INFLUENCE OF KNOWLEDGE OF THE END-POINT ON PACING DURING A 2000M ROWING TIME TRIAL

BY

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THESIS

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ABSTRACT

Introduction: A typical 2000 m rowing race requires maximal force production over six to eight minutes. Optimal distribution of energetic resources during such a race is vital for optimal performance. There is little research examining the pacing strategies employed by rowers, particularly at the sub-elite level.

Aim: The purpose of this investigation was to determine the influence of knowledge of the end-point on pacing during a 2000 m rowing time trial.

Methods: Eleven male rowers from a university rowing club volunteered to partake in the study. Each participant completed three experimental trials on an indoor rowing ergometer, each 2000 m. The only difference between the trials was the nature of the information provided beforehand. At the start of the *control trial*, participants were correctly informed about the distance to be covered. Participants were not informed of the distance to be completed in the *unknown trial*. For the *deceptive trial*, participants were told 1000 m would be completed, but when this distance was reached, they were told to continue for another 1000 m. During each trial muscle activity, power output, heart rate, performance time and perceptions of effort were measured.

Results: The control trial was significantly ($p<0.05$) faster than both the unknown and deceptive trials, however the deceptive trial was the fastest of all trials at the 1000 m distance. The unknown trial was slowest at 1000 m and at 2000 m. Muscle activity, RPE, heart rate and power output were significantly ($p<0.05$) lower in the unknown trial compared to the control and deceptive trial. The control trial exhibited a reverse J-shape pacing profile. The deceptive trial revealed a significant ($p<0.05$) reduction in performance time, heart rate, power output and muscle activity after the 1000 m interval. The first 500 m interval had the fastest performance and highest power output in all trials. In all trials, RPE was highest at the end of the 2000 m, the greatest reading being obtained at the end of the control trial. During the first 1000 m of the deceptive trial, the dependant variables were similar to those in the control trial. Once the deception was revealed, there was a significant ($p<0.05$) reduction in the muscle activity, heart rate and power output.

Conclusion: The results of this study indicate that accurate end-point information is crucial to the development of an optimal pacing strategy, and ultimately to successful performance. Rowers tend to adopt the reverse J-shape pacing profile most often due

to the tactical and physiological benefits offered by this strategy. Unknown or inaccurate end-point information resulted in performance decrements due to the uncertainty associated with the exercise bout.

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CHAPTER I

INTRODUCTION

BACKGROUND TO THE STUDY

Rowing is a high intensity sport requiring repetitive contraction of all major muscle groups (Hosea and Hannafin, 2012). Rowing races are completed over a wide range of distances, with the most common being a 2000m sprint. A typical 2000m race requires maximal power production over approximately 6-8 minutes, thereby taxing both the cardiovascular and musculoskeletal systems (Secher and Volianitis, 2007).

Optimal distribution of energetic resources during the course of a race is known as a pacing strategy (Muehlbauer *et al.*, 2010). This strategy is defined as the efficient use and distribution of energetic resources across an exercise task, so that all available energy stores are used before the end of the exercise task, but not so far from the end that a meaningful slowdown can occur (Abbiss and Laursen, 2008; Tucker and Noakes, 2009). An optimal pacing strategy is crucial to successful performance (Muehlbauer *et al.*, 2010). This is particularly relevant to a sport such as rowing where very high workloads are maintained for extended periods of time. Even a 1% improvement in performance can be the difference between winning and losing, thus optimising energetic resources is essential to successful performance.

The typical pacing strategy observed during a 2000m rowing race is known as the J-shaped profile (Garland, 2005; Brown *et al.*, 2010; Muehlbauer *et al.*, 2010). It is characterised by a fast start, after which a reduction in speed is seen and then a final increase in speed towards the end of the course (Abbiss and Laursen, 2008). Tactically and psychologically the fast start is advantageous (Shephard, 1998). Physiologically there does seem to be some merit to a fast start as a high starting exercise intensity allows a greater total oxygen uptake over the duration of the race (Secher and Volianitis, 2007). The end spurt occurs when teams or individuals attempt to secure a finishing position. However, this parabolic pacing profile is not necessarily optimal for a rowing race (Lynch, 2012). It has been suggested that an even pacing profile, where speed is kept constant throughout the race, would be more suitable to rowing (Billat *et al.*, 1999). Due to the fluid resistance of the water, even minor

fluctuations in speed can be energetically costly (Abbiss and Laursen, 2008). Thus, it is thought that performance may be improved if accelerations and decelerations are minimised throughout a race (Lynch, 2012).

While pacing strategies are known to be a vital component of optimal athletic performance, the physiological mechanisms concerning the regulation of exercise are widely debated in the literature (Bassett and Howley, 1997; Noakes, 2011; Shei and Mickleborough, 2013). The prevailing theory of fatigue does not explain the occurrence of a pacing strategy. Additionally, the exact mechanism by which factors such as training and previous experience optimize performance is still unknown. However, it is known that certain "inputs" are required for an optimal pacing strategy, such as exercise end-point information (Ansley *et al.*, 2004; Tucker, 2009). Training and preparation can be modified and improved by investigating and understanding the mechanisms pertaining to pacing strategies. Optimizing these inputs will cause an alteration in the outputs (such as muscle activation) and ultimately improve athletic performance.

A recent theory, known as anticipatory regulation, posits that exercise intensity is regulated largely sub-consciously in a complex, integrated manner (Tucker and Noakes, 2009; Swart *et al.*, 2009; Marino, 2014). A template rating of perceived exertion (RPE) is created by taking into account several internal and external factors such as environmental temperature, energy availability and previous experience of the exercise (Tucker, 2009). The template RPE, which contains information on how the athlete aims to distribute energetic resources during the event, is then compared to the conscious RPE during the exercise bout (Joseph *et al.*, 2008; Tucker and Noakes, 2009). If there is a mismatch between the template and conscious RPE, exercise intensity will be adjusted to avoid any hazardous changes to homeostasis (Tucker, 2009). Research has shown that a key component of the template RPE is precise end-point information about the exercise (Tucker, 2009). Deception studies have been designed to investigate this factor by manipulating the athlete's knowledge of the end-point of exercise. These studies are performed by providing participants with false information about the duration or distance of exercise to be completed. In so doing, the athlete's expectation and previous experience of the exercise task is modified causing the adopted pacing strategy to be suboptimal (Williams *et al.*, 2014). It is not often that rowers compete in events where the distance to be completed is unknown,

however the impact of deception on performance in rowing is currently unknown. By studying the effects of deception in rowing, coaches may be able to improve training methods and tactical pacing strategies, which could improve performance.

STATEMENT OF THE PROBLEM

The purpose of this investigation was to determine whether the absence or extension of a known end-point at the beginning of a rowing bout prevents rowers from adopting a suitable pacing strategy.

RESEARCH HYPOTHESES

It was expected that the pacing strategies adopted by the rowers, and the responses pertaining thereto, would be altered by the accuracy of the end-point information provided.

The accuracy of the end-point information was expected to alter the pacing strategies of the rowers and thereby affect their responses.

STATISTICAL HYPOTHESES

When comparing the responses obtained from each of the three trials, the following statistical hypotheses were applied:

Ho : $\mu_1 = \mu_2 = \mu_3$

Ha: $\mu_1 \neq \mu_2 \neq \mu_3$

Where: μ_1 = control trial

μ_2 = unknown trial

μ_3 = deceptive trial

STRUCTURE OF THE THESIS

Chapter 1 provides a brief overview of the study as well as the statement of the problem. It also posits research and statistical hypotheses pertaining to the study.

Chapter 2 provides a review of literature related to rowing, highlighting the physical characteristics of rowers. Additionally, it explains pacing strategies and theories of the physiological mechanism of pacing. Finally, information is provided on previous research which has attempted to manipulate the pacing strategy.

Chapter 3 details the methodology employed in the study including factors such as ethical approval, research design, measurements and equipment. It also covers statistical analyses, participant recruitment and the testing environment among other aspects.

Chapter 4 presents the findings of the investigation.

Chapter 5 discusses the findings presented in the previous chapter. These are related to previous scientific literature. Each variable is interpreted separately in order to provide a simple and thorough analysis of the results.

Chapter 6 concludes the study by summarising key findings and concepts. The limitations of the study are included, as well as recommendations for future research.

Documents considered relevant to the study are attached as appendices.

CHAPTER II

REVIEW OF LITERATURE

Overview of Rowing

Rowing is a sport widely participated in by countries around the world and includes a diverse range of classes such as coastal, adaptive, sprint and indoor rowing (Lynch, 2012). The sport is governed by the Fédération Internationale des Sociétés d'Aviron (FISA) and is included in the Olympic Games with a total of 22 events (Lynch, 2012). Rowing is divided into two categories: sweep rowing and sculling. Sweep rowing involves the use of a single oar per rower on one side of the boat and is performed in boats ranging from two to eight people, excluding the coxswain (Hagerman, 1983). Sculling can be performed by between one and four people and requires the rowers to manipulate two oars concurrently (Hagerman, 1983).

There exist perceptible similarities between the two types of rowing. However, it is important to note significant differences between the techniques of sweep rowing and sculling. Elite rowers tend to specialise in one of the two techniques in order to perfect that technique and develop the relevant muscles (Secher, 1993). The sculling technique is generally considered more difficult (McArthur, 1997). During sweep rowing the outside leg (furthest from the oar) contributes more force to the oar than the inside leg. In addition, the forces produced by each leg peak at different times during the stroke (Secher, 1993). As the oar enters the water, rowers assume a cramped posture in order to lengthen the stroke (Yoshiga and Higuchi, 2003). This posture is thought to restrict breathing and circulation, while also contributing to significant anatomical and physiological stress (Yoshiga and Higuchi, 2003; Hosea and Hannafin, 2012).

The sport is further divided into weight categories. Lightweight male rowers can have a maximum mass of 72.5 kg, while the average weight of the rowers in the boat must be less than 70.0 kg. Lightweight females have a weight limit of 59.0 kg with a mean crew limit of 57.0 kg (Secher and Volianitis, 2007). The heavyweight or open-weight categories have no weight limit for both males and females (Secher and Volianitis, 2007). The duration of a 2000m rowing event varies between 5.8 minutes at the elite

level, and 7.4 minutes at the sub-elite level, depending on the level of competition, weather and water conditions, and the type of boat used (Secher, 1983).

Rowing varies from many other types of competitive sport for several reasons. Firstly, rowing requires the active recruitment of almost every muscle group in the body. The arm, leg and back muscles are involved in each stroke (Secher, 1983; Hosea and Hannafin, 2012). This contrasts with cycling, for example, where the arms perform little work (Secher, 1983; Secher, 1993). Consequently, a rower's physiology is severely strained during a bout of "all-out" rowing with extreme changes in blood variables being an indication of this (Secher, 1993; Shephard, 1998). Secondly, the rowing stroke requires that most muscles interchange between static and dynamic exercise (Secher, 1993). This has important implications for breathing and circulation (Strange, 1999). Finally, rowers are required to be seated while both legs apply the same force concurrently. This is not usually the case in most sports (Secher, 1993). For example, during running one leg is predominantly performing work at a time (Secher, 1983). Therefore, an evaluation of rowing requires an alternative outlook in which the posture, muscle involvement, and the effects thereof are not only acknowledged but also investigated.

