have shown that upper body exercise may impede respiratory function compared to lower body exercise due to differences in muscle afferents and the greater need to stabilise the trunk (Blasio *et al.*, 2009, Romagnoli *et al.*, 2006, Martin *et al.*, 1991, Ramonatxo, 1996). Reducing the external oblique activation may assist in reducing the impedance to respiratory function. Anecdotally a number of participants suggested that they felt 'more out-of-breath' during $Post_{ABS}$ following the training period indicating changes in breathing patterns might have taken place allowing a less restrictive effect. In addition, abdominal fatigue, which has been reported in cyclists completing a 90% PMP test to exhaustion (Taylor *et al.*, 2006) and the reduced activation shown in this thesis may be an adaptation to the training. A further study could evaluate changes in breathing frequency and tidal volume following training, as faster/slow cadences are known to increase/decrease respiratory drive (Price *et al.*, 2007), and the data suggested that respiratory drive could be influenced by relative load at the same cadence.

With no significant differences in EMG activity at exhaustion in the continuous work tests, muscle activation may have reached maximal levels or a fatiguing end point. The data generated using EMG responses is by its nature more variable (Murley *et al.*, 2010, Bigland-Ritchie, 1981). Analysis of mean amplitudes indicated there was not a great variability pre and post training. The

methods for the recording the pre and post test EMG signal were as robust as possible and within the scope of current EMG knowledge and analysis techniques available. Therefore, greater power output was achieved with little change in EMG activity. As there was a greater power output and no true meaningful change in EMG this indicates a shift in the muscle function resulting in an improvement in power output. Thus, indicating that the Bonferroni post-hoc correction was not too conservative. Future studies are recommend to investigate the contribution of the external oblique and other muscles of the trunk to ACE performance in relation to factors such as the strength of these muscles and their relative contribution to ACE performance. Previous research, not related to ACE, has concentrated on their contribution to general power and stability (Willardson, 2007, Akuthota, 2004) and whether reducing the activation of these muscles reduces respiratory load and improves performance and this is recommended to be investigated during ACE.

6.4.3.iv Kinematic analysis

This is the first study to report kinematic data before and after high intensity ACE training. Following training there were significant changes in kinematics when compared to pre training. The post training results for all three kinematic variables ($C7_D$, elbow joint angle ROM and trunk rotational velocity) showed that participants alter their movement pattern relatively little from 30 s to T_{lim} and therefore participants, pre training, alter their body position at 30 s to 120 s. The significantly lower HR at 30 s for $Post_{100\%}$ against both $Pre_{100\%}$ and $Post_{ABS}$ trials may be an indication that this movement pattern is the most efficient i.e. there is a greater power output for the same O_2 consumption. A previous study has shown that as cadence increases, trunk rotation decreases (Price *et al.*, 2007). This is in contrast to studies examining, upper and lower body exercise (So *et al.*, 2004) and lower body exercise suggesting that untrained participants increase their range of movement after the induction of fatigue (Strang *et al.*, 2009, James *et al.*, 2010). For the continuous work tests the post training kinematics differ significantly from the pre training responses at 30 and 120 s. The response pre training indicates that participants alter their body movement patterns during exercise more than when trained. Therefore, trained participants adopted a relatively unchanged position and technique until exhaustion. In Chapter 5 the four exercise intensities examined all demonstrated the same trunk rotation velocity at exhaustion. This shows that the same trunk rotation

velocity occurs in trained and untrained participants at the point of fatigue regardless of exercise intensity. Therefore, the current data indicated that once a given trunk rotational velocity has been achieved fatigue is likely to occur i.e. a biomechanical end point has been reached. Trunk rotation velocity may be one of a number of limiting factors to exercise or a determinant of fatigue in the population studied. This may be related to the same end-point being reached for EMG external oblique (Figure 6.16) at T_{lim} regardless of resistive load. Contrary to this, while some participants reported they were aware of using their abdominal muscles, no participants reported that they ceased the T_{lim} trials due to abdominal fatigue. Further studies on the interaction between trunk rotational velocity and muscles of the trunk are recommended.

6.4.4 Conclusion

The results of this study have shown that a 6-week arm crank training programme can improve performance measures during a 30-s WAnT, and during high intensity constant work load performance. The uniqueness of the study is that it draws on physiological and biomechanical measurement to suggest how these improvement in performance occurred. The representative participants' data indicated improvements in economy at higher exercise intensities. Future, research is required to examine closely the aerobic response and metabolic and ventilatory adaptations. Additional information provided by EMG and kinematics suggest that the improvements in performance were not necessarily solely connected to changes in metabolic factors. Unlike a number of previous ACE studies (Marais *et al.*, 2004, Smith *et al.*, 2008, Smith *et al.*, 2006c, Ahlborg and Jensen-Urstad, 1991, Koppo *et al.*, 2002) all CWT studies were continued to T_{lim} allowing for a full comparison of the time course of fatigue i.e. from start to fatigue end point (T_{lim}). For the Wingate anaerobic test there were improvements in PPO and MPO, EMG activity in the anterior deltoid and infraspinatus was reduced and there was an increase in trunk rotational velocity. In general, at T_{lim} for the continuous work test physiological, EMG and kinematic responses were the same at the point of T_{lim}. However, with $Post_{ABS}$ it takes greater time to get to the same point and with $Post_{100\%}$ a greater power output can be maintained. Regardless of load the same kinematic end point before and after training is reached. However, physiology, EMG activation and kinematics may change prior to reaching T_{lim}. Further research is required to analyse the physiological and kinematic responses across the time course of the activity and

within each duty cycle and any intra-individual responses i.e. is a greater ACE power output related to greater or lesser body movement and are there bilateral differences in responses. The results also suggest that training the biceps brachii and external obliques may improve ACE performance independent of specific ACE training - a further training study would be needed to confirm this.

Chapter 7

General discussion

7.0 Overview of studies

While arm crank ergometry (ACE) is not an area new to research, in comparison to leg ergometry it is still relatively underexplored. This is despite a large number of sports that require a majority or a significant component of performance from the upper body such as, sailing (Easton *et al.*, 2007, Neville *et al.*, 2009), kayaking (Billat *et al.*, 1996, Forbes and Chilibeck, 2007), swimming (Hawley *et al.*, 1992) and gymnastics (Jemini *et al.*, 2006) or the specific sport of hand cycling (Hopman *et al.*, 1995, Lovell *et al.*, 2011b, Verellen *et al.*, 2011). Additionally, the benefits of upper body exercise have been applied to health scenarios (Bulthuis *et al.*, 2010, Schrieks *et al.*, 2011, Westhoff *et al.*, 2008, Pogliaghi *et al.*, 2006, Tew *et al.*, 2009, Ilias *et al.*, 2009). Despite these benefits and applications, the majority of early research concentrated on comparisons of cycling ergometry to ACE (Reybrouck *et al.*, 1975, Vokac *et al.*, 1975). However, recent research has been more specific in analysing physiological responses to various exercise intensities and durations (Lovell *et al.*, 2011a, Castro *et al.*, 2010, van Drongelen *et al.*, 2009, Smith *et al.*, 2008, Lusina *et al.*, 2008, Smith *et al.*, 2007a).

Despite an increase in ACE research, a number of key areas have remained relatively unexplored. One such area is that of the fatigue responses to different resistive loads and load optimisation in maximal and high intensity exercise. If the mechanisms of fatigue at different intensities were better understood then there is scope to improve the performance outcomes of ACE whether it is for sport, exercise or health benefits. Therefore, this thesis sought to answer a number of important research questions in these areas. These questions regarding fatigue and optimal loads at maximal and high intensity exercise were investigated using a combination of physiological, electromyographical and kinematic analysis. The combination of which has only been considered in three previous studies (Smith *et al.*, 2008, Bressel and Heise, 2004, Price *et al.*, 2007).

Study 1 was the first study to report fatigue and the physiological and biomechanical response during a maximal upper body test using the Wingate

anaerobic test (WAnT). This study involved a variety of resistive loads to examine fatigue and sought to re-examine the optimal resistive load (uncorrected and corrected) in relation to fatigue for upper body WAnT performance. In this study, uncorrected peak power output increased with resistive load whereas corrected peak power output did not. The analysis of the EMG activity found that the biceps brachii distinguished between loads for peak power output. This indicated that the contribution of the biceps brachii to power production during the WAnT and its increased activation when required to accelerate the flywheel rapidly. Additionally, all the EMG sites, apart from the vastus medialis and lateral soleus, demonstrated increased activation at fatigue/minimum power, which indicated that as resistive load increased EMG activation also increased. Kinematic results were less conclusive although there were changes in torso distance in relation to the ergometer and changes in trunk rotational velocity which may assist in power production. This study was unique in that it combined the analysis of EMG and kinematic data in conjunction with performance across a range of resistive loads. Participants are advised to concentrate on flexion of the upper arm and use muscles of the trunk to aid power production and trunk rotational velocity. A 4% body mass resistive load represents a combination of power and cadence. Although, if individuals want to train for power then as the EMG activation was the greatest at the 5% resistive loading and therefore is suggested to result in the greatest recruitment of muscle fibres then this may be a more appropriate resistive load.

For consistency, in study 2, the same EMG and kinematic parameters were examined as for study 1. The addition of respiratory measures provided a further layer of analysis to examine fatigue during high intensity upper body exercise. Additionally, this study examined the optimal load for continuous high intensity exercise performance and suggested that an exercise intensity between 90% and 100% of peak minute power (PMP) is sufficient to achieve VO_{2peak}. The main findings were that at exhaustion oxygen uptake and heart rate were similar across exercise intensities and that not all the exercise intensities were in the severe exercise domain as VO_{2peak} was not always achieved. However, there may be differences in the degree of anaerobic metabolism as the respiratory exchange ratio data indicated that RER increased with exercise intensity at fatigue. Contrary to study 1, there was no distinguishing muscle in terms of EMG activity. Electromyographic activity increased over time, indicative of peripheral

fatigue, in all trials and was greater at the 100% and 110% VO_{2peak} intensity trials especially for the flexor carpi ulnaris, biceps and triceps brachii. The data for trunk rotational velocity indicated that rotational velocity rather than trunk stabilisation occurs as fatigue increases which was also found with increasing fatigue for the WAnT in study 1. Therefore, trunk rotational velocity may respond more to the effects of fatigue than to the resistive load or the exercise intensity.

The results from study 1 and 2 emphasised the important contribution of a combined analysis (physiology and biomechanics) in understanding fatigue during maximal and high intensity ACE. Fatigue was not just physiological but accompanied by changes in muscle activation and kinematics, and therefore technique. This combination of analysis enabled possible links between the measured variables to be suggested such as increases in EMG activation at greater exercise intensities which may result in recruiting greater type II muscle fibres (detected through an increase in RER). Highlighted throughout both studies, and in the previous literature (Bernasconi *et al.*, 2006, Smith *et al.*, 2008, Yasuda *et al.*, 2002), was the absence of data concerning the influence of training on the multi-faceted nature of fatigue. For example, physiological and metabolic improvement may be demonstrated after training but this may not account for all the improvement in performance (Loftin *et al.*, 1988, Magel *et al.*, 1978). Performance improvements could be a result of a change in technique reflected in changes in EMG activation and/or kinematic changes (Gabriel, 2002, Chapman *et al.*, 2009). The previous two studies indicated the importance of the biceps brachii to WAnT performance and the influence of trunk rotational velocity. Therefore, the aim of study 3 was to examine changes in performance, physiology and biomechanics before and after an upper body exercise training programme.

To date, links between training responses specific to arm crank ergometry and changes in fatigue responses to maximal and high intensity ACE to exhaustion have not been reported. Study 3 therefore involved a combination of ACE training methods utilising maximal sprints with no restrictions on cadence and constant load efforts with a constant cadence. The results showed significant increases in PPO, mean PO and cadence for the WAnT. Therefore, the training study was successful in improving maximal intensity ACE. Despite study 1

indicating the significance of the biceps brachii to WAnT performance the only significant changes in EMG were a reduction following training for the anterior deltoid and infraspinatus muscles. Changes in trunk rotational velocity at corrected and uncorrected PPO indicated technique improved and resulted in performance improvements over changes in EMG activation. Although there was no change in $VO_{2peak,}$ there was an increase in peak minute power (PMP) following training. For the high intensity exercise test to exhaustion (at 100% of PMP) following training there was a significant increase in time to exhaustion (T_{lim}). For the second high intensity exercise test to exhaustion at the new and greater PMP time to exhaustion was close to the pre training time to exhaustion. The EMG results for the triceps brachii activation indicated this was reduced for post absolute and post 100% PMP and there was also a reduction in activation for the external oblique at 120 s after training. Kinematic analysis indicated that at post 100% PMP that elbow joint angle, trunk distance to the ACE and trunk rotational velocity changes minimally during the test and indicates an improvement in performance through a more consistent movement pattern.

The first two studies highlighted the importance of biomechanical analysis in understanding the physiology of performance and fatigue. Study 3 confirmed that changes in technique (a combination of EMG and kinematics) following training contributed to improvements in performance. Interestingly at point of exhaustion during the high intensity exercise test to exhaustion, the kinematics were the same before and after training i.e. training has improved technique and performance prior to T_{lim} but not at the end of the test i.e. regardless of training status kinematic fatigue is the same at the end of the test.

7.1 Limitations

Despite a well developed method, during the course of investing a number of further methodological issues were raised and future studies should consider accounting for the following observations. The EMG data was a robust as possible for the systems and processes used. However, further development of the method to normalise EMG activity may assist the interpretation of the EMG signal, a suggestion would be to analyse the signal against a variety of loads rather than just 80% of peak minute power. Although due to the variability in EMG activity (Murley *et al.*, 2010) the normalisation method should be consistent across trials and in the studies in this thesis this was the best current

practical method available (Rouffet and Hautier, 2008, Albertus-Kajee *et al.*, 2010). The results for the vastus medialis and soleus (refer to section 4.3.3.) suggested that there were no differences in muscle activation between PPO and end PO during the WAnT. However, the high standard deviations may have contributed to the statistically non-significant results and biological variations may have contributed to this. This may have been due to the normalisation technique that was deemed reliable for the upper body but potentially not for lower body. Future studies could examine a different technique to normalise the lower body such as that from the peak EMG activity during the WAnT test (Rana, 2006). An analysis at set time points or crank duty cycles may establish changes in EMG with changes in power output and kinematics. The ability to establish specific duty cycles was not available with the Monark ergometer. This may have resulted in missing data in the first second(s) of the WAnT that could corroborate the results that the greater biceps EMG was a result of an increase in load to overcome the inertia of flywheel.

The training study used a combination of training sessions to provide a sufficient stimulus for a training response based on the previous body of published work (Ziemann *et al.*, 2011, Magel *et al.*, 1978, Loftin *et al.*, 1988, Billat *et al.*, 1999). As the training programme was focussed around high intensity exercise future training studies could concentrate on comparing aerobic and anaerobic programmes such as a 30 min aerobic effort against one of the interval training or short maximal sprints to analyse if there are differences in the training response. A longer training programme (greater than six weeks) would allow for monitoring of training responses during the training period and may give an indicating of when these responses occurred. Therefore, it may be possible to establish specific time points where performance improvements occur e.g. when changes in the WAnT occur and if this is before/after or at the same time as the changes in the high intensity exercise test to exhaustion and peak minute power and VO_{2peak}.

7.2 Future work

Although positive correlations have been found between Wingate ACE performance for a number of sports such as swimming (Hawley *et al.*, 1992), handball (Kounalakis *et al.*, 2008) and gymnastic (Jemini *et al.*, 2006), there may not be a link for swimming (Guglielmo and Denadai, 2000). Further

research could investigate ACE training as a means to improve specific sports performance e.g. whether improvements in Wingate power output translate to improvements in swimming performance. As a relationships between ACE performance and performance sport have been shown a future training study could use upper body exercise to investigate if ACE training can improve training in specific sports, e.g. it may help to offset the effects of fatigue in judo, or improve the power output in swimming. Also, where the role requires a significant contribution from the upper body ACE training my assist in this e.g. fire service (Gentzler and Stader, 2010, Eglin and Tipton, 2005), forestry (Kurumatani *et al.*, 1992) and rescue using a stretcher (Knapik *et al.*, 2000).

Although the aerobic contribution to lower body WAnT performance has been examined (Smith and Hill, 1991, Hill and Smith, 1993) this has not been investigated across a variety of loads for the upper body and future studies could examine whether the aerobic contribution differs between loads. If this could be established then it would also help to explain changes in Wingate power output found in this thesis following training. Additional studies could examine in closer detail the aerobic responses during the continuous work test.

Further studies examining fatigue could consider additional analysis of the EMG signal for changes in frequency and muscle fibre conduction velocity which would add to EMG changes observed in this thesis (Rainoldi *et al.*, 1999, Taylor *et al.*, 2000, Cifrek *et al.*, 2009, Stewart *et al.*, 2011) and changes to these parameters following training (Aagaard, 2003). This was not possible for the current study the crank arm position could not be recorded with a Monark ergometer and such a detailed study would require crank arm position to be aligned with the EMG signal and torque (Smith *et al.*, 2008). Torque production could be analysed using SRM power cranks (SRM, Jülich, Welldorf, Germany), or using a Lode ergometer (Groningen, Netherlands).

Training studies could examine the influence of cadence on performance outcome, e.g. does training with a greater resistive load and low cadence compare to training with a greater cadence and lower resistive load for WAnT performance. As there was an important contribution of the trunk to performance (EMG and kinematic), it would be useful to investigate if core stability training alone and/or in combination with standard ACE training could

improve ACE performance. Also, specific weight training may be investigated for improvement in ACE performance as a previous study observed greater oxygen uptake post weight training (Swensen *et al.*, 1993). This would inform the results of this thesis reporting whether cadence training affects performance or whether it is just the 'maximal' effort regardless of the cadence or type of muscle contraction. Weight training programmes would confirm whether the adaptations were cardiorespiratory based or restricted to muscle recruitment or hypertrophy.

In all three studies the population were male students and not upper body trained, this provided continuity in the data collection and in study 3 provided an opportunity to train these participants to become upper body trained. Future research should examine a broad range of participants such as females and trained upper body athletes (male and female, general and specific (hand cycling) upper body sports) thereby adding to the data for the optimal load and physiological and biomechanical response for a specifically trained population.

7.3 Practical application

The results of this thesis indicate the importance of combining power output, EMG and kinematic analysis in research studies to provide a comprehensive study of the effects of fatigue and alterations in fatigue and exercise performance following training. Previous studies have demonstrated that in persons with tetraplegia (Johnson *et al.*, 2004, Jacobs, 2003) the appropriate WAnT load is dependent on the level of spinal cord lesion. The results of this thesis continue to emphasise the importance of technique, trunk rotational velocity and activation of the external oblique to fatigue and greater exercise performance and where possible training and testing should include this when absolute power output (peak or sustained) is required. The effects of training in this study demonstrated how effective 6 weeks of ACE training can be, therefore, given that arm crank training can improve wheelchair propulsion (Dicarlo, 1988, Sedlock *et al.*, 1988), this type of training could successfully improve short and long duration wheelchair propulsion. The training may also help where short bursts of speed are required e.g. wheelchair basketball, tennis, rugby (Goosey-Tolfrey *et al.*, 2006). Although individuals without trunk rotation ability (i.e. high level spinal cord injury) may not be able to benefit as much or, more likely, improve through other mechanisms a combination of training intensities may further improve beneficial adaptations to their lipid profile that have been found in

previous studies and can increase VO_{2peak} and reduce fat mass (Dolbow *et al.*, 2010). Study 2 suggested that at 80% of PMP would be beneficial to training in these individuals as this intensity is high enough to enable physiological responses similar to higher intensities but with a lower RPE and heart rate prior to exhaustion. Additionally, a training study could use only anaerobic training of short duration and high intensities that has been demonstrated to be beneficial for time to exhaustion and maximal uptake following body training (Burgomaster *et al.*, 2005, Gibala *et al.*, 2006).

Participants that are not wheelchair users can improve their cardiac function (Billman, 2002, Zwierska *et al.*, 2005) through ACE training. However, these participants may have contraindications to maximal exercise testing (Yosefy *et al.*, 2006) and submaximal estimates can be reliably used (Birkett and Edwards, 1998, Abadie and Schuler, 1999) to predict VO_{2peak}. Therefore, the results of this thesis indicate that, with some modification, training used in study 3 could be appropriate to participants to produce rapid improvements in strength or power output which should translate into improved functional ability such as, walking (Zwierska *et al.*, 2005) and mobility in elderly patients after total hip arthroplasty (Grange *et al.*, 2004).

Previous studies have shown a relationship between ACE performance and sports performance (Hübner-Woźniak *et al.*, 2006a, Evans *et al.*, 1993, Jemini *et al.*, 2006, Hawley *et al.*, 1992, Volianitis *et al.*, 2004a) and has been used as a battery test criteria in volleyball (Driss *et al.*, 1998), climbers (Mermier, 2000), javelin throwers (Bouhlel *et al.*, 2007) and surfers (Mendez-Villanueva and Bishop, 2005). Therefore, for athletes in a number of sports where time, location, practically or injury does not permit specific training then ACE training is likely to be beneficial to performance in these sports. The training study has shown that a short period of ACE training can result in large increases in ACE performance which is likely to provide beneficial outcomes for performance in these sports and should be incorporated into training programmes.

Appendix 1

Terminology used to describe arm and/or leg ergometry undertaken for at least 5 s.

Author(s)	Arm (A) leg (L) ergometry	Duration	No. of intervals	Terminology
Max effort				
Balmer *et al.*, 2004	A	30 s	1	Maximal intensity
McGawley and Bishop, 2006	L	5 x 6-s		Maximal effort
Winter *et al.*, 1996	L	10 s		Maximal intensity
Winter, 1991	Review article	Review article – exercise that is performed all-out		Maximal intensity exercise
High intensity				
Artioli *et al.*, 2007	A	30 s	4	Short term, high intensity
Aschenbach *et al.*, 2000	A	8 x 15-s		High intensity
Balsom *et al.*, 1993	L		5 x 6-s	High intensity
Bell and Cobner, 2007	L	30	1	Short-term maximal intensity
Bernasconi *et al.*, 2006	A	40 % VT* & VO_{2peak}		Heavy arm-cranking
Fukuba *et al.*, 2002	A & L	50% LT to max VO_{2peak}		Heavy exercise
Greer *et al.*, 2006	L	30 s	1	Short term, high intensity

Hussain et al., 1996	L	30 s	1	High intensity
Kounalakis *et al.*, 2009	A	30 s	1	High intensity
Koppo *et al.*, 2002	A & L	90% VO_{2peak}	1	High intensity
Schneider *et al.*, 2002	A & L	½ AT* & VO_{2peak}	1	Heavy exercise
Souissi *et al.*, 2007	L	30	1	High-intensity
Smith *et al.*, 2006c	A	70%	1	Heavy arm-crank
Anaerobic power				
Artioli *et al.*, 2008	A	30 s	1	Anaerobic power
Bell and Cobner, 2010	L	30 s	1	Anaerobic power
Chtourou *et al.*, 2011	L	30 s	2	Anaerobic power
Creer *et al.*, 2004	L	4 x 30 s	1	Anaerobic power
Driss *et al.*, 1998	A & L	6 s		Maximal anaerobic power
Hawley and Williams, 1991	A	30 s	1	Anaerobic power
Hill and Smith, 1991	L	30 s	1	Anaerobic power
Horswill *et al.*, 1992	A	30 s	1	Anaerobic power
Hubner-Wozniak *et al.*, 2004	A & L	30	1	Anaerobic power
Johnson *et al.*, 2004	A	30	1	Anaerobic power
Jemini *et al.*, 2006	A & L	30 s	1	Anaerobic components of fitness
Kounalakis *et al.*, 2008	A	30 s	1	Anaerobic power
Mermier, 2000	A & L	30 s	1	Anaerobic capacity
Minahan *et al.*, 2007	L	30 s	1	Anaerobic power
Patton *et al.*, 1985	L	30 s	2	Anaerobic test

Rana, 2006	L	30 s	1	WAnT
Roberts *et al.*, 1991	A	30 s	1	Anaerobic power
Souissi *et al.*, 2007	L	30 s	1	Anaerobic power
Stewart *et al.*, 2011	L	30 s	1	Anaerobic test
Terbizan and Seljevold, 1996	A & L	30 s	1	Anaerobic capacity
Ucok *et al.*, 2008	L	30 s	1	Maximal anaerobic power & capacity
Vandewalle *et al.*, 1985a	L	45 s	1	Anaerobic capacity
Weber *et al.*, 2006	A & L	30 s	1	Anaerobic power
Zagatto *et al.*, 2008	A & L	30 s	1	Anaerobic work capacity
Supra-maximal				
Bangsbo, 1996	L	30 s	1	Supramaximal
Bar-Or *et al.*, 1980	L	30 s	1	Supramaximal
Bernardi *et al.*, 2007	A	1000 W	1	Supramaximal
Biggerstaff *et al.*, 1997	A	30 s	1	Supramaximal
Goulopoulou *et al.*, 2009	L	30 s	1	Supramaximal
Hermens *et al.*, 2000	L	30 s	1	Supramaximal
Hunter *et al.*, 2003	L	30 s	1	Supramaximal
Marais *et al.*, 1999	A	110% & 120% max power on $VO_{2\ peak}$	1	Supramaximal
Niewiadomski, 2007	L	30 s	2	Supramaximal
Bar-Or *et al.*, 1977	L	30 s	1	Supramaximal
Üçok *et al.*, 2005	L	30 s	1	Supramaximal

All-out/sprint				
Aziz *et al.*, 2002	A	10 & 30 s	1	All-out
Bogdanis *et al.*, 2008	L	7 x 6 s	1	Sprint exercise
Lutoslawska *et al.*, 2003	A & L	30 s	1 each	Supramaximal
Sagiv, 2005	L	30 s	1	All-out
Smith *et al.*, 2007b	A	20 s	1	Sprint
Vanderthommen *et al.*, 1997	A	3-6 s		All-out
Vandewalle *et al.*, 1985a	L	45 s	1	All-out
Weber *et al.*, 2006	A & L	30 s	1	All-out
Miscellaneous				
Evans *et al.*, 1993	A	30 s	1	Muscular power
Lericollais *et al.*, 2009	L	60 s	1	Sustained anaerobic
MacIntosh *et al.*, 2003	L	30 s	4	Short-term work capacity
Medbo and Tabata, 1989	L	30 s 1 min, 2-3 min	1	Short lasting
Smith and Hill, 1991	L	30 s	23	Wingate power test

Note: VT is ventilatory threshold.

Appendix 2
Ethics form

Graduate School Ethics
SCHOOL OF HEALTH RESEARCH ETHICS FORM

This document is to be used by all School of Health students or staff undertaking research. Students must submit this form with their research proposal as instructed by their lecturers. Staff needing approval for staff research need to submit it to Sheryl Munday, Thornby 2.

1. Project title: Physiological and biomechanical responses during high intensity upper body exercise

2. Course of study:
Staff - sport and exercise Graduate School study

3. Student number
Or if staff, name: Christopher Talbot
√ I have read and agree to adhere to the School of Health guidelines for conducting ethical research

4. Supervisors' names: N/A

5. Use of human participants: Tick one of the following:
√ I am using human participants.
I am using archival data where individuals are identifiable
I am **not** using human participants or data where individuals are identifiable and therefore do **not** need to complete the remainder of this form.

6. Participants: Tick the box which most accurately describes your sample:
Children under 16 years
16-18 year olds
Adults over 65 years old
NHS Patients
Social Care Clients

Health or Social Care Professionals
Members of the public (general)
√ Members of the public (specific such as professional athletes, teachers, – describe here: Adult students (aged 18 -40) at The University of Northampton
Members of vulnerable groups (frail elderly, disabled athletes, recently bereaved, members of support groups – describe here :.....)
Other. If other, describe your sample here:

7. Issues for concern: Tick below any issue that relates to this research.

Involves the use of human organs
Will be carried out on NHS or Social Services site
Will be conducted using NHS equipment
Involves invasive techniques (e.g. Taking of blood)
√ Involves participants undertaking tasks they would not normally undertake
Involves any activity that might be described as an 'invasion of privacy'
Involves deception
Involves a topic that would be considered 'sensitive'
Involves the collection of data that is not anonymised (contains identifying information such as name and address)
Requires participants to have a certain level of fitness.

√ Requires participants to be screened (e.g., a medical questionnaire) before acceptance into study
Other. If other, describe here:

8. Methodology: Tick the appropriate box. Full details of what you will do and where it will happen, should be provided in the accompanying Proposal.
Questionnaires
Interviews

√ Experiments
Observations
Archival
Other. If other, state here:

9. Recruitment Process. Tick the process that best describes how you plan to recruit participants. Full details of how you will recruit and where it will happen, should be provided in the accompanying Proposal.

√ Via poster in a public place such as a library or community centre
'Packs' will be provided to named person in an organisation/group to be distributed on my behalf
Asking personal contacts to pass my information packs to their contacts
Will be asking friends/family
Cold calling
Other. If other, state here:

10. Recruitment material. Tick all the recruitment material you will be using. You **must** use the School of Health templates to produce those. In addition, they **must not** be used until seen and approved by your supervisor.
√ Recruitment poster
Recruitment letter to named person in an organisation/group who will be distributing 'Packs' on your behalf
Recruitment letter to potential participants

√Participant Information Sheet

√ Consent form
NHS ethics application form
Other. If other, state here:

11. Risk assessment: Some projects will require risk assessment for participants and/or researchers. In other words, there is a possibility that participants and/or researchers will get hurt collecting data. If so, a risk assessment must be conducted. Tick the appropriate box below concerning your need for risk assessment.

There is **no** risk of injury to participants and/or researchers, so **no** risk assessment will be conducted.

√ There is a potential of injury to participants and/or researchers, so risk assessment has been (or will be) conducted.

A copy of the risk assessment has been seen and approved by the Supervisor

Part B To be completed by staff:

Comments:

Accepted with no amendments ☐ Accepted with minor amendments ☐

Accepted with major amendments ☐ Not accepted ☐

Proposal to be returned to Ethics Committee Yes [ ] No [ ]

Signed on behalf of Ethics Advisory Group

Date.

Resubmission:

Date to be submitted by:

Signature on behalf of Ethics committee Date.

Tick which of the following needs to be developed. **Supervisor to sign off once satisfied**

	Is needed	Final copy seen and approved by supervisor
Recruitment poster		√
Recruitment letter to individuals		√
Recruitment letter to organisation		√
Participant Information Sheet		√
Consent form		√
External application		√
Other:		

Consent Form (Example from Chapter 4)

For Participating in the Study of:
Load optimisation for arm crank ergometry during a 30-s Wingate test and analysis of biomechanics and physiological factors affecting performance

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

	Please tick the boxes Yes	No
I have read the study information sheet & understand what is involved.	☐	☐
I understand that the information I disclose will remain confidential and that my data will be destroyed or returned to me after being collated.	☐	☐
I understand that I can withdraw my participation at any time.	☐	☐
I am willing for my blood pressure to be recorded.	☐	☐
I am willing for my upper arm muscle volume & circumference to be measured.	☐	☐
I am willing for my muscle activity to be recorded.	☐	☐
I am willing for my upper body strength to be recorded during a maximal effort.	☐	☐
I am willing for my body movement to be tracked during arm cranking.	☐	☐
I would like to receive a summarised report of the study.	☐	☐
I am willing to participate in this project.	☐	☐

Signed: Date:

Participant information sheet
(Example from Chapter 4)

PARTICIPANT INFORMATION SHEET

About The Researcher:

I am a PhD student at the University of Northampton. I am researching maximal and high intensity upper body exercise. This research will form part of my PhD thesis. Professor Carol Phillips, at the University of Northampton, is supervising this study.

Study Title:

Load optimisation for arm crank ergometry during a 30-s Wingate test and analysis of biomechanics and physiological factors affecting performance

Aim of Study:

The aim of the study is to understand how fatigue affects maximal intensity 30-s duration arm cranking.

What the study involves:
You will need to visit the laboratory 5 times and complete 4 trials, with a minimum of 3 days between each visit. Each visit will last for no more than 1 hour and you will need to be in the laboratory within 1 hour of the time of your first visit (e.g. first visit at 11:00 o'clock, second and subsequent visit between 10:00 and 12:00 o'clock). The first visit will give you the chance to practice stationary arm cranking* and for non-cycling data to be collected. On visits 2, 3, 4 and 5 (1 trial each visit) you will be asked to arm crank as hard and as fast as you can for 30-seconds against 4 different resistances (1 resistance on each visit).