Characteristics of the Elite Rower

It is important to clarify that while some of the characteristics mentioned below are genetically determined, others are the result of training, often over several years. As an example, height is predominantly a genetically determined trait and will not alter a great deal with training (Yang *et al.*, 2010). Rowers who are taller are more likely to be successful as is the case with many other genetically determined factors. However, rowers tend to develop other characteristics due to training over long periods of time. An example of this would be muscle fibre type (Rockl *et al.*, 2007). Consequently, genetically determined traits predispose certain people to rowing which then results in the expression of environmentally determined traits. This concept is illustrated in Figure 1 (page 7) and further explanation can be found in an article by Tucker and Collins (2012).

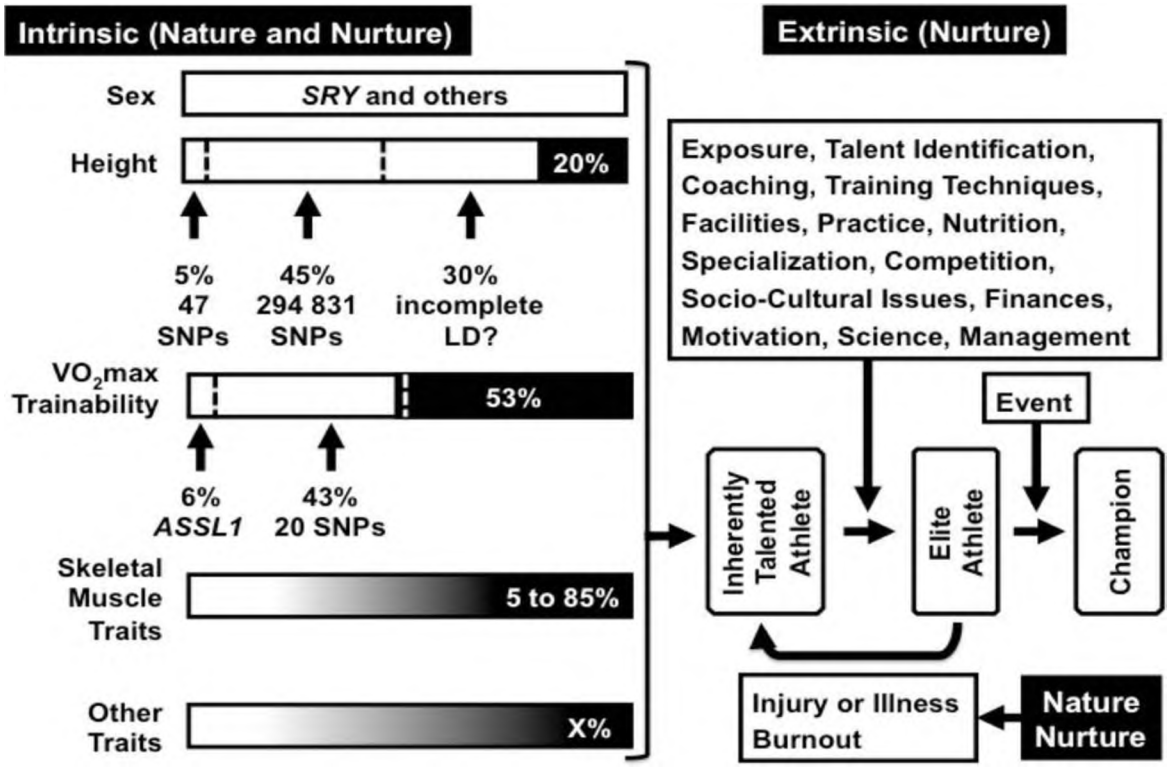

Figure 1: The complex relationship between environmental and genetic factors in determining athleticism (Tucker and Collins, 2012). SRY and others are genes which determine male sexual differentiation. Incomplete linkage disequilibrium refers to specific genes coding for height. SNPs are single nucleotide polymorphisms and ASSL1 is a gene which plays a large role in determining maximal oxygen uptake. Light shaded areas are nature, while dark shading indicates nurture.

Anthropometrical and Morphological Characteristics

Elite rowers are invariably large individuals with high aerobic work capacities (Spinks, 1986; Secher and Volianitis, 2007). They also have a higher lean muscle mass, shorter sitting height (relative to stature) and longer arms than sub-elite rowers in both males and females (Slater *et al.*, 2004; Mikulic, 2008; Cetinkhaya *et al.*, 2014). It is advantageous to recruit rowers with a large proportion of lean muscle mass as this ensures that the majority of the mass transported is active muscle rather than "dead weight" (Shephard, 1998). Data from over 600 oarsmen have shown that heavyweight rowers have an average height of 192 cm and average weight of 88 kg with a body fat percentage of between 10 and 11%. Lightweight individuals have a similar average height, but a decreased body fat percentage of between 7 – 8% (Hagerman, 1984). Elite female rowers weigh an average of 70 kg with an average height of 173 cm and between 12 and 25% body fat (Hagerman, 1984). Another study found that collegiate

rowers were significantly taller and heavier in comparison to their sedentary counterparts thus confirming that rowers have a distinct morphology compared to the general population (Cetinkhaya *et al.*, 2014). This morphology can be described as a trade-off between the competing demands of the weight limits and the maximisation of muscle mass (Schranz *et al.*, 2010). Initially, body fat percentage of elite rowers had been decreasing , possibly due to improved training programmes and dietary habits, while more recent research is required to confirm whether this trend is continuing (Hagerman, 1984; Shephard, 1998).

Aerobic and Ventilatory Characteristics

Compared to other endurance athletes, rowers tend to possess a large aerobic capacity (Astrand and Shephard, 2000). However, when relative values are calculated, misinterpretation is common because elite rowers consistently have a higher mass than amateur rowers (Izquierdo-Gabarren *et al.*, 2010). Therefore, absolute values may be more important than relative values when considering rowing as bodyweight is supported by the boat (Hagerman, 1984; Secher and Volianitis, 2007). Hagerman (1984) reported VO_{2max} values of elite male rowers in excess of 7.0 $L.min^{-1}$ and over 5.0 $L.min^{-1}$ in females, corresponding to relative values of over 80.0 $ml.kg^{-1}.min^{-1}$ for males and over 70.0 $ml.kg^{-1}.min^{-1}$ for females. In comparison, elite Kenyan long distances runners were found to have mean VO_{2max} values of 78.4 $ml.kg^{-1}.min^{-1}$ for males and 68.6 $ml.kg^{-1}.min^{-1}$ of females (Billat *et al.*, 2003). Another study found the mean VO_{2max} value for 35 male Olympic rowers to be 6.2 $L.min^{-1}$ corresponding to a relative value of 71.0 $ml.kg^{-1}.min^{-1}$ (Seiler, 1996). The same study tested 25 female Olympic rowers and found a mean value of 4.3 $L.min^{-1}$ or 58.6 $ml.kg^{-1}.min^{-1}$. Thus, rowers typically have large aerobic capacities, with some female values being within the top 10% of the best international female long distance runners (Astrand and Shephard, 2000). This evidence indicates that a large metabolic capacity is required to be a successful rower.

Rowers are required to breathe cyclically (in phase with the rowing stroke) while in a cramped posture and it has been proposed that this posture may impose significant restrictions on air conduction and respiratory muscle function (Yoshiga and Higuchi, 2003). Minute ventilation (V_E) is increased during rowing as would be expected in

maximal exercise (Lynch, 2012). Minute ventilation increases exponentially with the onset of rowing exercise until approximately the third minute (Secher and Volianitis, 2007). The rate of increase then slows but continues to rise until exercise terminates. Values as high as 250 - 270 $L.min^{-1}$ have been recorded in rowers and these values are reported to be the highest in competitive endurance sports (Secher and Volianitis, 2007). The high V_E and peak flow rate values negate the premise that the cramped posture impairs respiratory muscle function or airway conduction. However, performance may be improved by entrainment of the respiratory muscles (Volianitis *et al.*, 2001; Yoshiga and Higuchi, 2003). It follows that the high metabolic cost of rowing is compensated for by high minute ventilation and peak flow rates.

Muscle Force Production Capabilities

Competitive rowers generate much higher muscular forces compared to other endurance athletes and it is believed that this is due to the increased muscle mass of rowers (Secher and Volianitis, 2007). Rowers must combine both a high stroke force and optimal stroke length in order to produce an effective rowing stroke (Secher and Volianitis, 2007). Forces are transmitted through the trunk of the rower to the oar. Effective force transmission is essential as rowers produce approximately 75-80% of power with the legs and 20-25% of power with the arms (Secher and Volianitis, 2007). When compared to other endurance athletes, rowers have been shown to have the highest force-velocity relationship (Hagerman, 1984). This has been attributed to the difference in muscle fibre type proportions, since a close association has been found between muscle fibre type composition and force velocity curves (Hagerman, 1984). It has been shown that muscles which make the greatest contribution to the rowing stroke are comprised predominantly of slow twitch fibres, with the remaining fibres being fast oxidative-glycolytic (Steinacker, 1993).

In a comparison of isokinetic quadriceps strength among elite athletes, male rowers had the greatest absolute strength between 30 - 120 $°.s^{-1}$ compared to swimmers, cyclists and canoeists (Hagerman, 1984). The same study found female rowers to have relative leg strength values approaching those of elite male swimmers, cyclists and canoeists (Hagerman, 1984). However, strength endurance is also crucial to success in rowing since forces must be maintained for approximately 200 strokes at a

rate of above 34 strokes.min^{-1} during a 2000m race (Lynch, 2012). Elite athletes performed one minute of maximal repetition knee extension and it was shown that rowers achieved an average of 6637 Nm.min^{-1} while other elite athletes averaged 5000 Nm.min^{-1}, thus indicating the greater strength endurance of rowers (Hagerman, 1984).

Compared to sedentary individuals, rowers have almost double the muscular capillary density (Hagerman, 1984). The increased capillary density is most likely an adaptation of the muscle due to the high nutrient requirements of the muscles when rowing, as well as the increased muscle fibre area (Steinacker, 1999; Robbins *et al.*, 2009). In conjunction with this, rowers also possess high oxidative metabolic capacities (Steinacker, 1993). Rowing training increases mitochondrial density and oxidative capacity of fast and slow twitch muscle fibres (Steinacker, 1993). This, together with greater oxidative metabolic capacities, allow rowers to sustain the high metabolic cost typical of the sport.

Aerobic and Anaerobic Metabolism in Rowers

It is estimated that aerobic metabolism accounts for 75-80% of the energy required for a 2000m race, while anaerobic metabolism provides the remaining 20-25% (Hagerman, 1984; Secher and Volianitis, 2007). A more recent study found 87% aerobic and 13% anaerobic contributions for on water rowing, with slight differences of 84% aerobic and 16% anaerobic contributions for ergometer rowing (de Campos Mello *et al.*, 2009). Anaerobic energy metabolism is greatest at the start and finish of the race, while the middle stage of the race is primarily aerobic. A fast start is both tactically and physiologically beneficial for rowers. A physiological explanation for the initial spurt performed by rowers is the linear relationship between the rate of oxygen uptake increase at the onset of exercise and the work output (Secher and Volianitis, 2007). Thus, when exercise is started at a high intensity the total oxygen uptake and work output is larger than when exercise intensity is kept at an average intensity throughout a race (Secher and Volianitis, 2007).

These factors result in high power outputs at the start of the race as well as at the end when the crew are attempting to secure a finishing position (Lynch, 2012). Consequently, high blood lactate levels have been recorded in rowers following an

event. Values of up to 17.0 *m*mol.L^{-1} have been reported in elite rowers (Astrand and Shephard, 2000; Secher and Volianitis, 2007). This contrasts with elite cyclists, paddlers, swimmers and runners where peak lactate levels were found to be 10.8, 8.2, 4.3 and 9.0 *m*mol.L^{-1} respectively (Billat *et al.*, 1996). It is clear that rowing has a substantial anaerobic component. Therefore, success in rowing requires a high blood lactate tolerance (Hagerman, 1984; Lynch, 2012). While blood lactate concentration provides an indication of anaerobic metabolism, a more precise estimation has been made from oxygen debt. Oxygen deficit was calculated to be 88-97 ml.kg^{-1} in elite rowers, a value substantially larger than that reported in runners (Secher and Volianitis, 2007; de Campos Mello *et al.*, 2009).

Pacing Strategies

Optimal performance requires athletes to regulate the rate of work so that exercise output does not exceed the athlete's capacity for exercise. A pacing strategy is defined as the efficient use and distribution of energetic resources across an exercise task so that all available energy stores are used before the end of the exercise task, but not so far from the end that a meaningful slowdown can occur (Abbiss and Laursen, 2008; Tucker and Noakes, 2009). Pacing strategies are affected by numerous factors such as physiological, psychological and environmental variables (Tucker and Noakes, 2009). These variables determine how a pacing strategy is governed internally and externally by the athlete.

Scientific research investigating the pacing strategies employed during competition is scarce (Abbiss and Laursen, 2008). Investigation is required to establish which pacing strategy is most effective for different sports of varying durations and distances. Medal placings are commonly separated by performances within 1% of each other. Thus, understanding the effect that pacing strategies have on performance is fundamental to improving performance, offering an opportunity to acquire a competitive advantage in the highly competitive athletic domain.

Defining Pacing Strategies

The effect pacing strategies have on performance can only be investigated once the mechanism of these strategies has been understood. The majority of individual sporting events (including rowing) are of a "closed-loop" nature, implying that the athlete aims to finish a known distance in the shortest time possible (St Clair Gibson, Lambert and Noakes, 2001). These events are often head-to-head races making tactics, and sometimes teamwork, crucial to overall success (Abbiss and Laursen, 2008). Rowing requires teamwork and is often head-to-head in nature, with the exceptions being singles boats and ergometer rowing (Secher and Volianitis, 2007).

Abbiss and Laursen (2008) have identified six pacing strategies in closed loop sports.

1. A negative pacing strategy is identifiable when there is an increase in speed observed over the duration of the event. It is thought that this type of pacing strategy improves prolonged exercise performance by reducing the rate of carbohydrate depletion, lowering oxygen consumption and limiting the accumulation of fatigue-related metabolites early during the exercise. Negative pacing strategies are most often seen in middle distance events during athletic competition.
2. An all-out pacing strategy requires an athlete to use maximal energy to obtain as high a speed as possible and then attempting to maintain that speed for the duration of the event. This type of pacing strategy is commonly seen in short duration sprint events such as the 100m and 200m events. It is based on the fact that less energy is required to maintain a speed than to accelerate. Typically, speed tends to decrease following the acceleration phase, however, the event is usually completed before any substantial decrease occurs.
3. Positive pacing strategies are noted when an athletes speed declines over the course of the event, usually following a fast start. The fast start can be due to an unrealistic ambition, pacemaker, or due to the nature of the sport e.g. dive when swimming. It is commonly seen that the high starting speed causes a reduction in pace due to homeostatic disruptions e.g. heat or metabolite accumulation. A positive pacing strategy is common in sports such as 100m and 200m swimming events.
4. The even pacing strategy involves an athlete utilising a relatively constant pace throughout the event, usually lasting longer than two minutes. The basis for this

pacing strategy is that the athlete exercises at a level slightly below the fatigue threshold. This allows the athlete to continue exercising for extended periods without a large drop in pace. Even pacing is supported by several mathematical and power models and has been observed in track cyclists.

5. A parabolic-shaped (also known as J-shaped) pacing strategy has been shown to be adopted by rowers, particularly in 2000m events. This strategy involves a fast start, after which athletes slow down slightly and then the pace is increased maximally toward the end of the race. This type of pacing strategy is indicative of anticipatory regulation of exercise.
6. The final pacing strategy is known as the variable pacing strategy during which the athlete's exercise intensity and work rate fluctuates over the duration of the event. This type of pacing strategy is usually adopted in an attempt to counteract changing environmental conditions and is commonly monitored in cyclists and runners where environmental and geographical factors can have major impacts.

Previous Research on Rowing Pacing Strategies

Research has shown that rowers tend to adopt the parabolic-shaped pacing strategy, most often J-shaped, which is commonly observed in maximal extended activities (Garland, 2005; Abbiss and Laursen, 2008; Brown *et al.*, 2010; Muehlbauer *et al.*, 2010). Tactically, it is advantageous for rowers to start a race as fast as possible for three reasons: (a) inertia must be overcome quickly in order to reach top speed; (b) rowers are able to monitor the movements of competitors since they will be facing them; (c) the leading boat misses the wake of other boat/s (Shephard, 1998).

To the knowledge of the author, the first published study examining the pacing strategies adopted by rowers was conducted by Secher (1983), who described an "initial spurt" and an "end spurt" alluding to the J-shaped pacing profile. This study made use of performance analyses of the finalists of the 1974 FISA World Championships. A more comprehensive study was performed by Garland (2005) where split times were analysed during the 2000 Olympic Games as well as the 2001 and 2002 Indoor and Outdoor World Championships. The aforementioned study excluded boats if they were too far behind for a placing or too far ahead that it was not

necessary for maximal effort. This approach was taken in order to ensure that there was no deliberate slowing of pace during the race, which would impact on pacing strategy. The results showed that the first quarter of the race was completed at the highest speed with a subsequent drop in speed over the middle two quarters and a slight increase in speed over the final quarter, typical of the parabolic-shaped pacing strategy. Brown *et al*. (2010) followed Garland's study, examining races at the elite, national and sub-elite competitive levels, while utilising the same exclusion criteria for the boats. It was concluded that a parabolic-shaped pacing profile could describe all performances, both on-water and indoor. The most recent study examining the pacing strategies employed by rowers was performed by Muehlbauer *et al*. (2010) who also found that a parabolic-shaped pacing strategy was evident regardless of race type, boat rank or boat type. Muehlbauer *et al*. (2010) concluded that the parabolic-shaped race profile indicates an anticipatory control of speed and energy distribution over the course of the 2000m race.

The exclusion criteria used by Garland (2005) and later by Brown *et al*. (2010), as well as the limited sample size of the study conducted by Secher (1983) provides cause to question the conclusion of the typical pacing strategy employed during rowing competition. However, Muehlbauer *et al*. (2010), while having a smaller sample size, included all boats and concluded that a reversed J-shaped pacing strategy was exhibited by all boats. While certain aspects of the sport are revealed by observing pacing strategy during rowing competition (e.g. tactics), it does not imply that the established pacing profile is the physiologically optimal pattern (Lynch, 2012). Indeed, it has been posited that due to the fluid resistance encountered in a sport such as rowing, even minor fluctuations in speed can result in greater energy cost (Abbiss and Laursen, 2008). Thus, performance may be optimised by minimising accelerations and decelerations throughout the exercise bout (Secher, 1983; Abbiss and Laursen, 2008).

Consequently, the adoption of an even pacing profile has appeal from both a physiological and performance perspective. Theoretical support for an even pacing strategy has been found in power models and the second law of motion, however it requires that rowers exert a constant maximal force over the duration of the event (Billat *et al.*, 1999; Foster *et al.*, 2003). Furthermore, the time spent in the acceleration phase must be short relative to total duration and environmental conditions are required to be stable, in order to potentially maximise metabolic capacity (Lynch,

2012). To date, no published study has investigated whether an overall even pacing strategy would be effective in rowing races (Lynch, 2012). It is clear that the reverse J-shaped pacing profile is typically adopted by rowers. Therefore, if there was a change to this profile, it may point toward some of the factors which influence the production and adoption of a pacing strategy in athletes.

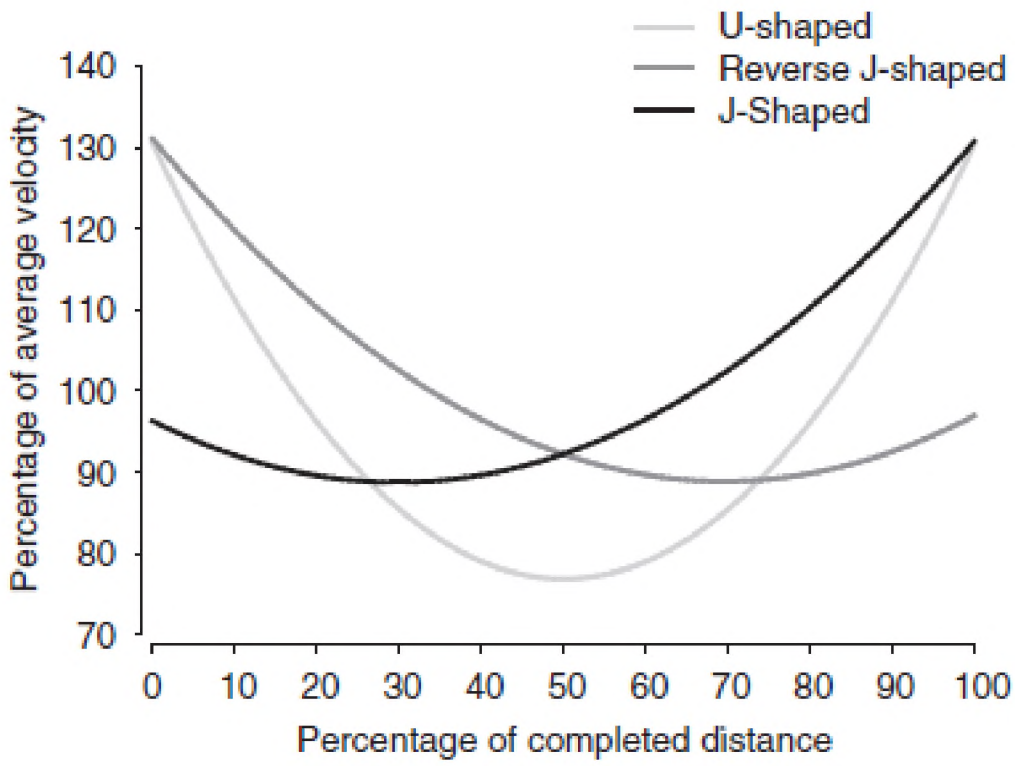

Figure 2: Example of U-shaped, J-shaped and reverse J-shaped pacing strategies (Abbiss and Laursen, 2008).