* arm cranking is pedalling a stationary bicycle using your arms

The information required:

On the first visit, resting blood pressure, maximal arm strength and girth will be measured. On visits 2, 3, 4 and 5, blood pressure (before and after exercise), electrical activity of muscle, and body movement using infra-red motion analysis will be recorded. All measurements are non-invasive. Please note, you will need to be aged between 18-40, have normal blood pressure and no cardiovascular conditions to take part in the study.

You may feel sick and dizzy after the test. However, with an active cool down these symptoms we soon pass. You may feel some muscle soreness, as would normally be expected after exercise.

Please be assured that you can decline participating at any time. In addition you are free to ask me any questions about the test procedure.

What will happen to the information?

The consent form and pre-test medical questionnaire completed prior to your visit, will be stored in a secure location and destroyed after 6 years. The identity of each participant (you) will remain anonymous throughout the research process and in the PhD thesis.
On completion of the data collection and dissertation, all data will either be destroyed or returned to the individual (you).

The information you disclose will be for my PhD research purposes only. It will not be given to any other party (e.g. your employer).

I will assign a number for your data and keep your data stored on a password protected PC. From then on you will be known only by your number. This will prevent anyone else from knowing your results.

Not sure about participating?

If you do not want to participate, that is okay, you have the right not to participate. You can also stop at any time if you do not want to finish the study; just let me know when you are ready to stop.

Your valued input:

I can make my results available to you when I have finished my study by sending you a short summary. Please let me know if you would like me to do this.

Contact the Researcher:

I hope the above information is helpful to you and gives you a better understanding and insight into my study. Please feel free to contact me at any time if you have any questions. **Chris Talbot, email chris.talbot@northampton.ac.uk**

Who has checked this research?

The Research Ethics Committee has approved this study.

The University of Northampton's Combined Liability Insurance Policy provides indemnity for students of the institution carrying out research work as part of their PhD.

Thank You

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

Post trial participant information

Post trial participant information

Thank you for taking part in this trial. Your next trial will take place:

on.....

time.....

Some things you should know after the trial:

You may feel some muscle soreness, as would normally be expected after exercise, in your upper body up to 72 hrs after the trial this is normal and to be expected.

If you have any health concerns please speak to your general practitioner (GP).

If you are unable to continue with the trials for whatever reason please let me know.

If you have any queries or require any further information please contact me on 01604 892479 or email chris.talbot@northampton.ac.uk.

Thanks

Chris Talbot
PhD student
The Knowledge Exchange

Participant information letter (Example from Chapter 4)

Everdon Building
Park Campus
Boughton Green Road
Northampton
NN2 7AL

Tel: 01604 892479

Dear

Research into maximal intensity upper body exercise

I am a PhD student at the University of Northampton. I am researching maximal intensity upper body exercise. This research will form part of my PhD thesis. Professor Carol Phillips, at the University of Northampton, is supervising this study.

This study involves arm cranking* for 30 seconds against a specific resistance. If you are interested in taking part in this study, and aged between 18 and 40 with no cardiovascular conditions, please see the attached participant information sheet, informed consent and pre-test medical questionnaire, which need to be completed at least 24 hours prior to your first visit. All testing will take place at The University of Northampton, Park Campus.

If you have any queries regarding the content of this letter or require any further information please contact me on 01604 892479 or email chris.talbot@northampton.ac.uk.

Thank you for your interest.

Chris Talbot
PhD student
The Knowledge Exchange

* arm cranking is pedalling a stationary bicycle using your arms

Strategy for dealing with physical problems or injuries that might occur during trials

Initial selection via information on the letter to possible participants, participant information sheet, pre-test medical questionnaire, blood pressure measurement and familiarisation trial should provide detailed screening. In the event of physical problems or injuries that might occur during a trial please see below.

The researcher is St John first aid (4 day first aid at work course) trained and always present when a participant is in the laboratory.

The most likely physical problem is dizziness. In the event of dizziness the following strategies will apply:

During the warm-up

The activity will stop and the participant will remain seated. If they continue to feel dizzy then they will be asked and/or assisted to lie on their back on a mat with their feet raised on a chair. When the dizziness has passed they will be asked to sit down and if feeling better to walk slowly around the laboratory. Assuming they feel well enough and their heart rate is back to a resting level they will be asked if they wish to leave.

During the trial

The trial will stop and the participant will remain seated. If able they will be asked to warm-down. If they continue to feel dizzy they will be asked and/or assisted to lie on their back on a mat with their feet raised on a chair. When the dizziness has passed they will be asked to sit down and of feeling better to walk slowly around the laboratory. Assuming they feel well enough and their heart rate is back to a resting level they will be asked if they wish to leave.

During the warm-down

If able they will be asked to continue the warm-down. If they continue to feel dizzy they will be asked and/or assisted to lie on their back on a mat with their feet raised on a chair. When the dizziness has passed they will be asked to sit

down and if feeling better to walk slowly around the laboratory. Assuming they feel well enough and their heart rate is back to a resting level they will be asked if they wish to leave.

Injuries

Due to the nature of the activity and pre-test screening it is unlikely that injuries will occur. If a participant is injured then the injury will be managed following the University of Northampton and St John first aider procedure.

If the injury occurs during the warm-up then warm-up will stop.

If the injury occurs during a trial or warm-down, if possible an alternative warm-down, to avoid dizziness, will be used, e.g. walking around the lab or cycling using the legs.

If a participant is unable to continue with the trial due to injury or illness etc then an incident form will be completed.

Strategy for dealing with physical problems or injuries that might after the trials

Injuries

Due to the nature of the activity and pre-test screening it is unlikely that injuries will occur. The participant may feel some soreness in the upper body especially the shoulders up to 72 hours after the trial.

A post-trail information sheet has been provided giving details of what to expect and what to do, this will be given out on completion of each trial.

If a participant is unable to continue with the study due to injury etc then an incident form will be completed.

Recruitment poster (Example from Chapter 4)
Research into upper body exercise

Participants needed

Are you: Male
Aged 18 – 40

The test: Would you be willing to participate in five maximal 30-second arm cranking* trials?

Each trial, including all measurements, will last no longer than 1 hour. Testing will take place in the Sport and Exercise Physiology Laboratory, Park Campus

Resting and post exercise blood pressure, upper body strength and body movement will be measured. All measurements are non-invasive.

What will I gain from the test?

Your resting blood pressure will be recorded

Your upper body strength will be recorded

You will know your maximal upper body power output

You will learn about research testing

Contact:
If you are interested contact, Chris Talbot (Technician – Sport and Exercise, Part-time Advanced Postgraduate), School of Health. chris.talbot@northampton.ac.uk

Everdon Building/Sports Hall
Everdon office 2 or Everdon Sport and Exercise Physiology Laboratory (Lab 1)
Tel: 01604 892479

* arm cranking is pedalling a stationary bicycle using your arms

Risk assessment

(General example)

THE UNIVERSITY OF NORTHAMPTON

Risk Assessment	
Activity: EXERCISE ON *MONARK* ERGOMETER CONVERTED TO AN ARM CRANK ERGOMETER	Reference No.:
	Assessed By: C. Talbot
Location: SPORT & EXERCISE PHYSIOLOGY LABORATORY (LAB 1) EVERDON BUILDING, PARK CAMPUS	Approved By:
	Issue Date: 01.01.2009
	Revision Date: 01.01.2010

Hazard	Consequences	Persons affected	Existing Control Measures (Where appropriate)	Severity	Likelihood	Total Risk	Additional Measures (Where appropriate)	Severity	Likelihood	Total Risk
ELECTRICAL										
1.5 V battery no know risks.										
GENERAL HAZARD										

Physical stress	Nausea, injury, illness, death	Participant	Screen participants, exclude participants at risk. Allow for familiarisation session prior to testing. A pretest consent form and medical questionnaire must be completed by the participants. Ensure participants are aware that the test can be stopped at any time for any reason. Ensure participants are aware of safest method of stopping the test. Ensure heart rate is monitored throughout the testing period. A warm-up and cool-down must be completed.	3	3	9					
Bike moving while in use	Injury	Staff, participants	Ensure the bike is firmly bolted to the table. Check bike after each test.	3	1	3					
Long hair and/or jewellery	Trapped in equipment	Participants	Hair tied back and jewellery and watches removed	3	1	3					
Arms too bent or locked out	Elbow, shoulder or head injury	Participants	With the hand firmly gripping the handle and the back straight, the elbow should be slightly bent with the handle at it furthest extension	2	2	4					
Slip	Bruising	Participants	Allow for familiarisation session prior to testing, and practice methods of stopping safely. Ensure hands are dry before gripping the handles and chair is firmly on the floor.	3	3	9					
FIRST AID											

Sweat, vomit	Contamination from sweat and/or vomit	Staff, students, agency staff, cleaners, contractors, visitors and participants	Bucket with disinfectant, paper towels to clean up vomit and sweat. Gloves & lab coat should be worn at all times. Advise participants not to consume a large meal within 3 hours of participating	3	3	9					
Risk of blood contaminating equipment	Contracting AIDS, Hepatitis B, or a blood bourne viral infection	Staff, students, agency staff, cleaners, contractors, visitors and participants	Screen participants, exclude participants at risk. Exclude any participant with obvious cuts on the hands or arms.	4	2	6					
COSHH											
Cleaning chemicals	Ill health effects, death	Staff, students, participants	Please refer to the COSHH/DSEAR Policy and Procedures and individual COSHH assessments for existing control measures. Cleaning chemicals, unless being used, must be kept in the cleaning cupboard.	3	3	9					
LOAD AND LIFTING											
Bulky and heavy	Back injury	Staff	Use wheels to move bike. When lifting bike use three people with two at the flywheel.	2	2	4					

RISK ASSESSMENT MATRIX

RISK						
Severity	5	5	10	15	20	25
	4	4	8	12	16	20
	3	3	6	9	12	15
	2	2	4	6	8	10
	1	1	2	3	4	5
		1	2	3	4	5
		Likelihood				

SEVERITY	
5	Multiple fatality
4	Fatality
3	Major injury
2	Minor injury
1	Negligible impact

LIKELIHOOD	
5	Almost Certain
4	Probable
3	Possible
2	Remote
1	Improbable

The aim is to reduce the risk by prevention or control measures so far as is reasonably practicable.

Explanatory Note:

Risk

16-25	Very high (Do not proceed without authorisation from the Directorate)
12-15	High
6-10	Medium
1-5	Low

Severity

Multi fatality	Self explanatory

Likelihood

Almost certain	Self explanatory
Probable	More likely than not to occur
Possible	Has the potential to occur
Remote	Unlikely to occur
Improbable	Occurrence is extremely unlikely

Fatality	Self explanatory
Major injury	Reportable incident under RIDDOR such as fracture of bones, dislocation, amputation, occupational diseases (e.g. asthma, dermatitis), loss of sight.
Minor injury	First aid administered. This would include minor, cuts, bruising, abrasions and strains or sprains of ligaments, tendons, muscles
Negligible impact	Self explanatory

Bibliography

Aagaard, P. (2003) Training-induced changes in neural function. *Exercise and Sport Science Reviews,* 31, 61-67.

Abadie, B. R. & Schuler, P. B. (1999) Estimation of arm maximal oxygen uptake (VO2max) from submaximal arm ergometer exercise in young male subjects. *Research in Sports Medicine: An International Journal,* 9, 15-23.

Ackland, D., C. & Pandy, M., G. (2009) Lines of action and stabilizing potential of the shoulder musculature. *Journal of Anatomy,* 215, 184-197.
Ahlborg, G. & Jensen-Urstad, M. (1991) Metabolism in exercising arm vs. leg muscle. *Clinical Physiology (Oxford, England),* 11, 459-468.

Akuthota, V. (2004) Core Strengthening. *Archives of Physical Medicine and Rehabilitation,* 85, S86-S92.

Al-Rahamneh, H. Q., Faulkner, J. A., Byrne, C. & Eston, R. G. (2010) Relationship Between Perceived Exertion and Physiologic Markers During Arm Exercise With Able-Bodied Participants and Participants With Poliomyelitis. *Archives of Physical Medicine & Rehabilitation,* 91, 273-277.

Albertus-Kajee, Y., Tucker, R., Derman, W. & Lambert, M. (2010) Alternative methods of normalising EMG during cycling. *Journal of Electromyography and Kinesiology,* 20, 1036-1043.

Amann, M. (2012) Significance of group III and IV muscle afferents for the endurance exercising human. *Clinical and Experimental Pharmacology and Physiology*. Ament, W. & Verkerke, G. J. (2009) Exercise and Fatigue. *Sports Medicine,* 39, 389-422.

Anders, C., Bretschneider, S., Bernsdorf, A. & Schneider, W. (2005) Activation characteristics of shoulder muscles during maximal and submaximal efforts. *European Journal Of Applied Physiology,* 93, 540-546.

Anderson, G. S. (1992) A Comparison of Predictive Tests of Aerobic Capacity. *Canadian Journal of Sport Science,* 17, 304-308.

Arslan, C. (2005) Relationship between the 30-second Wingate test and characteristics of isometric and explosive leg strength in young subjects. *Journal of Strength and Conditioning Research,* 19, 658-666.

Artioli, G. G., Gualano, B., Coelho, D. F., Benatti, F. B., Gailey, A. W. & Lancha Jr, A. H. (2007) Does Sodium-Bicarbonate Ingestion Improve Simulated Judo Performance? *International Journal of Sport Nutrition & Exercise Metabolism,* 17, 206-217.

Artioli, G. G., Gualano, B., Franchini, E., Bastista, R. N., Polacow, V. O. & Lancha Jr, A. H. L. (2008) Physiological, performance, and nutritional profile of the Brazilian olympic wushu (king-fu) team. *Journal of Strength & Conditioning Research,* 23.

Aschenbach, W., Ocel, J., Craft, L., Ward, C., Spangenburg, E. & Williams, J. (2000) Effect of oral sodium loading on high-intensity arm ergometry in college wrestlers. *Medicine & Science in Sports & Exercise,* 32, 669-675.

Astrand, P. O., Ekblom, B., Messin, R., Saltin, B. & Stenberg, J. (1965) Intra-arterial blood pressure during exercise with different muscle groups *Journal of Applied Physiology,* 20, 253-256.

Astrand, P. O. & Rodahl, K. (1986) *Textbook of Work Physiology, 3rd Ed.* New York: McGraw Hill International Editions.

Atkinson, G., Davison, R., Jeukendrup, A. & Passfield, L. (2003) Science and cycling: current knowledge and future directions for research. *Journal of Sports Sciences,* 21, 767-787.

Aziz, A. R., Lee, H. C. & Teh, K. C. (2002) Physiological characteristics of Singapore national water polo team players. *Journal of Sports Medicine & Physical Fitness,* 42, 315-319.

Baker, J., Gal, J., Davies, B., Bailey, D. & Morgan, R. (2001a) Power output of legs during high intensity cycle ergometry: Influence of hand grip. *Journal of Science and Medicine in Sport,* 4, 10-18.

Baker, J. S., Bailey, D. M. & Davies, B. (2001b) The relationship between total-body mass, fat-free mass and cycle ergometry power components during 20 seconds of maximal exercise. *Journal of Science and Medicine in Sport,* 4, 1-9.

Balady, G. J., Weiner, D. A., Rose, L. & Ryan, T. J. (1990) Physiological-responses to arm ergometry exercise relative to age and gender. *Journal of the American College of Cardiology,* 16, 130-135.

Balmer, J., Bird, S. R., Richard Davison, R. C., Doherty, M. & Smith, P. M. (2004) Mechanically braked Wingate powers: agreement between SRM, corrected and conventional methods of measurement. *Journal of Sports Sciences,* 22, 661-667.

Balsom, P. D., Soderlund, K., Sjodin, B. & Ekblom, B. (1993) Skeletal muscle metabolism during short duration high-intensity exercise influence of creatine supplementation *Acta Physiologica Scandinavica,* 154, 303-310.

Balter, J., E. & Zehr, E. P. (2006) Neural Coupling Between the Arms and Legs During Rhythmic Locomotor-Like Cycling Movement. *Journal of Neurophysiology,* 97, 1809-1818.

Bangsbo, J. (1996) Physiological factors associated with efficiency in high intensity exercise. *Sports Medicine,* 22, 299-305.

Bangsbo, J., Madsen, K., Kiens, B. & Richter, E. A. (1996) Effect of muscle acidity on muscle metabolism and fatigue during intense exercise in man. *Journal of Physiology,* 495, 587-596.

Bar-or, O. (1987) The Wingate Anaerobic Test. An update on methodology, reliability and validity. *Sports Medicine,* 4, 381-394.