Physiological Regulation of Pacing

The physiological mechanisms pertaining to the regulation of pacing are obscured and widely debated. It is possible that this uncertainty is due to the fact that an "optimal" distribution of work will be influenced by a variety of internal and external variables, such as nutritional state, course geography, race duration and environmental conditions (Abbiss and Laursen, 2008). However, it is known that pacing strategies

involve the complex interaction between multiple peripheral physiological systems and the brain, both consciously and sub-consciously (Tucker, 2009).

Theories of Exercise-Induced Fatigue

Fatigue and pacing strategies are inherently intertwined and one cannot be understood without the other within the context of exercise. Exercise fatigue is defined as the "progressive, transient and reversible loss of a muscle's ability to perform work" (Shei and Mickleborough, 2013). Currently, there is much controversy in the literature about how fatigue limits athletic performance (Basset and Howley, 1997). Two major and opposing hypotheses have been established, the first being the peripheral model of fatigue (also known as the Catastrophe model) and the other being the central model of fatigue (Basset and Howley, 1997; Kayser, 2003; Noakes and St Clair Gibson, 2004; Shephard, 2009; Marino, Gard and Drinkwater, 2011; Marino, 2014).

The peripheral fatigue model is the most widely known and accepted explanation for fatigue (Noakes, 2000; Shei and Mickleborough, 2013). It has been in place for nearly a century and has been ingrained in sport scientists and exercise physiologists. This is most likely due to early research methods and equipment which could easily explain peripheral features of fatigue, for example the classic VO_{2max} test or lactic acid quantification (Noakes, 2008). However, identifying central factors involved in fatigue would then have been almost impossible (Marino, 2014). Simplistically, the peripheral model of fatigue is based on the relationship between cellular supply and demand, specifically the adenosine triphosphate (ATP) molecule which is the major energy carrying molecule in the body (Shei and Mickleborough, 2013; Marino, 2014). Adenosine triphosphate is generated in the body via three main pathways: the adenosine triphosphate-phosphocreatine system (ATP-PCr), the glycolytic system and the oxidative phosphorylation system (Shei and Mickleborough, 2013). When exercise is performed, adenosine triphosphate is used by the working muscles (Noakes, 2000). The regeneration of adenosine triphosphate results in significant volume of waste products such as lactic acid and carbon dioxide, both of which increase the amount hydrogen (H^+) ions (Bassett and Howley, 1997; Noakes and St Clair Gibson, 2004). It is believed that the cardiovascular system reaches a point where its capacity is exceeded by increasing waste products and oxygen requirements during continuous exercise (Bassett and Howley, 1997; Noakes, 2000; Shephard, 2009; Marino, 2014). The peripheral fatigue model holds that these waste products,

coupled with glycogen and phosphagen depletion, impair the body's ability to provide and regenerate adenosine triphosphate and so progressively less work can be performed, ultimately resulting in exhaustion (Shephard, 2009; Shei and Mickleborough, 2013; Marino, 2014). Furthermore, these waste products may also reduce the force of muscle contraction by reducing calcium ion levels in active muscle and preventing actin-myosin cross bridge formation, giving rise to fatigue (Shei and Mickleborough, 2013).

Recently, an extension of the central fatigue model has been proposed in contrast to the peripheral fatigue model. The anticipatory regulatory model (or anticipatory-RPE model) is derived from central factors, such as nervous system transmission, motivation and motor unit recruitment. In other words, it occurs proximal to the neuromuscular junction (Shei and Mickleborough, 2013). This theory postulates that the decreased capacity for work is due to a decreased motor drive or command from the central nervous system (Kayser, 2003; Noakes, 2008). This decreased motor drive is produced by the central nervous system in response to changing peripheral physiological systems, thereby preventing catastrophic failure of the body (Shei and Mickleborough, 2013; Marino, 2014). Thus, the central nervous system adjusts work output in order to maintain organ system homeostasis (Bassett and Howley, 1997; Kayser, 2003). Research is yet to identify the exact triggers in the peripheral physiological systems, but several have been identified, such as the rate of heat storage, lactic acid accumulation, and decreases in pH and ATP (Albertus *et al.*, 2005). Additionally, there appears to be a large psychological/perceptual component linked to the decreased capacity for work (Tucker, 2009). The nervous system ultimately regulates muscle contraction and exercise capacity in order to prevent organ system failure (due to acidosis, hypoxia, hyperthermia) (Noakes *et al.*, 2004; Lambert, Noakes and St Clair Gibson, 2005).

Another model explaining self-paced exercise which has recently received attention is the psychobiological model. It is an effort-based decision-making model centred on motivational intensity theory (Pageaux, 2014). This model posits that the conscious regulation of pace is determined by five cognitive factors, being:

1. Perception of effort
2. Potential motivation

3. Knowledge of the end-point of exercise
4. Knowledge of distance/time to the end-point
5. Previous experience of perception of effort during exercise of varying intensity and duration

The psychobiological model places greater emphasis on the motivation of the athlete (Marcora and Staiano, 2010). For example, athletes would be more motivated during an event with competitors as opposed to a testing session in a laboratory (Pageaux, 2014). It also suggests that these factors are considered consciously compared to the anticipatory regulatory model which proposes more sub-conscious decision making. According to the psychobiological model, the decision as to when to terminate exercise is taken by the conscious brain (Blanchfield *et al.*, 2013; Pageaux, 2014). Thus, the psychobiological model posits that conscious perceived exertion affects conscious voluntary control of neural activation of muscles. While the anticipatory regulatory model suggests that sensory information is first analysed sub-consciously, which then affects neural activation of muscles and perceived exertion, also sub-consciously (Marcora, 2008). As in the anticipatory regulatory model, perception of effort is a key determinant of the psychobiological model.

The Importance of RPE

Borg's (1998) rating of perceived exertion (RPE) scale was originally designed to indicate the degree of strain experienced by a participant. The scale integrates information from the peripheral working muscles and joints, the central cardiovascular and respiratory system, and the central nervous system (Borg, 1982). Recently, the central nervous system has been implicated as a crucial factor for determining the point of volitional fatigue and thus is a crucial measure for any study examining pacing strategies (Tucker, 2009).

It is proposed that RPE is a key component of the system regulating exercise and protecting the athlete from continuing exercise to the point where bodily harm may occur (Tucker, 2009). Maximal ratings of perceived exertion have been shown to coincide with several of the suggested physiological limits to exercise (Tucker, 2009). In a study conducted by Baldwin *et al.* (2003), it was found that RPE increased linearly in both glycogen depleted and loaded states at a constant work rate, however the rate

of this increase was different. Ratings of perceived exertion were the same at exhaustion, but increased faster in the glycogen depleted state (Baldwin *et al.*, 2003). Based on this evidence, Tucker (2009) concluded that the conscious perception of effort increases at a rate that will ensure a maximal tolerable RPE is reached before the limiting factor (in this case muscle glycogen content) causes disruptions in homeostasis. Tucker (2009) then suggests that RPE is not only a direct result of afferent feedback, but also plays a role in anticipatory regulation and thus, exercise duration. That is, exercise terminates when extremely uncomfortable levels of RPE are reached, corresponding to the point where potentially harmful changes in homeostasis may occur (Swart *et al.*, 2009). Furthermore, the RPE functions as part of the system which adjusts exercise intensity ensuring the athletes complete the exercise safely, but with optimal performance (Tucker, 2009; Swart *et al.*, 2009).

Limitations of the Peripheral Fatigue Model

While there are still many proponents of the peripheral fatigue model, the anticipatory regulatory and psychobiological models have recently become widely recognized as they explain many of the limitations of the peripheral fatigue model (Basset and Howley, 1997; Shephard, 2009).

According to Noakes (2011) and colleagues, several limitations to the peripheral fatigue model can be explained by anticipatory regulation. Only the anticipatory regulatory model explains the variable pacing strategies observed in all competitions, while the peripheral fatigue model predicts that athletes can only ever follow one pacing strategy (Noakes, 2011). It has been shown that athletes regulate their pacing strategies within 60 seconds of being exposed to different oxygen concentrations in the air, indicating anticipatory regulation (Amann *et al.*, 2006). However, the peripheral fatigue model suggests that in the absence of the proposed fatigue mediators (such as lactate), athletes would commence the exercise at extreme, unsustainable outputs after and would slowly decline over the duration of the race until the metabolites reach equilibrium (Noakes, 2011). This observation is associated with another phenomenon known as the 'end-spurt' where athletes increase their output toward the end of an event when they should the most fatigued. The end-spurt has been shown to be due

to increased central motor drive, supporting the predictions of the anticipatory regulation model (Ansley *et al.*, 2004).

According to the peripheral fatigue model, athletes would have an almost maximal concentration of metabolites at the point when an end-spurt might occur, thus any increase in intensity would not be possible (Noakes, 2011). Another limitation to the peripheral fatigue model is that it requires 100% activation of motor units of the exercising limbs, since activity would not stop until all the muscle fibres have been recruited (Noakes and St Clair Gibson, 2004). It is now accepted that full muscle activation does not occur during exercise, supporting the anticipatory regulatory model (Amann *et al.*, 2006; Noakes, 2011). Certain drugs which act only on the central nervous system have been shown to improve exercise performance and this contradicts the peripheral fatigue model (Noakes, 2011). The peripheral fatigue model allows only drugs which act on the heart, lungs or muscles to influence performance. A study has shown improved exercise performance after amphetamine administration (which acts solely on the central nervous system) thereby confirming the anticipatory regulatory model assumptions (Swart *et al.,* 2009).

Anticipatory Regulation and Teleo-anticipation

An extension of the central fatigue hypothesis, the teleo-anticipatory system, is a theory explaining the prevention of hazardous changes to homeostasis during exercise (Joseph *et al.*, 2008; Tucker and Noakes, 2009). First proposed by Ulmer (1996), the teleo-anticipatory system (most likely found in the central nervous system) anticipates the end-point of exercise in a complex, integrated manner, with the anticipatory pacing strategy being a key component of this system (Tucker and Noakes, 2009). Exercise intensity is then regulated and the adopted pacing strategy is altered in order to ensure that potentially hazardous changes to homeostasis do not occur (Tucker and Noakes, 2009). In more simple terms, the athlete appears to constantly compare current fatigue levels (conscious rating of perceived exertion) at any stage during an event with the expected level of fatigue at that point (template rating of perceived exertion) (Tucker, 2009). The sensations are then balanced with the template RPE formed before the exercise began (Foster *et al.*, 2009). This template RPE contains information on how the athlete aims to distribute energetic

resources over the event as well as the maximum homeostatic disturbance the athlete is willing or capable of tolerating (Joseph *et al.*, 2008). In addition, the template RPE takes into account the work output capabilities of the athlete, past experience from other similar exercise events and feedback from both central and peripheral receptors (Joseph *et al.*, 2008; Foster *et al.*, 2009). Importantly, the template RPE also factors in the required distance or duration of the exercise bout (if it is known), after which a starting pace is self-selected which is anticipated to be optimal for the exercise bout (Tucker, 2009). The conscious RPE usually takes the form of a rating indicating by the athlete, while the template RPE is a theoretical construct that cannot be measured (Tucker, 2009).

Exercise work rate during self-paced exercise is regulated by means of a combination of feedback integration, which generates the conscious RPE, and anticipatory projection, which produces the RPE template (Tucker, 2009). Thus, the template RPE provides a means of interpretation for the conscious RPE throughout the exercise bout (Tucker, 2009). In rowing, the end-point of exercise is typically known and the general pacing profile has been determined. By affecting the athlete's knowledge of the end-point, it is possible to examine the physiological and psychological factors which influence fatigue and pacing.

Deception Studies: Manipulating Knowledge of the End-point

Deception studies are aimed at investigating the theories of fatigue. An optimal pacing strategy requires precise information on exercise duration or distance. If the end-point information is unknown or incorrect, performance will be affected (Williams *et al.*, 2014). Thus, these studies are performed by providing participants with false information about the duration or distance of exercise to be completed. In so doing, the athlete's expectation and previous experience of the exercise task is modified causing the adopted pacing strategy to be suboptimal (Williams *et al.*, 2014).

Unknown Duration

When the exercise duration and distance are not known, athletes reduce their work rate and utilise physiological resources more economically (Baden *et al.*, 2005; Billaut

et al., 2011). The athlete aims to maintain a reserve in anticipation of the end-point of exercise (Williams *et al.*, 2014). If the end-point does become known, athletes increase work rate as a complete pacing strategy can be formed (Tucker, 2009). When participants expect a longer duration, RPE values are lower even when exercise intensity is the same (Baden *et al.*, 2005). Thus, the uncertainty about the end-point causes the athlete to underutilise available resources which in turn causes lower RPE values in order to avoid premature fatigue (Tucker, 2009). Athletes who have no knowledge of the exercise end-point rely on afferent feedback from the periphery to a greater extent, while incorrect expectations of the distance or duration remaining prevent the appropriate interpretation of physiological afferents (Ansley *et al.*, 2004; Tucker, 2009; Williams *et al.*, 2014). In these cases, the rate of RPE increase is controlled by incorrect feedback interpretation. This prevents peak RPE values coinciding with the end-point of exercise, ultimately resulting in underperformance (Williams *et al.*, 2014).

Deceptive Information on Exercise Duration

When the expectation of exercise end-point is manipulated, significant changes in RPE have been found (Baden *et al.*, 2005). In these types of studies, athletes perform on the basis of expected rather than actual distance and therefore tend to underperform (Paterson and Marino, 2004). This is due to the mismatch between the template RPE, set in anticipation of exercise, and the actual RPE produced by the exercise (Ulmer, 1996). Performance times vary when information provided about duration is incorrect, however this type of deception has little effect on physiological parameters such has heart rate and power output (Nikolopoulos *et al.*, 2001). In this study, athletes performed each cycling time trials at a predetermined intensity which they perceived to be optimal for the expected distance. This established that athletes perform on the basis of perceived rather than actual distance remaining, indicating central control of pacing strategies (Nikolopoulos *et al.*, 2001; Williams *et al.*, 2014).

Unanticipated Changes in Duration

The predetermined template pacing strategy can be disturbed by altering the duration of the exercise task, leading to underperformance. Thus, one method of deception

studies is to inform participants of an unexpected modification to the duration of the exercise task (Williams *et al.*, 2014). These modifications can take the form of either a reduction or extension in duration. An underutilisation of resources occurs when exercise is terminated before expected, resulting in poor performance (Baden *et al.*, 2005; Tucker, 2009). It is thought that this occurs since the end spurt phenomenon cannot be fully exploited (Williams *et al.*, 2014). Similarly, an extension of the exercise duration results in suboptimal performance because all resources allocated to the exercise would have been used before exercise was truly completed. In these studies, RPE was clearly affected, while physiological measures were not (St Clair Gibson *et al.*, 2003; Baden *et al.*, 2005). It has been hypothesised that the RPE changes may be due to negative emotions associated with the increase in exercise duration (St Clair Gibson *et al.*, 2006).

Ultimately, the inconsistency between the information provided and the athlete's expectations has been found to influence performance; however, the true effect remains unclear (Williams *et al.*, 2014). Williams and colleagues (2014) conducted a review of deception studies and concluded that there is little consistency amongst research investigating these factors. It is posited that this is primarily due to differing methodologies, diverse measures and durations of exercise. Furthermore, the review included endurance exercise, short duration maximal exercise as well as strength exercise. This calls into question the validity of the review article. Nonetheless, it is clear that deception studies do alter performance and this warrants further investigation in order to determine the exact effects of different types of deception and the resulting impact on performance.

Incorrect or Complete Absence of Feedback

It is generally accepted that accurate performance feedback is valuable for athletes during exercise and this is highlighted in the anticipatory regulatory model (Ulmer, 1996; Noakes, Lambert and St Clair Gibson, 2005). However, literature concerning this factor and its role in the selection of a pacing strategy, is limited. One study which did investigate the impact of feedback found that distance blinded cyclists who received no feedback were able to develop the same pacing profile as cyclists who received distance feedback, although this only occurred after repeating the time trial

four times (Mauger, Jones and Williams, 2009). The authors conclude that prior experience of an exercise bout is more important to the development of an optimal pacing strategy than feedback during the event. Another study provided cyclists with incorrect distance feedback and found that the deceived cyclists produced similar performance times to cyclists presented with correct feedback (Albertus *et al.*, 2005). A more recent investigation examined the impact of absence of distance feedback on untrained individuals and found that there was no difference in performance compared to participants who received distance feedback (Williams, Bailey and Mauger, 2012). Thus, it seems feedback is not a prerequisite for optimal performance, especially if athletes have completed the same exercise bout before (Williams *et al.*, 2014). Furthermore, it is posited that when relevant experience is lacking, athletes tend to rely more on internal afferent feedback, such as perceived exertion, rather than external feedback (Williams *et al.*, 2014).

Knowledge of the end-point has been shown to be a crucial factor in determining the allocation of resources during exercise (St Clair Gibson and Noakes, 2004; St Clair Gibson *et al.*, 2006). Furthermore, perceived exertion is proposed to mediate the regulation and cessation of exercise (Tucker, 2009). Anticipation of the end-point influences perceived exertion as well as muscle activity, both of which seem to be coupled to pacing strategy (Billaut *et al.*, 2011; Hettinga *et al.*, 2006). The measurement of muscle activity is based on the assumption that changes indicate alterations in neural drive (Mauger, Jones and Williams, 2009). By measuring and analysing these factors, it may be possible to elucidate the mechanisms responsible for the selection of a pacing strategy during rowing.

Several studies have examined the pacing strategies which rowers employ during elite competition (Secher, 1983; Garland, 2005; Brown *et al.*, 2010; Muehlbauer *et al.*, 2010). These studies identified that the reverse J-shape pacing profile is most often utilised at this level. However, none of these studies attempted to determine the physiological factors which contribute to the adoption and maintenance of this pacing strategy. In many sports, a consistent pacing strategy is not adopted or suitable in every situation, while in rowing the same pacing profile is adopted and the end-point is typically known. Therefore, rowing provides a unique opportunity as a context to examine pacing strategies and fatigue models. Since the general pacing strategy is known, the mechanisms by which a pacing strategy is created, adopted and

maintained can be investigated. Furthermore, the physiological and psychological factors which influence the pacing strategy may be elucidated.

Summary of Reviewed Literature

Rowing is both a physically and technically demanding sport requiring the use of all major muscles groups (Secher, 1993; Secher and Volianitis, 2007). Rowers tend to have large aerobic capacities, high proportions of lean muscle mass and are capable of producing high forces over an extended period of time (Astrand and Shephard, 2000; Secher and Volianitis, 2007). A rower's performance is regulated by pacing strategies in order to prevent premature fatigue and to ensure optimal distribution of energetic resources over the course of the exercise bout (Abbiss and Laursen, 2008). This is crucial to success in the sport. Previous research indicates that both on-water and ergometer rowers consistently adopt the reverse J-shape pacing strategy (Garland, 2005; Muehlbauer *et al.*, 2010). However, the exact mechanisms by which this occurs are yet to be clarified. There is also evidence to suggest that the J-shaped pacing strategy may not be the physiologically optimal profile during rowing (Lynch, 2012).

The physiological regulation of pacing strategies has become a widely debated topic among exercise physiologists (Basset and Howley, 1997). The dogmatic peripheral fatigue model has been shown to have several fundamental limitations, which the central governor model appears to explain, causing the latter to grow in popularity amongst experts (Noakes, 2011). Nevertheless, there are still many factors pertaining to the regulation of exercise which are yet to be elucidated (Tucker, 2009). This has resulted in a growing number of deception studies which aim to expound on the novel principles related to pacing strategies and the regulation of exercise (Williams *et al.*, 2014). These studies are carried out by deceiving participants about the duration of the exercise bout and are aimed at investigating the theory of teleo-anticipation, which is an extension of the central fatigue model (Williams *et al.*, 2014).

CHAPTER III

METHODOLOGY

RESEARCH AIM

This study aimed to investigate the pacing strategies of experienced rowers when exercise duration was unknown or was changed unexpectedly. To date, most research on pacing strategies has attempted to explain the type of pacing strategy occurring in a specific sport. The pacing strategy most often adopted in rowing has already been identified in previous research. Therefore, this study aimed to examine the characteristics of the pacing strategy when the endpoint is manipulated. While rowers are rarely without knowledge of the endpoint in realistic scenarios, rowing provides a unique simulation, allowing the study of the nature of pacing strategies and the effects of deception in a continuous sport which requires constant contraction of all major muscle groups for a prolonged period. The results from this study examining pacing strategies during rowing may be applicable to the sport itself, similar continuous sports and all sports in general where the pacing strategy is not well understood.