Bar-or, O., Dotan, R. & Inbar, O. (1977) A 30-sec all-out ergometric test: its reliability and validity for anaerobic capacity. *Israel Journal of Medical Sciences,* 13, 326-327.

Bar-or, O., Dotan, R., Inbar, O., Rothstein, A. & Karlsson, J. (1980) Anaerobic capacity and muscle fiber type distribution in man. *International Journal of Sports Medicine,* 1, 82-85.

Bar-or, O. & Zwiren, L. D. (1975) Maximal oxygen consumption test during arm exercise reliability and validity. *Journal of Applied Physiology,* 38, 424-426.

Barfield, J.-P., Sells, P. D., Rowe, D. A. & Hannigan-Down, K. (2002) Practice Effect of the Wingate Anaerobic Test. *Journal of Strength and Conditioning Research,* 16, 472–473

Barnett, C., Carey, M., Proietto, J., Cerin, E., Febbraio, M. A. & Jenkins, D. (2004) Muscle metabolism during sprint exercise in man: influence of sprint training. *Journal of Science & Medicine in Sport,* 7, 314-322.

Baron, B., Moullan, F., Deruelle, F. & Noakes, T. D. (2011) The role of emotions on pacing strategies and performance in middle and long duration sport events. *British Journal of Sports Medicine,* 45, 511-517.

Barratt, P. R., Korff, T., Elmer, S. J. & Martin, J. C. (2011) The Effect of Crank Length on Joint-Specific Power during Maximal Cycling. *Medicine and Science in Sports and Exercise,* 43, 1689-1697.

Bassett, D. R. (1989) Correcting the Wingate test for changes in kinetic energy of the ergometer flywheel *International Journal of Sports Medicine,* 10, 446-449.

Bbc (2012) Sport Canoeing [online]. London: BBC. Available from http://www.bbc.co.uk/sport/0/canoeing/16432893 [Accessed 17th April 2012]. First ed. BBC.

Bediz, C. S., Goekbel, H., Kara, M., Uecok, K., Cikrikci, E. & Ergene, N. (1998) Comparison of the aerobic contributions to Wingate anaerobic tests performed with two different loads. *Journal of Sports Medicine & Physical Fitness,* 38, 30-34.

Bell, W. & Cobner, D. (2010) The Dynamics of Distance, Velocity and Acceleration of Power Output in the 30-s Wingate Anaerobic Test. *International Journal of Sports Medicine,* 32, 137-141.

Bell, W. & Cobner, D. M. (2007) Effect of individual time to peak power output on the expression of peak power output in the 30-s Wingate Anaerobic Test. International Journal of Sports Medicine, 28, 135-139.

Beneke, R., Pollmann, C., Bleif, I., Leithäuser, M. & Hütler, M. (2002) How anaerobic is the Wingate Anaerobic Test for humans? *European Journal of Applied Physiology,* 87, 388.

Bernardi, M., Quattrini, F. M., Rodio, A., Fontana, G., Madaffari, A., Brugnoli, M. & Marchetti, M. (2007) Physiological characteristics of America's Cup sailors. *Journal of Sports Sciences,* 25, 1141-1152.

Bernasconi, S., Tordi, N., Perrey, S., Parratte, B. & Monnier, G. (2006) Is the VO_2 slow component in heavy arm-cranking exercise associated with recruitment of type II muscle fibers as assessed by an increase in surface EMG? *Applied Physiology, Nutrition & Metabolism,* 31, 414-422.

Bhambhani, Y. N. (2004) Muscle Oxygenation Trends During Dynamic Exercise Measured by Near Infrared Spectroscopy. *Canadian Journal of Applied Physiology,* 29, 504-523.

Bhambhani, Y. N., Eriksson, P. & Gomes, P. S. (1991) Transfer effects of endurance training with the arms and legs. . *Medicine & Science in Sports & Exercise,* 23, 1035-1041.

Biggerstaff, K. D., Mcdonough, P., Moffatt, R. J. & Stamford, B. A. (1997) Effect of prior exercise on subsequent supramaximal arm ergometry: influence of maximal aerobic capacity (poster). *Medicine & Science in Sports & Exercise,* 29, S59.

Bigland-Ritchie, B. (1981) EMG/force relations and fatigue of human voluntary contractions. *Exercise And Sport Sciences Reviews,* 9, 75-117.

Billat, L. V. (2001a) Interval training for performance: a scientific and empirical practice: special recommendations for middle- and long-distance running. Part I: aerobic interval training. *Sports Medicine,* 31, 13-31.

Billat, L. V. (2001b) Interval training for performance: a scientific and empirical practice: special recommendations for middle- and long-distance running. Part II: anaerobic interval training. *Sports Medicine,* 31, 75-90.

Billat, V., Faina, M., Sardella, F., Marini, C., Fanton, F., Lupo, S., Faccini, P., De Angelis, M., Koralsztein, J. P. & Dalmonte, A. (1996) A comparison of time to exhaustion at VO_{2max} in elite cyclists, kayak paddlers, swimmers and runners. *Ergonomics,* 39, 267-277.

Billat, V. L., Flechet, B., Petit, B., Muriaux, G. & Koralsztein, J. P. (1999) Interval training at VO_{2max}: effects on aerobic performance and overtraining markers. *Medicine and Science in Sports and Exercise,* 31, 156-163.

Billman, G. E. (2002) Aerobic exercise conditioning: a nonpharmacological antiarrhythmic intervention. *Journal of Applied Physiology,* 92, 446-454.

Bird, S. & Davison, R. (1997) British Association of Sport and Exercise. Bird, S. & Davidon, R. (eds) Physiological Testing Guidelines, 3rd Ed. Leeds, UK: The British Association of Sport and Exercise Sciences.

Birkett, W. & Edwards, D. (1998) The use of one-arm crank ergometry in the prediction of upper body aerobic capacity. *Clinical Rehabilitation,* 12, 319-327.

Bishop, D., Bonetti, D. D. & Dawson, B. (2001) The effect of three different warm-up intensities on kayak ergometer performance. *Medicine & Science in Sports & Exercise,* 33, 1026-1032.

Blasio, A. D., Sablone, A., Civino, P., D'angelo, E., Gallina, S. & Ripari, P. (2009) Arm vs. combined leg and arm exercise: Blood pressure responses and ratings of perceived exertion at the same indirectly determined heart rate. *Journal of Sports Science & Medicine,* 8, 401-409.

Bogdanis, G., Papaspyrou, A., Lakomy, H. & Nevill, M. (2008) Effects of inertia correction and resistive load on fatigue during repeated sprints on a friction-loaded cycle ergometer. *Journal of Sports Sciences,* 26, 1437-1445.

Borg, G. (1998a) Borg's Perceived Exertion and Pain Sales. 1st Ed. ed. Champaign, IL Human Kinetics, Champaign, IL.

Borg, G. (1998b) *Borg's perceived exertion and pain scales* Human Kinetics: Champaign, Ill. Leeds: Human Kinetics.

Bouhlel, E., Chelly, M. S., Tabka, Z. & Shephard, R. (2007) Relationships between maximal anaerobic power of the arms and legs and javelin performance. *Journal of Sports Medicine & Physical Fitness,* 47, 141-146.

Bressel, E., Bressel, M., Marquez, M. & Heise, G. D. (2001) The effect of handgrip position on upper extremity neuromusuclar responses to arm cranking exercise. *Journal of Electromyography & Kinesiology,* 11, 291-298.

Bressel, E. & Heise, G. D. (2004) Effect of Arm Cranking Direction on EMG, Kinematic, and Oxygen Consumption Responses. *Journal of Applied Biomechanics,* 20, 129-143.

Brink-Elfegoun, T., Holmberg, H.-C., Ekblom, M. N. & Ekblom, B. (2007) Neuromuscular and circulatory adaptation during combined arm and leg exercise with different maximal work loads. *European Journal of Applied Physiology,* 101, 603-611.

Brooks, G. A., Fahey, T. D. & Baldwin, K. M. (2005) *Exercise physiology: human bioenergetics and its applications, 4th Ed.* Boston, MA: McGraw-Hill.

Brown, D., Knowlton, R., Hamill, J., Schneider, T. & Hetzler, R. (1990) Physiological and biomechanical differences between wheelchair-dependent and able-bodied subjects during wheelchair ergometry. *European Journal of Applied Physiology and Occupational Physiology,* 60, 179-182.

Bulthuis, Y., Drossaers-Bakker, W., Oosterveld, F., Van Der Palen, J. O. B. & Van De Laar, M. (2010) Arm crank ergometer is reliable and valid for measuring aerobic capacity during submaximal exercise. *Journal of Strength & Conditioning Research (Lippincott Williams & Wilkins),* 24, 2809-2815.

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

Burgomaster, K. A., Heigenhauser, G. J. F. & Gibala, M. J. (2006) Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *Journal of Applied Physiology,* 100**,** 2041-2047.

Burgomaster, K. A., Howarth, K. R., Phillips, S. M., Rakobowchuk, M., Macdonald, M. J., Mcgee, S. L. & Gibala, M. J. (2008) Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *The Journal of Physiology,* 586**,** 151-160.

Burgomaster, K. A., Hughes, S. C., Heigenhauser, G. J. F., Bradwell, S. N. & Gibala, M. J. (2005) Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *Journal of Applied Physiology,* 98**,** 1985-1990.

Buśko, K. (2011) Influence of two high-intensity intermittent training programmes on anaerobic capacity in humans. *Biology of Sport,* 28.

Calbet, J. A., Holmberg, H. C., Rosdahl, H., Van Hall, G., Jensen-Urstad, M. & Saltin, B. (2005) Why do arms extract less oxygen than legs during exercise? *American Journal Of Physiology. Regulatory, Integrative And Comparative Physiology,* 289**,** R1448-58.

Caputo, F. & Denadai, B. S. (2008) The highest intensity and the shortest duration permitting attainment of maximal oxygen uptake during cycling: effects of different methods and aerobic fitness level. *European Journal of Applied Physiology,* 103**,** 47-57.

Carolan, B. & Cafarelli, E. (1992) Adaptations in coactivation after isometric resistance training. *Journal of Applied Physiology,* 73**,** 911-917.

Carpenter, J. E., Blasier, R. B. & Pellizzon, G. G. (1998) The effects of muscle fatigue on shoulder joint position sense. *American Journal of Sports Medicine,* 26**,** 262-265.

Casaburi, R., Barstow, T. J., Robinson, T. & Wasserman, K. (1992) Dynamic and steady-state ventilatory and gas exchange responses to arm exercise. *Medicine & Science in Sports & Exercise,* 24, 1365-1374.

Castro, R. R. T., Pedrosa, S., Chabalgoity, F., Sousa, E. B. & Nobrega, A. C. L. (2010) The influence of a fast ramp rate on peak cardiopulmonary parameters during arm crank ergometry. *Clinical Physiology and Functional Imaging,* 30, 420-425.

Chapman, A., Vicenzino, B., Blanch, P. & Hodges, P. (2007) Leg muscle recruitment during cycling is less developed in triathletes than cyclists despite matched cycling training loads. *Experimental Brain Research,* 181, 503-518.

Chapman, A., Vicenzino, B., Blanch, P. & Hodges, P. (2009) Do differences in muscle recruitment between novice and elite cyclists reflect different movement patterns or less skilled muscle recruitment? *Journal of Science and Medicine in Sport,* 12, 31-34.

Chapman, A. R., Vicenzino, B., Blanch, P. & Hodges, P. W. (2008) Patterns of leg muscle recruitment vary between novice and highly trained cyclists. *Journal of Electromyography and Kinesiology* 18, 359–371.

Chapman, A. R., Vicenzino, B., Blanch, P., Knox, J. J. & Hodges, P. W. (2010) Intramuscular fine-wire electromyography during cycling: Repeatability, normalisation and a comparison to surface electromyography. *Journal of Electromyography and Kinesiology,* 20, 108-117.

Christine M. Adreani, Janeen M. Hill, A. & Kaufman, M. P. (1997) Responses of group III and IV muscle afferents to dynamic exercise. *Journal of Aplied Pysiologiy,* 82, 1811-1817.

Chtourou, H., Zarrouk, N., Chaouachi, A., Dogui, M., Behm, D. G., Chamari, K., Hug, F. O. & Souissi, N. (2011) Diurnal Variation in Wingate-Test Performance and Associated Electromyographic Parameters. *Chronobiology International: The Journal of Biological & Medical Rhythm Research,* 28, 706-713.

Cifrek, M., Medved, V., Tonkovic, S. & Ostojic, S. (2009) Surface EMG based muscle fatigue evaluation in biomechanics. *Clinical Biomechanics,* 24, 327-340.

Clarys, J. P. (2000) Electromyography in sports and occupational settings: an update of its limits and possibilities. *Ergonomics,* 43, 1750-1762.

Clausen, J. P., Klausen, K., Rasmussen, B. & Trap-Jensen, J. (1973) Central and peripheral circulatory changes after training of the arms or legs. *The American Journal of Physiology,* 225, 675-682.

Collins, M. H., Pearsall, D. J., Zavorsky, G. S., Bateni, H., Turcotte, R. A. & Montgomery, D. L. (2000) Acute effects of intense interval training on running mechanics *Journal of sports Sciences,* 18, 83-90.

Cooke, C. B. (1996) Maximal Oxygen Uptake, Economy and Efficiency. In: Eston, R. & Reilly, T. (eds). Kinathropometry and Exercise Physiologiy Laboratory Manual: Test, Procedures and Data. pp. 161-191, Second London: Routledge.

Corbett, J. (2009) Effect of task familiarisation on distribution of energy during a 2000 m cycling time trial. *British Journal of Sports Medicine,* 43, 770-774.

Coyle, E. F., Sidossis, L. S., Horowitz, J. F. & Beltz, J. D. (1992) Cycling efficiency is related to the percentage of Type I muscle fibers. *Medicine & Science in Sports & Exercise,* 24, 782-788.

Creer, A. R., Ricard, M. D., Conlee, R. K., Hoyt, G. L. & Parcell, A. C. (2004) Neural, Metabolic, and Performance Adaptations to Four Weeks of High Intensity Sprint-Interval Training in Trained Cyclists. *International Journal of Sports Medicine,* 25.

Dalsgaard, M. K., Volianitis, S., Yoshiga, C. C., Dawson, E. A. & Secher, N. H. (2004) Cerebral metabolism during upper and lower body exercise. *J Appl Physiol,* 97, 1733-1739.

Davies, C. J. & Sargeant, A. J. (1975) Effects of training on the physiological response to one- and two-leg work. *Journal of Applied Physiology,* 38, 377-381.

Davis, J. A., Vodak, P., Wilmore, J. H., Vodak, J. & Kurtz, P. (1976) Anaerobic threshold and maximal aerobic power for three modes of exercise. *Journal of Applied Physiology,* 41, 544-550.

Davis, J. M. (1995) Central and peripheral factors in fatigue *Journal of Sports Sciences,* 13, S49-S53.

De Groot, S., De Bruin, M., Noomen, S. P. & Van Der Woude, L. H. (2008) Mechanical efficiency and propulsion technique after 7 weeks of low-intensity wheelchair training. *Clinical Biomechanics (Bristol, Avon),* 23, 434-441.

Dicarlo, S. E. (1988) Effect of arm ergometry training on wheelchair propulsion endurance of individuals with quadriplegia. *Physical Therapy,* 68, 40-44.

Doheny, E. P., Lowery, M. M., Fitzpatrick, D. P. & O'malley, M. J. (2008) Effect of elbow joint angle on force-EMG relationships in human elbow flexor and extensor muscles. *Journal of Electromyography & Kinesiology,* 18, 760-770.

Dolbow, D. R., Miller, J., Harnish, C., Hunter, P., Ashrafa, G. & Gater, D. R. (2010) Arm Crank Exercise Increases VO_{2peak} and Reduces Body Fat Mass in Older Adult With Chronic Paraplegia. *Clinical Kinesiology (Online Edition).*

Dorel, S., Drouet, J.-M., Couturier, A., Champoux, Y. & Hug, F. O. (2009) Changes of Pedalling Technique and Muscle Coordination during an Exhaustive Exercise. *Medicine & Science in Sports & Exercise,* 41**,** 1277-1286.

Dotan, R. & Bar-or, O. (1983) Load optimization for the Wingate Anaerobic Test. *European Journal of Applied Physiology & Occupational Physiology,* 51**,** 409-417.

Driss, T., Vandewalle, H. & Monod, H. (1998) Maximal power and force-velocity relationships during cycling and cranking exercises in volleyball players: correlation with the vertical jump test. *Journal of Sports Medicine & Physical Fitness,* 38**,** 286-293.