RESEARCH DESIGN

A within-group design was utilised with 11 participants completing three experimental trials (Table I, page **Error! Bookmark not defined.**). Each participant was required to attend four sessions, the first being an introductory session with the remaining three being the experimental trials. Each trial was separated by at least three days.

EXPERIMENTAL TRIALS

All trials took the form a 2000m self-paced rowing ergometer time trial completed within a laboratory setting. Participants were instructed to cover the distance in the shortest time possible. No feedback information was provided to participants regarding their performance or physiological measures. The experimental trials were identical in nature although the information provided prior to each trial differed. The three

experimental trials were referred to as the control trial (CT), the unknown trial (UT) and the deceptive trial (DT).

Control Trial (CT)

In this trial, participants were accurately informed of the 2000m distance to be covered. Instructions were given to perform the trial in as short a time as possible, mimicking a time trial.

Unknown Trial (UT)

During the unknown trial, participants were not informed of the distance to be completed during the trial and they were told to row as fast as possible until the researcher told them stop. Information was not given to participants at any point during the trial. Only once the 2000m distance had been covered were the participants told to stop. Thus, the trial required maximal rowing for an apparently indefinite period. This uncertainty was used to indicate whether a lack of end-point information affected the performance of the participants and whether a more suitable pacing strategy was adopted or not. Upon conclusion of the trial, participants signed a confidentiality declaration to prevent information being shared with other participants in the study.

Deceptive Trial (DT)

At the beginning of the deceptive trial participants were incorrectly informed that only 1000m was to be completed. As the participant reached the 1000m distance the researcher instructed a further 1000m. Deception was used to evaluate the impact of incorrect knowledge of the end-point on performance. Furthermore, deception was used to assess how pacing may be altered when the end-point of exercise is shifted. Another confidentiality declaration was signed following this trial to ensure participants did not share information with others participating in the study.

Order of Trials

The control trial was the first experimental condition completed. This was done for all participants and the trial served as a reference point. The participants were then randomly assigned to performing either the deceptive trial followed by the unknown trial, or the unknown trial followed by the deceptive trial. Thus, there were two possible trial orders: CT; UT; DT or CT; DT; UT. Randomisation was achieved by allowing a blinded external individual to pick one of the trial orders for each participant. The randomisation process ensured a learning effect did not occur. Although participants signed confidentiality declarations (appendix A, page 105), randomisation also functioned to minimise the effect of communication between participants by reducing the chance of participants anticipating the workload.

Selection of the Exercise Task

The majority of competitive rowing events are completed over a distance of 2000m, for example at the Olympic Games (Lynch, 2012). Consequently, much research has utilised the distance of 2000m (Schabort *et al.*, 1999; Ingham *et al.*, 2002; Garland, 2005; Brown *et al.*, 2010; Gallagher *et al.,* 2010; Muehlbauer *et al.*, 2010; Bell *et al.,* 2013). Therefore, a distance of 2000m was selected for direct comparison to previous studies as well as for practical relevance. Additionally, this is not a distance unfamiliar to experienced rowers. This is significant since pacing strategies are optimised with experience of the exercise distance/duration, in the form of a more accurate anticipatory RPE template (Tucker, 2009).

ETHICS

Ethical approval was granted by the Department of Human Kinetics and Ergonomics Ethics Committee, Rhodes University (application reference number: HKE-2015-3). Although the deception of athletes is anaccurate way of studying pacing strategies, this should be undertaken with extreme caution. In the current study, if participants were correctly informed of the exercise end-point, the results would be invalid as they would have been able to anticipate the exercise requirements and thus adopt a typical

pacing strategy. Therefore, if participants had complete knowledge of the hypotheses and methodology of this study, it would have rendered the results null and void.

In the current study, several measures were employed to ensure the safety and well-being of the participants. The participants were experienced rowers meaning that they had a good understanding of the nature and requirements of the sport. They were highly trained in rowing and were at peak fitness at the time of testing. The protocol design resulted in the same distance being completed in each trial. Furthermore, the distance was that of a typical rowing race and participants would have completed far greater distances during training. All the measures aimed to ensure that participants were able to complete all three trials without great difficulty and without a significant a risk of injury.

Additionally, data collected from the deceptive trials were not used until participants had been fully informed of the deception. Following this, participants were asked if their data may be used and if they agreed, only then did analyses begin. This process was completed by using a letter of information (appendix A, page 99), pre-trial consent form (appendix A, page 103), post-protocol information letter (appendix A, page 107) and a post-trial consent form (appendix A, page 109).

The order of the information given to participants was:

1. A pre-protocol information letter was given to potential participants if interest was shown in the study (appendix A, page 99).
2. A verbal explanation of the research was then provided to potential participants.
3. Participants were then required to sign a pre-trial consent form (appendix A, page 103).
4. Following the unknown and deceptive trials, participants were required to sign confidentiality declaration forms (appendix A, page 105) in order to ensure that the nature of the deception was not shared between participants.
5. Once participants had completed all three experimental trials, a post-consent form was signed (appendix A, page 109).
6. A post-protocol information letter (appendix A, page 107) was also provided at this time.

TESTING ENVIRONMENT

All trials were completed in the Department of Human Kinetics and Ergonomics, Rhodes University, Grahamstown, South Africa on a Concept II rowing ergometer (Model D, Morrisville, USA) fitted securely to the floor to prevent movement and slipping during the trials. The ergometer monitor, which was placed at the participant's head height, was used to provide feedback to the researcher during the course of each trial, while the participant was not allowed to see the monitor. The testing room was well ventilated and the temperature was kept constant at 15 - 25°C at the time of testing. Testing was postponed if the temperature was outside this range. No verbal encouragement was given to participants in order to maintain consistency between trials.

PARTICIPANT SELECTION AND INCLUSION CRITERIA

Participants were recruited from the Rhodes University Rowing Club. Only males between the ages of 18-25 years of age were recruited for the study. All participants were, at the time of testing, following the same training programme and thus were of similar in-season training status. Participants with no recent history of injury (six months) or illness (one month) were recruited.

PILOT TESTING

A pilot test was performed to ensure that all equipment was functioning correctly. Specific analyses of the variables to be measured were then carried out. These analyses were performed to assess whether the 2000m trial was in fact eliciting true pacing responses. Pilot testing was performed on two male university rowers who were not recruited for the study. The pilot tests verified that the rowers adopted a J-shaped or reverse J-shaped pacing profile during a standard 2000m ergometer trial. Finally, the pilot testing ensured that the researcher was familiar with all equipment and procedures to be used in the current investigation.

DEPENDANT AND INDEPENDANT VARIABLES

Dependant Variables

The measures used to analyse the possible changes in pacing were total time as well as time intervals (recorded at 500m splits), heart rate, muscle recruitment, central ratings of perceived exertion and power output. All measures were recorded during each trial.

Independent Variables

The independent variable in this study was the accuracy of the exercise end-point information provided to the participants during each trial.

MEASUREMENTS AND EQUIPMENT

Rowing Ergometer

All tests were performed on the Concept II Model D Rowing Ergometer (Morrisville, USA) which provides a fixed resistance and requires no calibration. The participants were familiar with this type of ergometer and had all used it for training and performance assessments. The ergometer was set to a drag factor 125 before every trial. This drag factor was chosen as it accurately represents that of on-water rowing and was consistently used by the participants during training. Furthermore, the Concept II Ergometer has been shown to be highly reliable when compared to on-water rowing, particularly when combined with a 2000m test protocol (Schabort *et al.*, 1999). Finally, prior to testing the ergometer was checked for cleanliness and functionality. To prevent slippage of the ergometer, tape was wrapped around the base to secure it to the floor.

ANTHROPOMETRIC PROCEDURES

Stature

Stature was measured to the nearest millimetre using a Harpenden stadiometer (Holtain Ltd, Crymych, United Kingdom). Before the measurement was taken, participants were asked to remove shoes and socks. The participants were positioned

with the heels and shoulders against the stadiometer and told to stare directly forward, after which the measurement was taken. All measurements were taken by the same researcher. This process ensured standardisation amongst the measurements.

Body Mass

A calibrated Toledo® electronic scale (Model 8142, Port Melbourne, Australia) was used to measure body mass to the nearest gram. The scale was calibrated prior to the measurement. Participants removed all clothing and accessories, excluding the rowing tri suit. The tri suit mass was considered negligible. The participants were then told to stand still on the centre of the scale. Once the digital monitor remained unchanged for 3 seconds, the value was recorded.

Body Mass Index

Body mass index was calculated using the equation:

$$\text{BMI } (kg.m^{-2}) = \text{Mass } (kg) \div \text{Stature}^2 \ (m)$$

PHYSIOLOGICAL MEASURES

Heart Rate

Heart rate was recorded using a Polar FT1 Heart Rate Monitor, (©Polar Electro, Kempele, Finland). This was to ensure reference values were recovered following the warm-up and preceding the start of the test. Heart rate served as an indicator of exercise intensity during the testing, providing evidence as to whether the participant was performing maximally or not. The heart rate belt was coated with aqueous conductive gel and then placed at the level of the 4^{th} and 5^{th} intercostal space, just above the xiphoid process. The belt was then tightened to ensure accurate monitoring.

Electromyography and Muscle Activity

The ME6000 Megawin Biomonitor (Mega Electronics Ltd, Kuopio, Finland) was used to record muscle activity in four muscles. The muscles selected were biceps femoris, vastus medialis, erector spinae and medial deltoid. These muscles were selected

based on previous research examining EMG during rowing (Peltonen *et al.*, 1997; Turpin *et al.*, 2011; Fleming *et al.*, 2014).

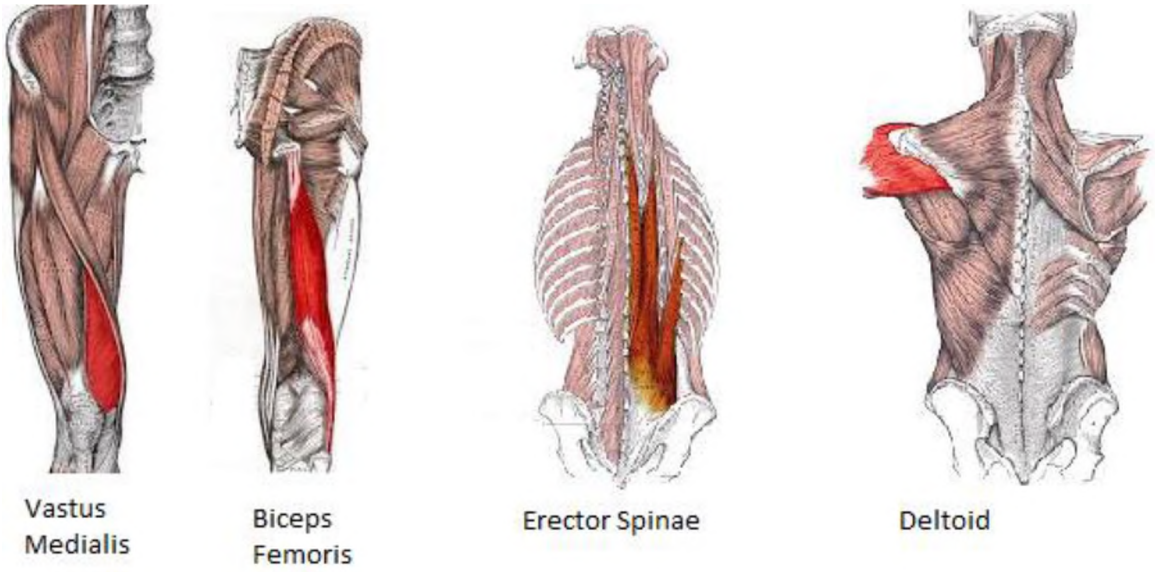

Figure 3: Muscles to be analysed via EMG (images from http://www.wikipedia.org).