Duchateau, J., Semmler, J. G. & Enoka, R. M. (2006) Training adaptations in the behaviour of human motor units. *Journal of Applied Physiology,* 101**,** 1766-1775.

Easton, C., Findlay, C., Morrison, G. & Spurway, N. C. (2007) Effects of dynamic upper-body exercise on lower-limb isometric endurance. *Journal of Sports Sciences,* 25**,** 1101-1107.

Ebaugh, D. D., Mcclure, P. W. & Karduna, A. R. (2006) Effects of shoulder muscle fatigue caused by repetitive overhead activities on scapulothoracic and glenohumeral kinematics. *Journal of Electromyography and Kinesiology,* 16**,** 224-235.

Edwards, A. M., Clark, N. & Macfadyen, A. M. (2003) Lactate and ventilatory thresholds reflect the training status of professional soccer

players where maximum aerobic power is unchanged. *Journal of Sports Science & Medicine,* 2, 23-29.

Eglin, C. M. & Tipton, M. J. (2005) Can firefighter instructors perform a simulated rescue after a live fire training exercise? *European Journal of Applied Physiology,* 95, 327-334.

El-Sayed, M. S. & Younesian, A. (2005) Lipid profiles are influenced by arm cranking exercise and training in individuals with spinal cord injury. *Spinal Cord: The Official Journal Of The International Medical Society Of Paraplegia,* 43, 299-305.

Enders, A. J., Hopman, M. & Binkhorst, R. A. (1994) The relation between upper arm dimensions and maximal oxygen uptake during arm exercise. *International Journal of Sports Medicine,* 15, 279-282.

Enoka, R. M. & Duchateau, J. (2008) Muscle fatigue: what, why and how it influences muscle function. *The Journal Of Physiology,* 586, 11-23.

Esfarjani, F. & Laursen, P. B. (2007) Manipulating high-intensity interval training: effects on VO_{2max}, the lactate threshold and 3000 m running performance in moderately trained males. *Journal of Science & Medicine in Sport,* 10, 27-35.

Eston, R. G. & Brodie, D. A. (1986) Responses to arm and leg ergometry. *British Journal of Sports Medicine,* 20, 4-6.

Evans, S. A., Eckerson, J. M., Housh, T. J. & Johnson, G. O. (1993) Muscular Power of the Arms in High School Wrestlers. *Pediatric Exercise Science,* 5, 72-77.

Fardy, P. S., Webb, D. & Hellerstein, H. K. (1977) Benefits of arm exercise in cardiac rehabilitation. *Physician & Sportsmedicine,* 5, 30-32.

Faupin, A., Gorce, P. & Meyer, C. (2011) Effects of type and mode of propulsion on hand-cycling biomechanics in nondisabled subjects. *Journal of Rehabilitation Research & Development,* 48**,** 1049-1059.

Fernandes, R. J., Keskinen, K. L., Colaã§O, P., Querido, A. J., Machado, L. J., Morais, P. A., Novais, D. Q., Marinho, D. A. & Vilas Boas, J. P. (2008a) Time limit at VO_{2max} velocity in elite crawl swimmers. *International Journal of Sports Medicine,* 29**,** 145-150.

Fernandes, R. J., Keskinen, K. L., Colaco, P., Querido, A. J., Machado, L. J., Morais, P. A., Novais, D. Q., Marinho, D. A. & Vilas Boas, J. P. (2008b) Time limit at VO_{2max} velocity in elite crawl swimmers. *International Journal of Sports Medicine,* 29**,** 145-150.

Field, A. (2009) Discovering Statistics using SPSS, 3rd Ed. London: Sage.

Fitts, R. H. (1996) Muscle fatigue: the cellular aspects. *American Journal of Sports Medicine,* 24**,** S9-S13.

Fitts, R. H. (2008) The cross-bridge cycle and skeletal muscle fatigue. *Journal of Applied Physiology,* 104**,** 551-558.

Forbes, S. C. & Chilibeck, P. D. (2007) Comparison of a kayaking ergometer protocol with and arm crank protocol for evaluating peak oxygen consumption. *Journal of Strength & Conditioning Research,* 21**,** 1282-1285.

Franchini, E., Takito, M. Y., Kiss, M. A. P. D. M. & Sterkowicz, S. (2005) Physcial Fitness and Anthropomtrical Differences between Elite and Non-Elite Judo Player. *Biology of Sport,* 22**,** 315-328.

Franklin, B. A. (1985) Exercise testing, training and arm ergometry. *Sports Medicine,* 2**,** 100-119.

Franklin, B. A. (1989) Aerobic exercise training programs for the upper body. *Medicine & Science in Sports & Exercise,* 21, S141-S148.

Franklin, B. A., Vander, L., Wrisley, D. & Rubenfire, M. (1994) Trainability of arms versus legs in men previous myocardial infarction. *Chest,* 105, 262-264.

Franklin, K. L., Gordon, R. S., Davies, B. & Baker, J. S. (2008) Assessing accuracy of measurements for a Wingate Test using the Taguchi method. *Research In Sports Medicine* 16, 1-14.

Frauendorf, H., Kobryn, U., Gelbrich, W. & Lange, T. (1989) Changes of the EMG and its relationship to the cardiopulmonary parameters during two-arm cranking of disabled men. *Biomedica Biochimica Acta,* 48, S521-S524.

Frauendorf, H., Kobryn, U., Hoffmann, B., Gelbrich, W., Ransch, E. & Erdmann, U. (1986) Sex and age-related behaviour of the integrated EMG during one-arm cranking. *Biomedica Biochimica Acta,* 45, S85-S87.

Fukuba, Y., Hayashi, N., Koga, S. & Yoshida, T. (2002) VO2 kinetics in heavy exercise is not altered by prior exercise with a different muscle group. *J Appl Physiol,* 92, 2467-2474.

Gabriel, D. A. (2002) Changes in kinematic and EMG variability while practicing a maximal performance task. *Journal of Electromyography and Kinesiology,* 12, 407-412.

Gastin, P. B. (2001) Energy system interaction and relative contribution during maximal exercise. *Sports Medicine,* 31, 725-741.

Gates, P. E., George, K. P. & Campbell, I. G. (2003) Concentric adaptation of the left ventricle in response to controlled upper body exercise training. *Journal of Applied Physiology,* 94, 549-554.

Geiser, C. F., O'connor, K. M. & Earl, J. E. (2010) Effects of Isolated Hip Abductor Fatigue on Frontal Plane Knee Mechanics. *Medicine & Science in Sports & Exercise,* 42, 535-545.

Gentzler, M. & Stader, S. (2010) Posture stress on firefighters and emergency medical technicians (EMTs) associated with repetitive reaching, bending, lifting, and pulling tasks. *Work,* 37, 227-239.

Gibala, M. J., Little, J. P., Van Essen, M., Wilkin, G. P., Burgomaster, K. A., Safdar, A., Raha, S. & Tarnopolsky, M. A. (2006) Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *The Journal Of Physiology,* 575, 901-911.

Giovani, D. & Nikolaidis, P. T. (2012) Differences in Force-velocity Characteristics of Upper and Lower Limbs of Non-competitive Male Boxers. *International Journal of Exercise Science,* 5, 106-113.

Girard, O., Mendez-Villanueva, A. & Bishop, D. (2011) Repeated-Sprint Ability – Part I: Factors Contributing to Fatigue. *Sports Medicine,* 41, 673-694.

Goosey-Tolfrey, V., Castle, P. & Webborn, N. (2006) Aerobic capacity and peak power output of elite quadriplegic games players. *British Journal of Sports Medicine,* 40, 684-687.

Goulopoulou, S., Fernhall, B. & Kanaley, J. A. (2009) Hemodynamic responses and linear and non-linear dynamics of cardiovascular autonomic regulation following supramaximal exercise. *European Journal of Applied Physiology,* 105, 525-531.

Graham, K. S. A. M. (1989) Variability of time to exhaustion and oxygen deficit in supramaximal exercise. *Australian Journal of Science and Medicine in Sport* 24, 11- 4.

Grange, C. C., Maire, J., Groslambert, A., Tordi, N., Dugue, B., Pernin, J. N. & Rouillon, J. D. (2004) Perceived exertion and rehabilitation with arm crank in elderly patients after total hip arthroplasty: a preliminary study. *Journal of Rehabilitation Research & Development,* 41, 611-619.

Grant, S., Craig, I., Wilson, J. & Aitchison, T. (1997) The relationship between 3 km running performance and selected physiological variables. *Journal of Sports Sciences,* 15, 403-410.

Green, H. J. (1997) Mechanisms of muscle fatigue in intense exercise. *Journal of Sports Sciences,* 15, 247-256.

Greer, F., Morales, J. & Coles, M. (2006) Wingate performance and surface EMG frequency variables are not affected by caffeine ingestion. *Applied Physiology, Nutrition & Metabolism,* 31, 597-603.

Guglielmo, L. G. A. & Denadai, B. S. (2000) Assessment of anaerobic power of swimmers: the correlation of laboratory tests on an arm ergometer with field tests in a swimming pool *Journal of Strength & Conditioning Research,* 14, 395-398.

Hautier, C. A., Arsac, L. M., Deghdegh, K., Souquet, J., Belli, A. & Lacour, J. R. (2000) Influence of fatigue on EMG/force ratio and cocontraction in cycling. *Medicine & Science in Sports & Exercise,* 32, 839-843.

Hawley, J. A. & Williams, M. M. (1991) Relationship between upper body anaerobic power and freestyle swimming performance. *International Journal of Sports Medicine,* 12, 1-5.

Hawley, J. A., Williams, M. M., Hamling, G. C. & Walsh, R. M. (1989) Effects of a task-specific warm-up on anaerobic power. *British Journal of Sports Medicine,* 23, 233-236.

Hawley, J. A., Williams, M. M., Vickovic, M. M. & Handcock, P. J. (1992) Muscle power predicts freestyle swimming performance. *British Journal of Sports Medicine,* 26**,** 151-155.

Hazell, T. J., Macpherson, R. E. K., Gravelle, B. M. R. & Lemon, P. W. R. (2010) 10 or 30-s sprint interval training bouts enhance both aerobic and anaerobic performance. *European Journal of Applied Physiology,* 110**,** 153-160.

Helge, J. W. (2010) Arm and leg substrate utilization and muscle adaptation after prolonged low-intensity training. *Acta Physiologica,* 199**,** 519-528.

Hermens, H. J., Freriks, B., Disselhorst-Klug, C. & Rau, G. (2000) Development of recommendations for SEMG sensors and sensor placement procedures. *Journal of Electromyography and Kinesiology,* 10**,** 361-374.

Herzog, W., Leonard, T. R., Joumaa, V. & Mehta, A. (2008) Mysteries of Muscle Contraction. *Journal of Applied Biomechanics,* 24**,** 1-13.
Hicks, A. L., Kent-Braun, J. & Ditor, D. S. (2001) Sex differences in human skeletal muscle fatigue. *Exercise & Sport Sciences Reviews,* 29**,** 109-112.

Hijikata, T., Wakisaka, H. & Niida, S. (1993) Functional combination of tapering profiles and overlapping arrangements in nonspanning skeletal muscle fibers terminating intrafascicularly. *The Anatomical Record,* 236**,** 602-610.

Hill, D. W., Poole, D. C. & Smith, J. C. (2002) The relationship between power and the time to achieve .VO(2max). *Medicine and Science in Sports and Exercise,* 34**,** 709-714.

Hill, D. W. & Rowell, A. L. (1996) Running velocity at VO_{2max}. *Medicine & Science in Sports and Exercise,* 28, 114-119.

Hill, D. W. & Smith, J. C. (1993) Gender differences in anaerobic capacity: role of aerobic contribution. *British Journal of Sports Medicine,* 27, 45-48.

Hill, D. W., Wiliams, S. E. & Burt, S. E. (1997) Repsonse to Exercise at 92% and 100% of the Velocity Associated with VO_{2max}. *International Journal of Sports Medicine,* 18, 325-329.

Hill, S. W. & Smith, J. C. (1991) Circadian rhythm in anaerobic power and capacity. *Canadian Journal of Sport Sciences,* 16, 30-32.

Hjeltnes, N. (1977) Oxygen uptake and cardiac output in graded arm exercise in paraplegics with low level spinal lesions. *Scandinavian Journal of Rehabilitation Medicine,* 9, 107-113.

Holmberg, H. C., Rosdahl, H. & Svedenhag, J. (2007) Lung function, arterial saturation and oxygen uptake in elite cross country skiers: influence of exercise mode. *Scandinavian Journal of Medicine & Science in Sports,* 17, 437-444.

Holmes, K. C. (1998) A Molecular Model for Muscle Contraction. *Acta crystallographica.*

Hooker, S. P. & Wells, C. L. (1991) Physiologic responses to arm crank exercise with and without hand grasping. *Clinical Kinesiology (Online Edition),* 45, 3-8.

Hopker, J. G., Coleman, D. A., Wiles, J. D. & Galbraith, A. (2009) Familiarisation and reliability of sprint test indices during laboratory and field assessment. *Journal of Sports Science and Medicine,* 8, 528-532.

Hopkins, W. G., Schabort, E. J. & Hawley, J. A. (2001) Reliability of power in physical performance tests. *Sports Medicine,* 31, 211-234.

Hopman, M. T. E., Van Teeffelen, W. M., Brouwer, J., Houtman, S. & Binkhorst, R. A. (1995) Physiological responses to asynchronous and synchronous arm-cranking exercise. *European Journal of Applied Physiology & Occupational Physiology,* 72, 111-114.

Horswill, C. A., Miller, J. E., Scott, J. R., Smith, C. M., Welk, G. & Van Handel, P. (1992) Anaerobic and aerobic power in arms and legs of elite senior wrestlers. *International Journal of Sports Medicine,* 13, 558-561.

Hsu, W.-L., Krishnamoorthy, V. & Scholz, J. P. (2006) An alternative test of electromyographic normalization in patients. *Muscle & Nerve,* 33, 232-241.

Hübner-Woźniak, E., Kosmol, A., Głaz, A. & Kusior, A. (2006a) The Evaluation of Upper Limb Muscles Anaerobic Performance of Elite Wrestlers and Boxers. *Research Yearbook,* 12, 218-221.

Hubner-Wozniak, E., Kosmol, A., Lutoslawska, G. & Bem, E. Z. (2004) Anaerobic performance of arms and legs in male and female free style wrestlers. *Journal of Science & Medicine in Sport,* 7, 473-480.

Hübner-Woźniak, E., Lutoslawska, G., Kosmol, A. & Zuziak, S. (2006b) The effect of training experience on arm muscle anaerobic performance in wrestlers. *Human Movement,* 7, 147-152.

Hug, F. & Dorel, S. (2009) Electromyographic analysis of pedaling: A review. *Journal of Electromyography and Kinesiology,* 19, 182-198.

Hunter, A. M., Lambert, M. I., Nobbs, L. & Noakes, T. D. (2003) Effects of supramaximal exercise on the electromyographic signal. *British Journal of Sports Medicine,* 37, 296-299.

Hussain, S. T., Smith, R. E., Medbak, S., Wood, R. F. & Whipp, B. J. (1996) Haemodynamic and metabolic responses of the lower limb after high intensity exercise in humans. *Experimental Physiology,* 81**,** 173-187.

Huxley, A. F. (2000) Cross-bridge action: present views, prospects, and unknowns. *Journal Of Biomechanics,* 33**,** 1189-1195.

Ilias, N. A., Xian, H., Inman, C. & Martin Iii, W. H. (2009) Arm exercise testing predicts clinical outcome. *American Heart Journal,* 157**,** 69-76.

Inbar, O., Bar-or, O. & Skinner, J. (1996) *The Wingate Anaerobic Test., 1st Ed.* Champaign, IL: Human Kinetics.

Invernizzi, P., Caporaso, G., Longo, S., Scurati, R. & Alberti, G. (2008) Correlations between upper limb oxygen kinetics and performance in elite swimmers. *Sport Sciences for Health,* 3**,** 19-25.

Ishida, K., Takaishi, T. & Miyamura, M. (1994) Ventilatory responses at the onset of passive movement and voluntary exercise with arms and legs. *Acta Physiologica Scandinavica,* 151**,** 343-352.

Jacobs, P. L., Johnson, B., Somarriba, G. A. & Carter, A. B. (2005) Reliability of upper extremity anaerobic power assessment in persons with tetraplegia. *The Journal Of Spinal Cord Medicine,* 28**,** 109-113.

Jacobs, P. L., Mahoney, E. T. & Johnson, B. (2003) Reliability of arm Wingate Anaerobic Testing in persons with complete paraplegia. *The Journal Of Spinal Cord Medicine,* 26**,** 141-144.

Jacobs, P. L. M., E. T. Johnson, B. M. (2003) Load determination for arm Wingate anaerobic testing in persons with tetraplegia. *Medicine & Science in Sports & Exercise,* 35.

James, C. R., Scheuermann, B. W. & Smith, M. P. (2010) Effects of two neuromuscular fatigue protocols on landing performance. *Journal of Electromyography & Kinesiology,* 20**,** 667-675.

James, D. V. B., Sandals, L. E., Wood, D. M. & Jones, A. M. (2007a) Pulmonary Gas Exchange. In: E. M. Winter., A. M. Jones., R. C. R. Davison., P. D. Bromley. & T. H. Mercer. (eds). BASES Sport and Exercise Physiology Testing Guidelines. pp. 101 - 111. 1st Ed. ed.

James, D. V. B., Wood, D. M., Maberly, T. C. B. & Croix, M. D. S. (2007b) Optimized versus corrected peak power during friction-braked cycle ergometry in males and females. *Journal of Sports Sciences,* 25**,** 859-867.

Jemini, M. M., Sands, W. A., Friemel, F. O., Stone, M. H. & Cooke, C. B. (2006) Any effect of gymnastics training on upper-body and lower-body aerobic and power components in national and international males gymnasts? *Journal of Strength & Conditioning Research,* 20**,** 899-907.

Jensen-Urstad, M., Hallback, I. & Sahlin, K. (1993) High anaerobic energy release during submaximal arm exercise. *Clinical Physiology (Oxford, England),* 13**,** 81-87.

Jensen-Urstad, M., Hallback, I. & Sahlin, K. (1995) Effect of hypoxia on muscle oxygenation and metabolism during arm exercise in humans. *Clinical Physiology,* 15**,** 27-37.

Jensen-Urstad, M. A. A., G. (1992) Is the high lactate release during arm exercise due to a low training status? *Clinical Physiology (Oxford, England),* 12**,** 487-496.

Johnson, B. M., Mahoney, E. T., Jacobs, P. L., Carter, A. B. & Somarriba, G. A. (2004) Effect of variable loading in the determination of upper-limb anaerobic power in persons with tetraplegia. *Journal of Rehabilitation Research & Development,* 41**,** 9-14.

Jones, A. M. (1998) A five year physiological case study of an Olympic runner. *British Journal of Sports Medicine,* 32, 39-43.

Kamen, G. & Gabriel, D. A. (2010) *Essentials of Electromyography, 1st Ed.* Champaign, III: Human Kinetics.

Kang, J., Chaloupka, E. C., Mastrangelo, M. A. & Angelucci, J. (1999) Physiological responses to upper body exercise on an arm and a modified leg ergometer. *Medicine & Science in Sports & Exercise,* 31, 1453-1459.

Kang, J., Chaloupka, E. C., Mastrangelo, M. A., Donnelly, M. S., Martz, W. P. & Robertson, R. J. (1998) Regulating exercise intensity using ratings of perceived exertion during arm and leg ergometry. *European Journal of Applied Physiology & Occupational Physiology,* 78, 241-246.

Kang, J., Robertson, R. J., Goss, F. L., Dasilva, S. G., Suminski, R. R., Utter, A. C., Zoeller, R. F. & Metz, K. F. (1997) Metabolic efficiency during arm and leg exercise at the same relative intensities. *Medicine & Science in Sports & Exercise,* 29, 377-382.

Karlsson, J., Bonde-Petersen, F., Henriksson, J. & Knuttgen, H. G. (1975) Effects of previous exercise with arms or legs on metabolism and performance in exhaustive exercise. *Journal of Applied Physiology,* 38, 763-767.

Katch, V. & Henry, F. M. (1972) Prediction of running performance from maximal oxygen debt and intake. *Medicine & Science in Sports,* 4, 187-191.

Kay, D., Frank, E. M., Cannon, J., St Clair-Gibson, A., Lambert, M. I. & Noakes, T. D. (2001) Evidence for neuromuscular fatigue during high-intensity cycling in warm, humid conditions. *European Journal of Applied Physiology,* 84, 115-121.

Kenney, L. W. (1995) ACSM's Guidline for Exercise Testing and Prescription, Willliams & Wilkin.

Kenney, L. W. (2005) ACSM's Guidline for Exercise Testing and Prescription, 7th Ed. Philadelphia: Willliams & Wilkin.

Kilen, A., Gizzi, L., Jensen, B. R., Farina, D. & Nordsborg, N. B. (2012) Changes in human muscle oxygen saturation and mean fiber conduction velocity during intense dynamic exercise-effect of muscular training status. *Muscle & Nerve,* 46, 746-754.

Klimstra, M. D., Thomas, E. & Paul Zehr, E. (2011) Biomechanical outcomes and neural correlates of cutaneous reflexes evoked during rhythmic arm cycling. *Journal Of Biomechanics,* 44, 802-809.

Knapik, J. J., Harper, W., Crowell, H. P., Leiter, K. & Mull, B. (2000) Standard and alternative methods of stretcher carriage: performance, human factors, and cardiorespiratory responses. *Ergonomics,* 43, 639-652.

Koga, S., Shiojiri, T., Shibasaki, M., Fukuba, Y., Fukuoka, Y. & Kondo, N. (1996) Kinetics of oxygen uptake and cardiac output at onset of arm exercise. *Respiration Physiology,* 103, 195-202.

Koppo, K. & Bouckaert, J. (2005) Prior Arm Exercise Speeds the VO_2 Kinetics during Arm Exercise above the Heart Level. *Medicine & Science in Sports & Exercise,* 37, 613-619.

Koppo, K., Bouckaert, J. & Jones, A. M. (2002) Oxygen uptake kinetics during high-intensity arm and leg exercise. *Respiratory Physiology & Neurobiology,* 133, 241-250.

Korge, P. (1995) Factors limiting adenosine triphosphatase function during high intensity exercise. Thermodynamic and regulatory considerations. *Sports Medicine,* 20**,** 215-225.

Kounalakis, S. N., Bayios, I. A., Koskolou, M. D. & Geladas, N. D. (2008) Anaerobic capacity of the upper arms in top-level team handball players. *International Journal of Sports Physiology and Performance,* 3**,** 251-261.

Kounalakis, S. N., Koskolou, M. D. & Geladas, N. D. (2009) Oxygen Saturation in the Triceps Brachii Muscle during an Arm Wingate Test: The Role of Training and Power Output. *Research in Sports Medicine,* 17**,** 171-181.

Krustrup, P., Soderlund, K., Mohr, M. & Bangsbo, J. (2004) Slow-twitch fiber glycogen depletion elevates moderate-exercise fast-twitch fiber activity and O_2 uptake. *Medicine & Science in Sports & Exercise,* 36**,** 973-982.

Kurumatani, N., Yamaguchi, B., Dejima, M., Enomoto, Y. & Moriyama, T. (1992) Aerobic capacity of forestry workers and physical demands of forestry operations. *European Journal of Applied Physiology and Occupational Physiology,* 64**,** 546-551.

Lake, M. J. & Cavanagh, P. R. (1996) Six weeks of training does not change running mechanics or improve running economy. *Medicine & Science in Sports & Exercise,* 28**,** 860-869.

Lakomy, H. K. (1985) Effect of load on corrected peak power output generated on friction loaded ergometers. *Journal of Sports Sciences,* 3**,** 240.

Lakomy, H. K. (1986) Measurement of work and power output using friction-loaded cycle ergometers. *Ergonomics,* 29**,** 509-517.

Lambert, M. I., Kolbe, T., Selley, E. A. & Dennis, S. C. (1995) The relationship between critical power and running performance. *Journal of Sports Sciences,* 13**,** 34-15.

Laurent, M. C., Meyers, M. C., Robinson, C. A. & Green, M. G. (2007) Cross-validation of the 20- versus 30-s Wingate anaerobic test *European Journal of Aplied Physiology,* 100**,** 645-651.

Lee, H.-M., Liau, J.-J., Cheng, C.-K., Tan, C.-M. & Shih, J.-T. (2003a) Evaluation of shoulder proprioception following muscle fatigue. *Clinical Biomechanics (Bristol, Avon),* 18**,** 843-847.

Lee, H.-M., Liau, J.-J., Cheng, C.-K., Tan, C.-M. & Shih, J.-T. (2003b) Evaluation of shoulder proprioception following muscle fatigue. *Clinical Biomechanics,* 18**,** 843-847.

Leedham, J. S. & Dowling, J. J. (1995) Force-length, torque-angle and EMG-joint angle relationships of the human in vivo biceps brachii. *European Journal of Applied Physiology and Occupational Physiology,* 70**,** 421-426.

Leicht, A. S., Sealey, R. M. & Sinclair, W. H. (2009) The Reliability of $VO_{(2peak)}$ Determination in Healthy Females during an Incremental Arm Ergometry Test. *International Journal of Sports Medicine,* 30**,** 509-515.

Leicht, A. S. & Spinks, W. L. (2007) Effect of shoulder angle on physiological responses during incremental peak arm crank ergometry. VO_{2peak} criteria. *Journal of Sports Sciences,* 25**,** 443-452.

Lepretre, P. M., Koralsztein, J. P. & Billat, V. L. (2004) Effect of exercise intensity on relationship between VO_{2max} and cardiac output. *Medicine & Science in Sports & Exercise,* 36**,** 1357-1363.

Lericollais, R., Gauthier, A., Bessot, N., Sesbastian, B. & Davenne, D. (2009) TIME-OF-DAY EFFECTS ON FATIGUE DURING A SUSTAINED ANAEROBIC TEST IN WELL-TRAINED CYCLISTS. *Chronobiology International: The Journal of Biological & Medical Rhythm Research,* 26, 1622-1635.

Leveque, J. M., Brisswalter, J., Bernard, O. & Goubault, C. (2002) Effect of paddling cadence on time to exhaustion and V_{O2} kinetics at the intensity associated with VO_{2max} in elite white-water kayakers. *Canadian Journal of Applied Physiology,* 27, 602-611.

Lindsay, F. H., Hawley, J. A., Myburgh, K. H., Schomer, H. H., Noakes, T. D. & Dennis, S. C. (1996) Improved athletic performance in highly trained cyclists after interval training. *Medicine and Science in Sports and Exercise,* 28, 1427-1434.

Linossier, M. T., Denis, C., Dormois, D., Geyssant, A. & Lacour, J. R. (2011) Ergometric and metabolic adaptation to a 5-s sprint training programme. . *European Journal of Applied Physiology and Occupational Physiology* 67, 408-14.

Linossier, M. T., Dormois, D., Fouquet, R., Geyssant, A. & Denis, C. (1996) Use of the force-velocity test to determine the optimal braking force for a sprint exercise on a friction-loaded cycle ergometer. *European Journal of Applied Physiology and Occupational Physiology,* 74, 420-427.

Loftin, M., Boileau, R. A., Massey, B. H. & Lohman, T. G. (1988) Effect of arm training on central and peripheral circulatory function. *Medicine & Science in Sports & Exercise,* 20, 136-141.

Lovell, D., Mason, D., Delphinus, E., Eagles, A., Shewring, S. & Mclellan, C. (2011a) Does Upper Body Strength and Power Influence Upper Body Wingate Performance in Men and Women? *International Journal of Sports Medicine,* 32, 771-775.

Lovell, D. I., Mason, D., Delphinus, E. & Mclellan, C. (2011b) A Comparison of Asynchronous and Synchronous Arm Cranking During the Wingate Test. *International Journal of Sports Physiology & Performance,* 6, 419-426.

Lusina, S. J., Warburton, D. E., Hatfield, N. G. & Sheel, A. W. (2008) Muscle deoxygenation of upper-limb muscles during progressive arm-cranking exercise. *Applied Physiology, Nutrition, and Metabolism* 33, 231-238.

Lutoslawska, G., Hübner-Wozniak, E. & Kosmol, A. (2003) Blood lactate response to 30 s arm cranking and leg cycling in elite wrestlers. *Medicina Sportiva,* 7, E69-E76.

Lyons, S., Richardson, M., Bishop, P., Smith, J., Heath, H. & Giesen, J. (2007) Excess post-exercise oxygen consumption in untrained men following exercise of equal energy expenditure: comparisons of upper and lower body exercise. *Diabetes, Obesity and Metabolism* 9, 889-894.

Macintosh, B. R., Rishaug, P. & Svedahl, K. (2003) Assessment of peak power and short-term work capacity. *European Journal of Applied Physiology,* 88, 572-579.

Magel, J. R., Foglia, G. F., Mcardle, W. D., Gutin, B. & Pechar, G. S. (1975) Specificity of swim training on maximum oxygen uptake. *Journal of Applied Physiology,* 38, 151-155.

Magel, J. R., Mcardle, W. D., Toner, M. & Delio, D. J. (1978) Metabolic and cardiovascular adjustment to arm training. *Journal of Applied Physiology: Respiratory, Environmental And Exercise Physiology,* 45, 75-79.

Mannion, A. F., Jakeman, P. M. & Willan, P. L. (1995) Skeletal muscle buffer value, fibre type distribution and high intensity exercise performance in man. *Experimental Physiology,* 80, 89-101.

Marais, G., Dupont, L., Garcin, M., Vanvelcenaher, J. & Pelayo, P. (2001) RPE responses during arm and leg exercises: effect of variations in spontaneously chosen crank rate. *Perceptual And Motor Skills 2001* 92, 253-62.

Marais, G., Dupont, L., Maillet, M., Weissland, T., Vanvelcenaher, J. & Pelayo, P. (2002a) Cardiorespiratory and efficiency responses during arm and leg exercises with spontaneously chosen crank and pedal rates. *Ergonomics,* 45, 631-639.

Marais, G., Dupont, L., Maillet, M., Weissland, T., Vanvelcenaher, J. & Pelayo, P. (2002b) Spontaneously chosen crank rate variations in submaximal arm exercise with inexperienced subjects. Effects on cardiorespiratory and efficiency parameters. *International Journal of Sports Medicine,* 23, 120-124.

Marais, G., Dupont, L., Vanvelcenaher, J., Clarys, J. P. & Pelayo, P. (2004) Effects of spontaneously chosen crank rate variations on electromyographic responses in sub-maximal arm exercise in inexperienced subjects. *European Journal of Applied Physiology,* 92, 598-601.

Marais, G., Weissland, T., Robin, H., Vanvelcenaher, J. M., Lavoie, J. M. & Pelayo, P. (1999) Physiological effects of variations in spontaneously chosen crank rate during sub-maximal and supra-maximal upper body exercise. *International Journal of Sports Medicine,* 20, 239-245.

Marino, F. E., Kay, D., Cannon, J., Serwach, N. & Hilder, M. (2002) A reproducible and variable intensity cycling performance protocol for warm conditions. *Journal of Science & Medicine in Sport,* 5, 95-107.

Marks, C. R. C., Hylland, K. E. & Terrell, J. (2012) Stability Ball Sitting versus Chair Sitting During Sub-maximal Arm Ergometry. *International Journal of Exercise Science,* 5, 16-25.

Marquardt, J., Bacharach, D. & Kelly, J. (1993) Predicting 30 second minimum power from 20 second Wingate test. *Medicine & Science in Sports & Exercise,* 25 (Sup), S109.

Marsh, G. D., Paterson, D. H., Govindasamy, D. & Cunningham, D. A. (1999) Anaerobic Power of the Arms and Legs of Young and Older Men. *Experimental Physiology,* 84, 589-597.

Martin, J. C., Wagner, B. M. & Coyle, E. F. (1997) Inertial-load method determines maximal cycling power in a single exercise bout. *Medicine & Science in Sports & Exercise,* 29, 1505-1512.

Martin, P. G., Smith, J. L., Butler, J. E., Gandevia, S. C. & Taylor, J. L. (2006) Fatigue-sensitive afferents inhibit extensor but not flexor motorneurons in humans. *Journal of Neuroscience,* 28, 4796-4802.

Martin, T. W., Zeballos, R. J. & M, W. I. (1992) Use of arm crank exercise in the detection of abnormal pulmonary gas exchange in patients at low altitude. *Chest,* 102 169-175

Martin, T. W., Zeballos, R. J. & Weisman, I. M. (1991) Gas exchange during maximal upper extremity exercise. *Chest,* 99, 420-425.

Mauger, A. R., Jones, A. M. & Williams, C. A. (2010) Influence of exercise variation on the retention of a pacing strategy. *European Journal Of Applied Physiology*.