The Megawin uses analogue channels which were attached to the muscles. Four channels were used to measure each of the four muscles, while a single digital channel was attached to an inactive muscle. Participant's hair was shaved using a disposable razor and wiped with an alcohol swab. This ensured a clean patch of skin where the electrodes were to be placed, to prevent interference with the EMG signal. Alcohol swabs were then used to wipe these areas in order to prevent infection and irritation to the skin as well as minimise interference with the electrodes. The EMG electrodes were placed on the belly of the muscles and it was ensured that the electrodes were well secured with Elastoplast tape before testing began. The placement of electrodes also ensured that crosstalk was minimised and that the wires did not touch. Participants were required to perform 10 maximal rowing strokes, for normalisation and calibration of the EMG measures during the experimental trials. Once the testing began, information from the EMG system was captured on a laptop using the MegaWin Software (Mega Electronics Ltd, Kuopio, Finland). The sampling frequency was set at 1000 Hz. Analysis of the raw EMG data was performed by using a data reduction tool. Data Analysis Version 4.08 (Goebel, Human Kinetic and Ergonomics Department, Rhodes University, Grahamstown, South Africa) was used to calculate average EMG of each muscle.

PERCEPTUAL MEASURES

Central Ratings of Perceived Exertion (RPE)

The Borg scale ranges from a value of 6 to 20, with 6 being minimal exertion and 20 being maximal exertion/exhaustion (appendix B, page 113). It is purely a subjective measure, although the ratings do provide a good indication of the exertion of the participants. Central RPE was therefore used as a subjective indication of the exercise fatigue experienced by participants, while also allowing comparisons between trials as to whether different end-point information may produce different RPE responses. RPE was recorded every 500m during the 2000m trial.

PERFORMANCE MEASURES

Total Time

The total time to complete the 2000m trial was recorded as an absolute measure of performance. This allowed comparison to previous trials performed by the participants and provided an indication of the intensity with which the trial was being completed. Further, the time taken to complete each 500m was recorded and used to identify whether an alternative pacing strategy was adopted or whether the classic J-shaped pacing profile was utilised. The time data was measured using the Concept II Ergometer Monitor and then recorded onto a laptop.

Power Output

The average power output (watts) of the participants was measured continuously on the Concept II Ergometer Monitor. This provided further information of the pacing strategy of the rowers. Additionally, when combined with the EMG data, power output identified differences in muscle recruitment between the trials. Average power output was recorded every 500m for each trial. This value was obtained from the monitor on the rowing ergometer.

EXPERIMENTAL PROCEDURES

All experimental sessions were held in the Department of Human Kinetics and Ergonomics, Rhodes University, Grahamstown, South Africa. It was made clear to participants that on the day of testing, a small meal should be eaten 2 hours prior to the test. Participants were asked not to consume alcohol, medication or participate in any heavy training 24 hours prior to the test as these activities may affect the results. These requests were made to ensure participants came to each trial in a similar nutritional and pre-exercise state. Upon arrival at the testing venue, participants were asked again whether they had compiled with the above requests and if not, another testing session was scheduled.

Session 1: Habituation and Introduction

The first session enabled the researcher to provide background information to the investigation, which was partially accomplished through a pre-protocol information letter (appendix A, page 99). Participants were provided with a letter of information fully explaining the requirements of the study and the risks involved. A consent form was then completed by each participant, after which stature and mass were measured and body mass index was calculated and recorded as previously described (appendix A, page 103). Following this, the participants were familiarised with the equipment and scales to be used in the experimental sessions. In particular, the Borg RPE scale was clearly explained to each participant (appendix B, page 113). It was ensured that the participants fully understood the scale as well as the respective values and verbal anchors. In addition, pictures and explanations were used to familiarise participants with the EMG electrode placement and the experimental trial protocol.

Session 2, 3 and 4: Experimental Trials

Participants were asked to dress in the clothing which would typically be worn for a race or performance assessment, namely a Spandex tri-suit. The heart rate monitor was attached to obtain resting heart rate. The areas for the EMG electrode were shaved using a disposable razor and wiped with a Kendall Webcol alcohol swab to ensure a clean patch of skin. The athletes were then allowed to perform a self-directed,

low intensity warm-up which consisted of light rowing as well as dynamic stretching of the participant's choice. Participants were advised to warm-up in the same manner as if a race were about to be completed. A rest period was then observed allowing heart rate to return to within 10 beats.min^{-1} of the reference value, after which the experimental trial began. All participants completed the control trial as the first experimental session. Randomisation of the order of the consecutive trials was carried out. Following each experimental trial, participants signed a confidentiality declaration to prevent the nature of the deception being shared between participants. All trials resulted in 2000m being rowed, however the type of end-point information provided was different. Heart rate, RPE, power output and time were recorded every 500m. A post-consent form was signed by participants indicating that deception had occurred and that participants allowed the analyses of the data. Following the final trial participants also received a post-trial information letter (appendix A, page 109).

STATISTICAL ANALYSES

Descriptive statistical analyses were performed in order to quantitatively determine the main features of the data. Following this, inferential statistics were performed. Statistica software (STATISTICA© Version 12.0, StatSoft Inc, 2014) was used to perform both the descriptive and inferential statistics. A one way analysis of variance (ANOVA) with repeated measures for the three trials was used to compare the responses between each trial. A confidence interval of 95% was set. In addition, Tukey post hoc analyses were performed on the data to determine where the significance was located. Cohen's d was calculated and used to indicate effect size where significance was found. The effect size was calculated using pooled standard deviations, allowing comparisons between different conditions. Preference was given to conventional statistics when interpreting results, while effect size was used to indicate the practical importance of the result. If d was calculated to be less than 0.2 it was considered a trivial difference, between 0.2 and 0.5 was considered a small effect size, between 0.5 and 0.8, a medium effect size and greater than 0.8 was considered a large effect size. The smallest worthwhile change was calculated for the control trial. This value was obtained by multiplying the interindividual standard deviation by 0.2 as per the effect size interpretation.

CHAPTER IV

RESULTS

INTRODUCTION

The results are presented as mean values accompanied by the corresponding standard deviation as well as the effect size (d) where appropriate. Performance time data is presented first followed by psychophysical and physiological measures.

Eleven experienced rowers agreed to participate in the study. The descriptive statistics of the participants are shown in Table (page 38).

Limitations and Considerations

When interpreting these results, it is important to consider certain factors. Power output was measured in such a manner that the findings presented represent average power output for the whole trial being rather than for each 500m interval. Power output is only compared to power output because it was measured differently to the other variables. Thus, comparing power output to the other variables would be inappropriate. . Hence, conclusions drawn regarding power output are done so tentatively and comparisons are made between power output data of each trial rather than comparing power output to other measures.

Additionally, rowing ability varied substantially between participants and all participants were below the elite level. Participants were young (mean age of 22 years) and had not have been exposed to research testing in the laboratory. This may have impacted their responses despite extensive habituation. While all participants were similarly trained and following the same training programme, the pool of rowers from which the sample was drawn was small and skill level may have varied substantially. This is due to participants experiencing different amounts of coaching and had mixed amounts of experience in the sport. The high degree of variation within some measures may be indicative of this. Lastly, it is important to recognize that individuals respond to exercise differently (Bouchard and Rankinen, 2001). Thus, variation may not necessarily be due to the small sample, but rather due to the way in which

individuals responded to the different conditions. The results should therefore be interpreted within the context of these limitations.

BASELINE MEASURES

Table I: Mean anthropometric and demographic data

MEASURE	MEAN	SD	CV (%)
Age (years)	22	2	7.50
Years of Rowing (years)	4	2	61.59
Races Completed	19	16	83.80
Stature (m)	1.80	0.10	5.46
Mass (kg)	79.68	9.25	11.61
BMI ($kg.m^{-2}$)	24.49	1.86	7.59

BMI = Body Mass Index, SD = Standard Deviation, CV = Coefficient of Variation

Noteworthy is the high coefficient of variation for both the number of years participants had rowed for as well as for the number of races completed. This was to be expected since the number of events varies from year to year at University level. Table II (page 38) also indicates a low variability for age, stature, mass and BMI.