Mcconnell, T. R. (1988) Practical Considerations in the Testing of VO_{2max} in Runners. *Sports Medicine,* 5, 57-68.

Mcgawley, K. & Bishop, D. (2006) Reliability of a 5 x 6-s maximal cycling repeated-sprint test in trained female team-sport athletes. *European Journal of Applied Physiology,* 98, 383-393.

Mckay, B. R., Paterson, D. H. & Kowalchuk, J. M. (2009) Effect of short-term high-intensity interval training vs. continuous training on O_2 uptake kinetics, muscle deoxygenation, and exercise performance. *Journal of Applied Physiology,* 107, 128-138.

Mclellan, T. M., Cheung, S. S. & Jacobs, I. (1995) Variability of Time to Exhaustion During Submaximal Exercise. *Canadian Journal of Applied Physiology-Revue Canadienne De Physiologie Appliquee,* 20, 39-51.

Mclester, J. R. (1997) Muscle contraction and fatigue: the role of adenosine 5'-diphosphate and inorganic phosphate. *Sports Medicine,* 23, 287-305.

Medbo, J. I., Gramvik, P. & Jebens, E. (1999) Aerobic and anaerobic energy release during 10 and 30 s bicycle sprints. *Acta Kinesiologiae Universitatis Tartuensis,* 4, 122-146.

Medbo, J. I. & Tabata, I. (1989) Relative importance of aerobic and anaerobic energy release during short-lasting exhausting bicycle exercise. *Journal of Applied Physiology,* 67, 1881-1886.

Mendez-Villanueva, A. & Bishop, D. (2005) Physiological Aspects of Surfboard Riding Performance. *Sports Medicine,* 35, 55-70.

Mercier, B., Granier, P., Mercier, J., Trouquet, J. & Prefaut, C. H. (1993) Anaerobic and aerobic components during arm-crank exercise in sprint and middle-distance swimmers. *European Journal of Applied Physiology & Occupational Physiology,* 66, 461-466.

Mermier, C. M. J., Jeffrey M; Parker, Daryl L; Swan, Jacob G (2000) Physiological and anthropometric determinants of sport climbing performance. *British Journal of Sports Medicine,* 34, 359-366.

Metter, E. J., Talbot, L. A., Schrager, M. & Conwit, R. A. (2004) Arm-cranking muscle power and arm isometric muscle strength are

independent predictors of all-cause mortality in men. (Abstract). *Scandinavian Journal of Medicine & Science in Sports,* 14, 203-203.

Micklewright, D., Alkhatib, A. & Beneke, R. (2006) Mechanically versus electro-magnetically braked cycle ergometer: performance and energy cost of the Wingate Anaerobic Test. *Eur J Appl Physiol,* 96, 748-751.

Midgley, A. W., Mcnaughton, L. R. & Carroll, S. (2007) Effect of the VO_2 time-averaging interval on the reproducibility of VO_{2max} in healthy athletic subjects. *Clinical Physiology & Functional Imaging,* 27, 122-125.

Miller, T. L., Mattacola, C. G. & Santiago, M. C. (2004) Influence of varied, controlled distances from the crank axis on peak physiological responses during arm crank ergometry. *Journal of Exercise Physiology Online,* 7, 61-67.

Millet, G. Y., Divert, C., Banizette, M. & Morin, J.-B. (2010) Changes in running pattern due to fatigue and cognitive load in orienteering. *Journal of Sports Sciences,* 28, 153-160.

Minahan, C., Chia, M. & Inbar, O. (2007) Does power indicate capacity? 30-s Wingate anaerobic test vs. maximal accumulated O_2 deficit. *International Journal of Sports Medicine,* 28, 836-843.

Moffatt, R. J., Chitwood, L. F. & Biggerstaff, K. D. (1994) The influence of verbal encouragement during assessment of maximal oxygen uptake. *Journal of Sports Medicine & Physical Fitness,* 34, 45-49.

Morgan, D. L. & Allen, D. G. (1999) Early events in stretch-induced muscle damage. *Journal of Applied Physiology,* 87, 2007-2015.

Morton, R. H. & Hodgson, D. J. (1996) The relationship between power output and endurance: a brief review. . *European Journal of Applied Physiology & Occupational Physiology,* 73, 491-502.

Mossberg, K., Willman, C., Topor, M. A., Crook, H. & Patak, S. (1999) Comparison of asynchronous versus synchronous arm crank ergometry. *Spinal Cord,* 37, 569.

Mukherjee, G., Bhowmik, P. & Samanta, A. (2001) Physical fitness training for wheelchair ambulation by the arm crank propulsion technique. *Clinical Rehabilitation,* 15, 125-132.

Muraki, S., Tsunawake, N. & Yamasaki, M. (2004) Limitation of muscle deoxygenation in the triceps during incremental arm cranking in women. *European Journal of Applied Physiology,* 91, 246-252.

Murley, G. S., Menz, H. B., Landorf, K. B. & Bird, A. R. (2010) Reliability of lower limb electromyography during overground walking: A comparison of maximal- and sub-maximal normalisation techniques. *Journal of Biomechanics,* 43, 749-756.

Murray, W. M., Buchanan, T. S. & Delp, S. L. (2000) The isometric functional capacity of muscles that cross the elbow. *Journal of Biomechanics,* 33, 943-952.

Mygind, E. (1995) Fibre characteristics and enzyme levels of arm and leg muscles in elite cross-country skiers. *Scandinavian Journal of Medicine & Science in Sports,* 5, 76-80.

Neville, V., Pain, M. T. G. & Folland, J. P. (2009) Aerobic power and peak power of elite America's Cup sailors. *European Journal of Applied Physiology,* 106, 149-157.

Niewiadomski, W., Gąsiorowska, A., Krauss, B., Mróz, a and Cybulski, G (2007) Suppression of heart rate variability after supramaximal exertion. *Clinical Physiology and Functional Imaging,* 27, 309 - 319.

Nindl, B. C., Mahar, M. T., Harman, E. A. & Patton, J. F. (1995) Lower and upper body anaerobic performance in male and female adolescent athletes. *Medicine and Science in Sports and Exercise,* 27, 235-241.

Ogata, H. & Yano, T. (2005) Kinetics of oxygen uptake during arm cranking with the legs inactive or exercising at moderate intensities. *European Journal of Applied Physiology,* 94, 17-24.

Ortiz, A., Olson, S. L., Etnyre, B., Trudelle-Jackson, E. E., Bartlett, W. & Venegas-Rios, H. L. (2010) Fatigue efffects on knee joint stability during two jump taks in women. *Journal of Strength & Conditioning Research* 24, 1019-1027.

Pandolf, K. B., Billings, D. S., Drolet, L. L., Pimental, N. A. & Sawka, M. N. (1984) Differentiated ratings of perceived exertion and various physiological responses during prolonged upper and lower body exercise *European Journal of Applied Physiology and Occupational Physiology* 53, 5-11.

Patton, J. F., Murphy, M. M. & Frederick, F. A. (1985) Maximal power outputs during the Wingate Anaerobic Test. *International Journal of Sports Medicine,* 6, 82-85.

Pendergast, D., Cerretelli, P. & Rennie, D. W. (1979) Aerobic and glycolytic metabolism in arm exercise. *Journal Of Applied Physiology: Respiratory, Environmental And Exercise Physiology,* 47, 754-760.

Pendergast, D. R. (1989) Cardiovascular, respiratory, and metabolic responses to upper body exercise. *Medicine & Science in Sports & Exercise,* 21, S121-S125.

Pimental, N. A., Sawka, M. N., Billings, D. S. & Trad, L. A. (1984) Physiological responses to prolonged upperbody exercise. *Medicine & Science in Sports & Exercise,* 16, 360-365.

Pogliaghi, S., Terziotti, P., Cevese, A., Balestreri, F. & Schena, F. (2006) Adaptations to endurance training in the healthy elderly: arm cranking versus leg cycling. *European Journal of Applied Physiology,* 97, 723-731.

Powers, S. K., Beadle, R. E. & Mangum, M. (1984) Exercise efficiency during arm ergometry: effects of speed and work rate. *Journal of Applied Physiology,* 56, 495-499.

Price, M. J. & Campbell, I. G. (1997) Determination of peak oxygen uptake during upper body exercise. *Ergonomics,* 40, 491-499.

Price, M. J. & Campbell, I. G. (2002) Thermoregulatory responses during prolonged upper body exercise cool and warm conditions. *Journal of Sports Sciences,* 20, 519-527.

Price, M. J. & Campbell, J. G. (1998) Thermoregulatory and physiological responses of wheelchair athletes to prolonged arm crank ergometry and wheelchair ergometry. . *Journal of Sports Sciences,* 16, 57-57.

Price, M. J., Collins, L., Smith, P. M. & Goss-Sampson, M. (2007) The effects of cadence and power output upon physiological and biomechanical responses to incremental arm-crank ergometry. *Applied Physiology, Nutrition & Metabolism,* 32, 686-692.

Rainoldi, A., Galardi, G., Maderna, L., Comi, G., Lo Conte, L. & Merletti, R. (1999) Repeatability of surface EMG variables during voluntary isometric contractions of the biceps brachii muscle. *Journal of Electromyography and Kinesiology,* 9, 105-119.

Ramonatxo, M. P., J. Prefaut, C. (1996) Differences in mouth occlusion pressure and breathing pattern between arm and leg incremental exercise. *Acta Physiologica Scandinavica,* 158, 333-341.

Rana, S. R. (2006) Effect of the Wingate test on mechanomyography and electromyography. *Journal of Strength & Conditioning Research,* 20, 292-297.

Rasmussen, B., Klausen, K., Clausen, J. P. & Trap-Jensen, J. (1975) Pulmonary ventilation, blood gases, and blood pH after training of the arms or the legs. *Journal of Applied Physiology,* 38, 250-256.

Rayment, I., Holden, H. M., Whittaker, M., Yohn, C. B., Lorenz, M., Holmes, K. C. & Milligan, R. A. (1993) Structure of the actin-myosin complex and its implications for muscle contraction. *Science (New York, N.Y.),* 261, 58-65.

Reybrouck, T., Heigenhauser, G. F. & Faulkner, J. A. (1975) Limitations to maximum oxygen uptake in arm and leg, and combined arm-leg ergometry. *Journal of Applied Physiology,* 38, 774-779.

Roberts, A. J., Termin, B., Reilly, M. F. & Pendergast, D. R. (1991) Effectiveness of Biokinetic Training on Swimming Peformance in Collegiate Swimmers. *Journal of Swimming Research,* 7, 5-11.

Roland Van Den, T. & Mario, M. (2011) Effect of training on the kinematics and performance in overarm throwing in experienced female handball players. *Portuguese Journal of Sport Sciences,* 11, 125-128.

Romagnoli, I., Gorini, M., Gigliotti, F., Bianchi, R., Lanini, B., Grazzini, M., Stendardi, L. & Scano, G. (2006) Chest wall kinematics, respiratory muscle action and dyspnoea during arm vs. leg exercise in humans. *Acta Physiologica (Oxford, England),* 188, 63-73.

Rosler, K., Hoppeler, H., Conley, K. E., Claassen, H., Gehr, P. & Howald, H. (1985a) Transfer effects in endurance exercise. Adaptations in trained and untrained muscles. *European Journal of Applied Physiology and Occupational Physiology,* 54, 355-362.

Rosler, K., Hoppeler, H., Conley, K. E., Claassen, H., Gehr, P. & Howald, H. (1985b) Transfer effects in endurance exercise. Adaptations in trained and untrained muscles. *European Journal of Applied Physiology,* 54, 355-362.

Rouffet, D. M. & Hautier, C. A. (2008) EMG normalization to study muscle activation in cycling. *Journal of Electromyography & Kinesiology,* 18, 866-878.

Rudroff, T., Barry, B. K., Stone, A. L., Barry, C. J. & Enoka, R. M. (2007) Accessory muscle activity contributes to the variation in time to task failure for different arm posture and loads. *Journal of Applied Physiologiy,* 102, 1000-1006.

Sagiv, M. B.-S., D, Sagiv, M. And Goldhammer, E. (2005) Left Ventricular Function at Peak All-Out Anaerobic Exercise in Older Men. *Gerontology* 51.

Sahlin, K. (1992) Metabolic factors in fatigue. *Sports Medicine,* 13, 99-107.

Sahlin, K., Tonkonogi, M. & Soderlund, K. (1998) Energy supply and muscle fatigue in humans. *Acta Physiologica Scandinavica,* 162, 261-266.

Sandbakk, Ø., Holmberg, H.-C., Leirdal, S. & Ettema, G. (2010) Metabolic rate and gross efficiency at high work rates in world class and national level sprint skiers. *European Journal of Applied Physiology* 109, 473-81.

Santos, E. L., Novaes, J. S., Reis, V. M. & Giannella-Neto, A. (2010) Low sampling rates bias outcomes from the Wingate test. *International Journal of Sports Medicine,* 31, 784-789.

Sargeant, A. J. (1994) Human power output and muscle fatigue. *International Journal of Sports Medicine,* 15, 116-121.

Sargeant, A. J., Hoinville, E. & Young, A. (1981) Maximum leg force and power output during short-term dynamic exercise. *Journal of Applied Physiology,* 51, 1175-1182.

Savard, G., Kiens, B. & Saltin, B. (1987) Central cardiovascular factors as limits to endurance; with a note on the distinction between maximal oxygen uptake and endurance fitness. IN MACLEOD, D., MAUGHAN, R., NIMMO, M., REILLY, T. & WILLIAMS, J. (Eds.) *Exercise, Benefits, Limits and Adaptations.* E. & F. N. Spon Ltd.

Sawka, M. N. (1986) Physiology of upper body exercise. *Exercise & Sport Sciences Reviews,* 14, 175-211.

Sawka, M. N., Foley, M. E., Pimental, N. A., Toner, M. M. & Pandolf, K. B. (1983) Determination of maximal aerobic power during upper-body exercise. *Journal Of Applied Physiology: Respiratory, Environmental and Exercise Physiology,* 54, 113-117.

Schabort, E. J., Hawley, J. A., Hopkins, W. G. & Blum, H. (1999) High reliability of performance of well-trained rowers on a rowing ergometer. *Journal of Sports Sciences,* 17, 627-632.

Schneider, D. A., Wing, A. N. & Morris, N. R. (2002) Oxygen uptake and heart rate kinetics during heavy exercise: a comparison between arm cranking and leg cycling. *European Journal of Applied Physiology,* 88, 100-106.

Schneider, S., Brümmer, V., Abel, T., Askew, C. D. & Strüder, H. K. (2009) Changes in brain cortical activity measured by EEG are related to individual exercise preferences. *Physiology & Behavior,* 98, 447-452.

Schrieks, I. C., Barnes, M. J. & Hodges, L. D. (2011) Comparison study of treadmill versus arm ergometry. *Clinical Physiology & Functional Imaging,* 31, 326-331.

Sealey, R. M., Spinks, W. L., Leicht, A. S. & Sinclair, W. H. (2010) Identification and reliability of pacing strategies in outrigger canoeing ergometry. *Journal of Science and Medicine in Sport,* 13.

Secher, N. H., Ruberg-Larsen, N., Binkhorst, R. A. & Bonde-Petersen, F. (1974) Maximal oxygen uptake during arm cranking and combined arm plus leg exercise. *Journal of Applied Physiology,* 36, 515-518.

Sedlock, D., Knowlton, R. & Fitzgerald, P. (1988) The effects of arm crank training on the physiological responses to silbmaximal wheelchair ergometry. *European Journal of Applied Physiology and Occupational Physiology,* 57, 55-59.

Sedlock, D. A. (1991) Postexercise energy expenditure following upper body exercise. *Research Quarterly for Exercise & Sport,* 62, 213-216.
Serresse, O., Lortie, G., Bouchard, C. & Boulay, M. R. (1988) Estimation of the contribution of the various energy systems during maximal work of short duration. *International Journal of Sports Medicine,* 9, 456-460.

Shephard, R. J. (1984) Tests of maximal oxygen uptake: a critical review. *Sports Medicine,* 1, 99-124.

Shiomi, T., Maruyama, H., Saito, A. & Umemura, M. (2000) Physiological Responses and Mechanical Efficiency during Different Types of Ergometric Exercise. *Journal of Physical Therapy Science,* 12, 67-73.

Smith, J. C. & Hill, D. W. (1991) Contribution of energy systems during a Wingate power test. *British Journal of Sports Medicine,* 25, 196-199.

Smith, P. M., Amaral, I., Doherty, M., Price, M. J. & Jones, A. M. (2006a) The Influence of Ramp Rate on VO_{2peak} and "Excess" VO_2 during Arm Crank Ergometry. *International Journal of Sports Medicine,* 27, 610-616.

Smith, P. M., Chapman, M. L., Hazlehurst, K. E. & Goss-Sampson, M. A. (2008) The influence of crank configuration on muscle activity and torque production during arm crank ergometry. *Journal of Electromyography & Kinesiology,* 18, 598-605.

Smith, P. M., Davison, R. C. R. & Price, M. J. (2002a) Blood lactate profile after two different arm crank ergometry tests. (Abstract). *Journal of Sports Sciences,* 20, 58-59.

Smith, P. M., Doherty, M. & Price, M. J. (2002b) The influence of protocol design on peak physiological responses to arm crank ergometry. (Abstract) *Journal of Sports Sciences,* 20, 27-28.

Smith, P. M., Doherty, M. & Price, M. J. (2006b) The Effect of Crank Rate on Physiological Responses and Exercise Efficiency Using a Range of Submaximal Workloads During Arm Crank Ergometry. *International Journal of Sports Medicine,* 27, 199-204.

Smith, P. M., Doherty, M. & Price, M. J. (2007a) The effect of crank rate strategy on peak aerobic power and peak physiological responses during arm crank ergometry. *Journal of Sports Sciences,* 25, 711-718.

Smith, P. M., Mccrindle, E., Doherty, M., Price, M. J. & Jones, A. M. (2006c) Influence of crank rate on the slow component of pulmonary O_2 uptake during heavy arm-crank exercise. *Applied Physiology, Nutrition & Metabolism,* 31, 292-301.

Smith, P. M. & Price, M. J. (2007) Upper-body exercise. In: E. M. Winter., A. M. Jones., R. C. R. Davison., P. D. Bromley. & T. H. Mercer. (eds).

BASES Sport and Exercise Physiology Testing Guidelines. pp. 138 - 144. 1st Ed. ed.

Smith, P. M., Price, M. J., Davison, R. C. R., Scott, D. & Balmer, J. (2007b) Reproducibility of power production during sprint arm ergometry. *Journal of Strength & Conditioning Research,* 21, 1315-1319.

Smith, P. M., Price, M. J. & Doherty, M. (2001) The influence of crank rate on peak oxygen consumption during arm crank ergometry. *Journal of Sports Sciences,* 19, 955-960.

So, R., Chan, K. M., Appel, R. & Yuan, Y. (2004) Changes in the multi-join kinematics and co-ordination after repetitive windsurfing pumping task. *Journal of Sports Medicine and Physical Fitness,* 44, 249-257.

Souissi, N., Bessot, N., Chamari, K., Gauthier, A., Sesboue, B. & Davenne, D. (2007) Effect of Time of Day on Aerobic Contribution to the 30-s Wingate Test Performance. *Chronobiology International: The Journal of Biological & Medical Rhythm Research,* 24, 739-748.

Spencer, M., Fitzsimons, M., Dawson, B., Bishop, D. & Goodman, C. (2006) Reliability of a repeated-sprint test for field-hockey. *Journal of Science and Medicine in Sport,* 9, 181-184.

Stamford, B. A., Cuddihee, R. W., Moffatt, R. J. & Rowland, R. (1978) Task specific changes in maximal oxygen uptake resulting from arm versus leg training. *Ergonomics,* 21, 1-9.

Stenberg, J., Astrand, P. O., Ekblom, B., Royce, J. & Saltin, B. (1967) Hemodynamic response to work with different muscle groups, sitting and supine. *Journal of Applied Physiology,* 22, 61-70.

Stewart, D., Farina, D., Shen, C. & Macaluso, A. (2011) Muscle fibre conduction velocity during a 30-s Wingate anaerobic test. *Journal of Electromyography and Kinesiology,* 21**,** 418-422.

Stickley, C. D., Hetzler, R. K. & Kimura, I. F. (2008) Prediction of anaerobic power values from an abbreviated WAnT protocol. *Journal of Strength & Conditioning Research (Lippincott Williams & Wilkins),* 22**,** 958-965.

Stoggl, T. L. & Muller, E. (2009) Kinematic Determinants and Physiological Response of Cross-Country Skiing at Maximal Speed. *Medicine & Science in Sports & Exercise,* 41**,** 1476-1487.

Strang, A. J., Berg, W. P. & Hieronymus, M. (2009) Fatigue-induced early onset of anticipatory postural adjustments in non-fatigued muscles: support for a centrally mediated adaptation. *Experimental Brain Research.* **,** 197**,** 245-254.

Swaine, I. L. & Winter, E. M. (1999) Comparison of cardiopulmonary responses to two types of dry-land upper-body exercise testing modes in competitive swimmers. *European Journal of Applied Physiology & Occupational Physiology,* 80**,** 588-590.

Swensen, T., Mancuso, P. & Howley, E. T. (1993) The effect of moderate resistance weight training on peak arm aerobic power. *International Journal of Sports Medicine,* 14**,** 43-47.

Szygula, Z., Gawroñski, W. & Kalinski, M. I. (2003) Fatigue during exercise. *Medicina Sportiva,* 7**,** 57-67.

Tabata, I., Irisawa, K., Kouzaki, M., Nishimura, K., Ogita, F. & Miyachi, M. (1997) Metabolic profile of high intensity intermittent exercises. *Medicine and Science in Sports and Exercise,* 29**,** 390-395.

Tarara, D. (1995) A comparison of peak VO_2 and HR responses during arm crank ergometry versus upper-body pedalling. Eugene, Ore.; United States Microform Publications, Int'l Inst for Sport & Human Performance, University of Oregon.

Taylor, B. J., How, S. C. & Romer, L. M. (2006) Exercise-induced abdominal fatigue in healthy humans. *Journal of Aplied Pysiologiy,* 100, 1554-1562.

Taylor, J. L., Butler, J. E. & Gandevia, S. C. (2000) Changes in muscle afferents, motorneurons and motor drive during muscle fatigue. . *European Journal of Aplied Physiology,* 83 106-115.

Terbizan, D. J. & Seljevold, P. J. (1996) Physiological profile of age-group wrestlers. *Journal of Sports Medicine & Physical Fitness,* 36, 178-185.

Tew, G., Shah, N., Irena, Z. & John, M. S. (2009) Limb-specific and cross-transfer effects of arm-crank exercise training in patients with symptomatic peripheral arterial disease. *Clinical Science* 117, 405–413.

Timmons, J. A., Jansson, E., Fischer, H., Gustafsson, T., Greenstaff, P. L., Ridden, J., Rachman, J. & Sundverg, C. J. (2005) Modulation of extracellular matrix genes reflects the magnitude of physiological adaptations to aerobic exercise training in humans. *BMC Biology*. BioMed Central.

Tordi, N., Belli, A., Mougin, F., Rouillon, J. R. & Gimenez, M. (2001) Specific and transfer effects induced by arm or leg training. *International Journal of Sports Medicine,* 22, 517-524.

Tortora, G. J. & Grabowski, S. R. (2003) *Principles of Anatomy & Physiology, 10th Ed.* New York, USA: John Wiley & Sons, Inc.

Tulppo, M. P., Makikallio, T. H., Laukkanen, R. T. & Huikuri, H. V. (1999) Differences in autonomic modulation of heart rate during arm and leg exercise. *Clinical Physiology,* 19, 294-299.

Tyler, C. & Sunderland, C. (2008) The Effect of Ambient Temperature on the Reliability of a Preloaded Treadmill Time-Trial. *International Journal of Sports Medicine,* 29, 812-816.

Üçok, K., Gökbel, H. & Okudan, N. (2005) The load for the Wingate test: according to the body weight or lean body mass. *European Journal of General Medicine,* 2 10-13.

Ucok, K., Mollaoglu, H., Akgun, L. & Genc, A. (2008) Anaerobic performance in patients with chronic low back pain. *Journal of Back & Musculoskeletal Rehabilitation,* 21, 99-104.

Van Drongelen, S., Maas, J. C., Scheel-Sailer, A. & Van Der Woude, L. H. (2009) Submaximal arm crank ergometry: Effects of crank axis positioning on mechanical efficiency, physiological strain and perceived discomfort. *Journal of Medical Engineering & Technology,* 33, 151-157.

Van Someren, K. A. & Palmer, G. S. (2003) Prediction of 200-m sprint kayaking performance. *Canadian Journal of Applied Physiology,* 28, 505-517.

Vanderthommen, M., Francaux, M., Johnson, D., Dewan, M., Lewyckyj, Y. & Sturbois, X. (1997) Measurement of the power output during the acceleration phase of all-out arm cranking exercise. *International Journal of Sports Medicine,* 18, 600-606.

Vandewalle, H., Peres, G., Heller, J. & Monod, H. (1985a) All out anaerobic capacity tests on cycle ergometers. *European Journal of Applied Physiology and Occupational Physiology,* 54, 222-229.

Vandewalle, H., Peres, G., Heller, J. & Monod, H. (1985b) All out anaerobic capacity tests on cycle ergometers. A comparative study on men and women. *European Journal Of Applied Physiology And Occupational Physiology,* 54, 222-229.

Vandewalle, H., Peres, G. & Monod, H. (1987) Standard anaerobic exercise tests. *Sports Medicine,* 4, 268-289.

Verellen, J., Meyer, C., Janssens, L. & Vanlandewijck, Y. (2011) Peak and submaximal steady-state metabolic and cardiorespiratory responses during arm-powered and arm-trunk-powered handbike ergometry in able-bodied participants *2012,* 11.

Voight, M. L., Hardin, J. A., Blackburn, T. A., Tippett, S. & Canner, G. C. (1996) The effects of muscle fatigue on the relationship of arm dominance to shoulder proprioception. *Journal of Orthopaedic & Sports Physical Therapy,* 23, 348-352.

Vokac, Z., Bell, H., Bautz-Holter, E. & Rodahl, K. (1975) Oxygen uptake/heart rate relationship in leg and arm exercise, sitting and standing. *Journal of Applied Physiology,* 39, 54-59.

Volianitis, S., Yoshiga, C. C., Nissen, P. & Secher, N. H. (2004a) Effect of fitness on arm vascular and metabolic responses to upper body exercise. *American Journal of Physiology. Heart and Circulatory Physiology,* 286, 1736-1741.

Volianitis, S., Yoshiga, C. C., Vogelsang, T. & Secher, N. H. (2004b) Arterial blood pressure and carotid baroreflex function during arm and combined arm and leg exercise in humans. *Acta Physiologica Scandinavica,* 181, 289-295.

Walker, R., Powers, S. & Stuart, M. K. (1986) Peak oxygen uptake in arm ergometry: effects of testing protocol. *British Journal of Sports Medicine,* 20, 25-26.

Walker, S., Davis, L., Avela, J. & Hakkinen, K. (2012) Neuromuscular fatigue during dynamic maximal strength and hypertrophic resistance loadings. *Journal of Electromyography and Kinesiology,* 3, 356-362.

Warren, G. L., Cureton, K. J., Dengel, D. R., Graham, R. E. & Ray, C. A. (1990) Is the gender difference in peak VO_2 greater for arm than leg exercise? *European Journal of Applied Physiology & Occupational Physiology,* 60, 149-154.

Washburn, R. A. & Seals, D. R. (1984) Peak oxygen uptake during arm cranking for men and women. *Journal of Applied Physiology,* 56, 954-957.

Weber, C. L., Chia, M. & Inbar, O. (2006) Gender Differences in Anaerobic Power of the Arms and Legs-A Scaling Issue. *Medicine & Science in Sports & Exercise,* 38, 129-137.

Weissland, T., Marais, G., Robin, H., Vanvelcenaher, J. & Pelayo, P. (1999) Relationship in humans between spontaneously chosen crank rate and power output during upper body exercise at different levels of intensity *European Journal of Applied Physiology,* 79, 230-236.

Weissland, T., Pelayo, P., Vanvelcnaher, J., Marais, G., Lavoie, J. M. & Robin, H. (1997) Physiological effects of variations in spontaneously chosen crank rate during incremental upper-body exercise. *European Journal of Applied Physiology & Occupational Physiology,* 76, 428-433.

Westerblad, H., Allen, D. G. & Lannergren, J. (2002) Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News in Physiological Sciences,* 17, 17-21.

Westhoff, T. H., Schmidt, S., Gross, V., Joppke, M., Zidek, W. & Dimeo, F. (2008) The cardiovascular effects of upper-limb aerobic exercise in hypertensive patients. *Journal of Hypertension,* 26, 1336-1342.

Willardson, J. M. (2007) Core Stability Training: Applications to Sports Conditioning Programs. *Journal of Strength and Conditioning Research,* 21, 979-985.

Williams, C. & Wragg, C. (2004) Data Analysis and Research for Sport and Exercise Science, 1st Ed. London: Routledge.

Wilmore, J. H. & Costill, D. L. (1999) *Physiology of Sport and Exercise, 2nd Ed.* Champagne, Ill: Human Kinetics.

Winter, E. M. (1991) Cycle ergometry and maximal intensity exercise. *Sports Medicine,* 11, 351-357.

Winter, E. M., Brown, D., Roberts, N. K. A., Brookes, F. B. C. & Swaine, I. L. (1996) Optimized and corrected peak power output during friction-braked cycle ergometry. *Journal of Sports Sciences,* 14, 513-521.

Winter, E. M., Eston, R. G. & Lamb, K. L. (2001) Statistical analyses in the physiology of exercise and kinanthropometry. *Journal of Sports Sciences,* 19, 761-775.

Winter, E. M. & Maclaren, D. P. (2001) Assessment of maximal-intensity exercise. In: Eston, R & Reilly, T. (eds). Kinanthropometry and exercise physiology laboratory manual: tests, procedures and data. pp. 263-288, 2nd Ed. London; United Kingdom: Routledge.

Wright, R. L., Wood, D. M. & James, D. V. B. (2007) Effect of Starting Cadence on Sprint-Performance Indices in Friction-Loaded Cycle Ergometry. *International Journal of Sports Physiology & Performance,* 2, 22-33.

Xu, F. & Rhodes, E. C. (1999) Oxygen Uptake Kinetics During Exercise. *Sports Medicine,* 27, 313-327.

Yasuda, N., Ruby, B. C. & Gaskill, S. E. (2002) Substrate utilization during arm and leg exercise relative to the ventilatory threshold in men. *Journal of Sports Medicine & Physical Fitness,* 42, 403-408.

Yasuda, N., Ruby, B. C. & Gaskill, S. E. (2006) Substrate oxidation during incremental arm and leg exercise in men and women matched for ventilatory threshold. *Journal of Sports Sciences,* 24, 1281-1289.

Yasuda, N., S. E. Gaskill, B. C. Ruby (2008) No gender-specific differences in mechanical efficiency during arm or leg exercise relative to ventilatory threshold. *Scandinavian Journal of Medicine & Science in Sports* 18, 205-212.

Yosefy, C., Jafari, J., Klainman, E., Brodkin, B., Handschumacher, M. D. & Vaturi, M. (2006) The prognostic value of post-exercise blood pressure reduction in patients with hypertensive response during exercise stress test. *International Journal of Cardiology,* 111, 352-357.

Zagatto, A. M., Papoti, M. & Gobatto, C. A. (2008) Anaerobic capacity may not be determined by critical power model in elite table tennis players. *Journal of Sports Science & Medicine,* 7, 54-59.

Zehr, E. P. & Chua, R. (2000) Modulation of human cutaneous reflexes during rhythmic cyclical arm movement. *Experimental Brain Research,* 135, 241-250.

Ziemann, E., Grzywacz, T., Luszczyk, M., Laskowski, R., Olek, R. A. & Gibson, A. L. (2011) Aerobic and anaerobic changes with high-intensity interval training in active college-aged men. *Journal of Strength & Conditioning Research* 25, 1104-1112.

Ziemba, A. W., Chwalbinska-Moneta, J., Kaciuba-Uscilko, H., Kruk, B., Krzeminski, K., Cybulski, G. & Nazar, K. (2003) Early effects of short-term training. *Journal of Sports Medicine and Physical Fitness,* 43, 57-63.

Zwierska, I., Walker, R. D., Choksy, S. A., Male, J. S., Pockley, A. G. & Saxton, J. M. (2005) Upper- vs lower-limb aerobic exercise rehabilitation in patients with symptomatic peripheral arterial disease: a randomized controlled trial. *Journal of Vascular Surgery.*, 42, 1122-1130.