PERFORMANCE TIMES

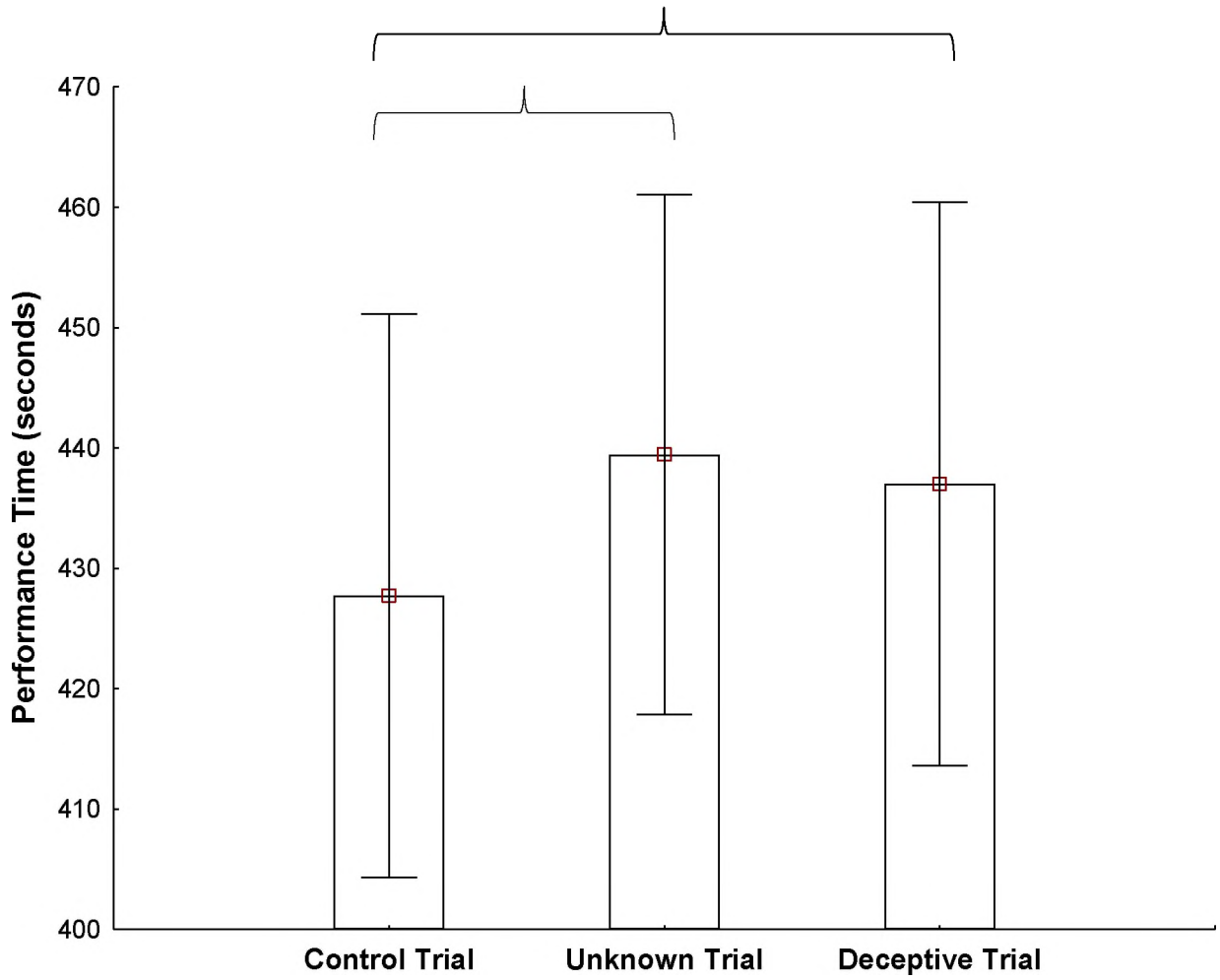

Figure 4: Mean cumulative performance times (seconds) for all three trials. ⌐¬ denotes a significant difference ($p<0.05$) between the corresponding trials.

With respect to total performance times during each trial, the control trial (427.73 ±34.81s, SWC = 7.0s) was significantly ($p<0.05$; d = 0.35 and 0.27 respectively) faster than both the unknown trial (439.45 ±32.18s) and deceptive trial (437.00 ±34.86s). There was no significant difference between the unknown trial and deceptive trial .

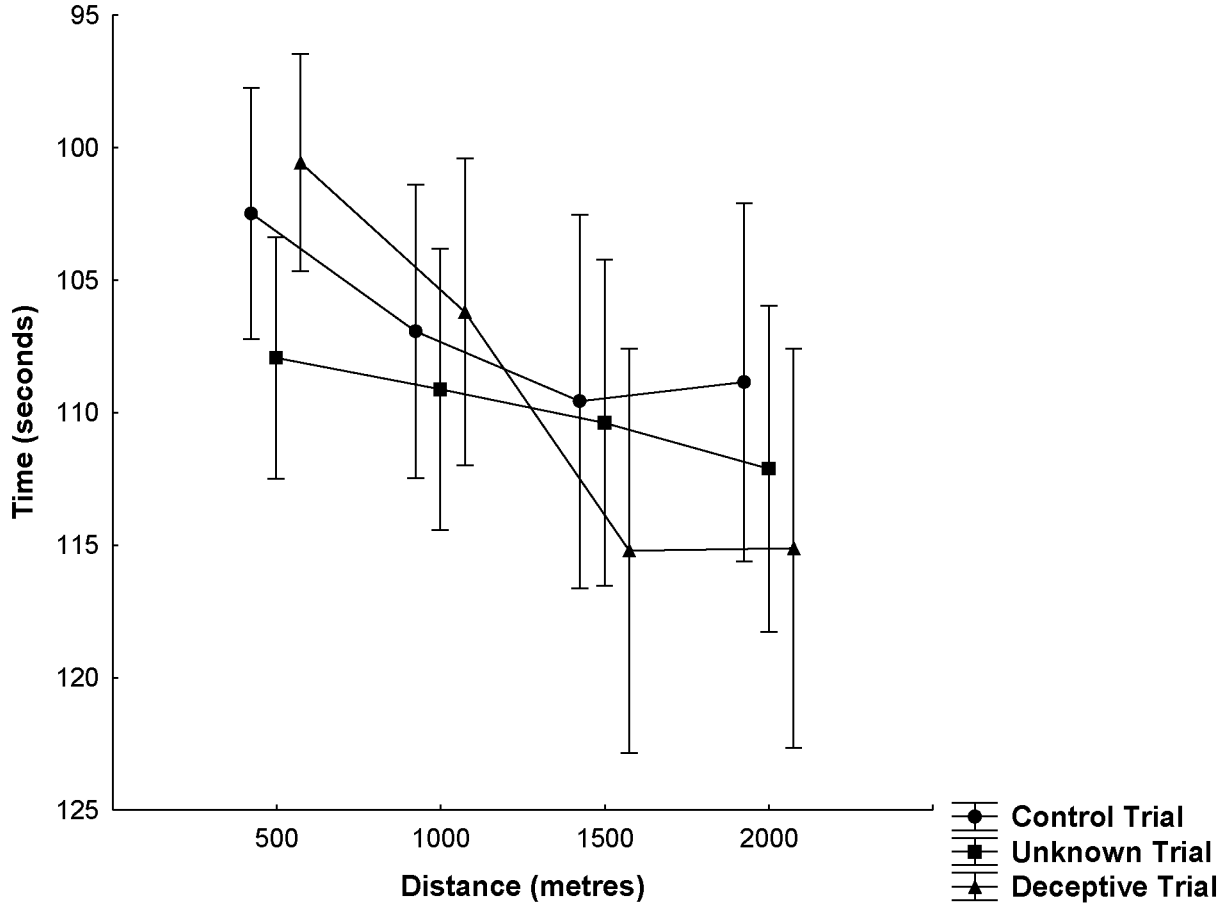

Figure 5: Mean performance time of each distance interval for each condition.

Analysis of the performance time data revealed that the first 500 metres of each condition was the fastest. The deceptive trial had the fastest and the slowest split amongst all trials, the fastest being the first 500m (100.55 ±6.11s) and the slowest being the 1500m (115.18 ±11.45s) split. Notably, the slowest split for the unknown trial was at 2000m (112.10 ±9.16s), while for the control (109.55 ±10.50s) and deceptive (115.18 ±11.35s) trials it at 1500m.

Table I: Tukey Post Hoc analysis of performance times data at 500m intervals.

Measure	CT 500m (102,45s)	CT 1000m (106,91s)	CT 1500m (109,55s)	CT 2000m (108,82s)	UT 500m (107,91s)	UT 1000m (109,09s)	UT 1500m (110,36s)	UT 2000m (112,09s)	DT 500m (100,55s)	DT 1000m (106,18s)	DT 1500m (115,18s)	DT 2000m (115,09s)
CT 500m		0,030817	0,000141	0,000313	0,002724	0,000202	0,000124	0,000123	0,924952	0,136089	0,000123	0,000123
CT 1000m	0,030817		0,612662	0,924952	0,999631	0,836065	0,217238	0,005408	0,000313	0,999984	0,000123	0,000123
CT 1500m	0,000141	0,612662		0,999984	0,974004	1,000000	0,999948	0,662053	0,000123	0,250825	0,001721	0,002163
CT 2000m	0,000313	0,924952	0,999984		0,999852	1,000000	0,983096	0,287582	0,000123	0,612662	0,000313	0,000387
UT 500m	0,002724	0,999631	0,974004	0,999852		0,998246	0,709876	0,055787	0,000130	0,961620	0,000133	0,000136
UT 1000m	0,000202	0,836065	1,000000	1,000000	0,998246		0,996577	0,415875	0,000123	0,463424	0,000562	0,000690
UT 1500m	0,000124	0,217238	0,999948	0,983096	0,709876	0,996577		0,961620	0,000123	0,055787	0,013230	0,016431
UT 2000m	0,000123	0,005408	0,662053	0,287582	0,055787	0,415875	0,961620		0,000123	0,000854	0,370335	0,415875
DT 500m	0,924952	0,000313	0,000123	0,000123	0,000130	0,000123	0,000123	0,000123		0,001721	0,000123	0,000123
DT 1000m	0,136089	0,999984	0,250825	0,612662	0,961620	0,463424	0,055787	0,000854	0,001721		0,000123	0,000123
DT 1500m	0,000123	0,000123	0,001721	0,000313	0,000133	0,000562	0,013230	0,370335	0,000123	0,000123		1,000000
DT 2000m	0,000123	0,000123	0,002163	0,000387	0,000136	0,000690	0,016431	0,415875	0,000123	0,000123	1,000000	

Approximate Probabilities for Post Hoc Tests Error: Within MSE = 8,5240, df = 60,000. Mean times presented in brackets, significance highlighted in red.

Within Conditions

The results of the post hoc analysis indicate that the 500m split was significantly (p<0.05; d = 0.58, 0.79 and 0.73) faster than all other splits in the control trial. However, this was the only significance found in the control trial. Interestingly, the 1500m split was the slowest during the control trial and performance time improved over the last 500m (2000m split). While there were no significant differences found between the splits of the unknown trial, a moderate effect size (d = 0.52) was found between the 500m split and the 2000m split. Performance time increased over the course of the trial in an approximately linear fashion, resulting in the fastest split being the 500m split and 2000m the slowest. The deceptive trial showed marked variation between each split. The 500m split was significantly (p<0.05; d = 0.75, 1.60 and 1.61 respectively) faster than the 1000m, 1500m and 2000m splits. In addition, the 1000m split was significantly (p<0.05; d = 0.89 and 0.89 respectively) faster than the 1500m and 2000m splits. The trial had a very fast start and first 1000m. However, there was a large reduction in speed over the last half of the trial, after the participants were informed that another 1000m was to be completed.

Between Conditions

Analysis of the 500m interval revealed a significantly (p<0.05; d = 0.79 and 1.14 respectively) increased unknown performance time (107.91 ±6.77s) compared to the control (102.45 ±7.05s) and deceptive (100.55 ±6.11s) performance times. While both the control (106.91 ±8.23s) and deceptive (106.18 ±8.61s) trials were faster than the unknown (109.10 ±7.89) trial at the 1000m interval, only the deceptive trial was significantly faster (p<0.05; d = 0.35). At the 1500m interval, the deceptive (115.18 ±11.35s) trial had the slowest time of all conditions, with the unknown (110.36 ±9.16s) and control (109.55 ±10.49s) trials being significantly faster (p<0.05; d = 0.52 and 0.47 respectively). The control trial (108.82 ±10.06s) was significantly (p<0.05; d = 0.59) faster than the deceptive (115.10 ±11.19s) condition at the 2000m interval.

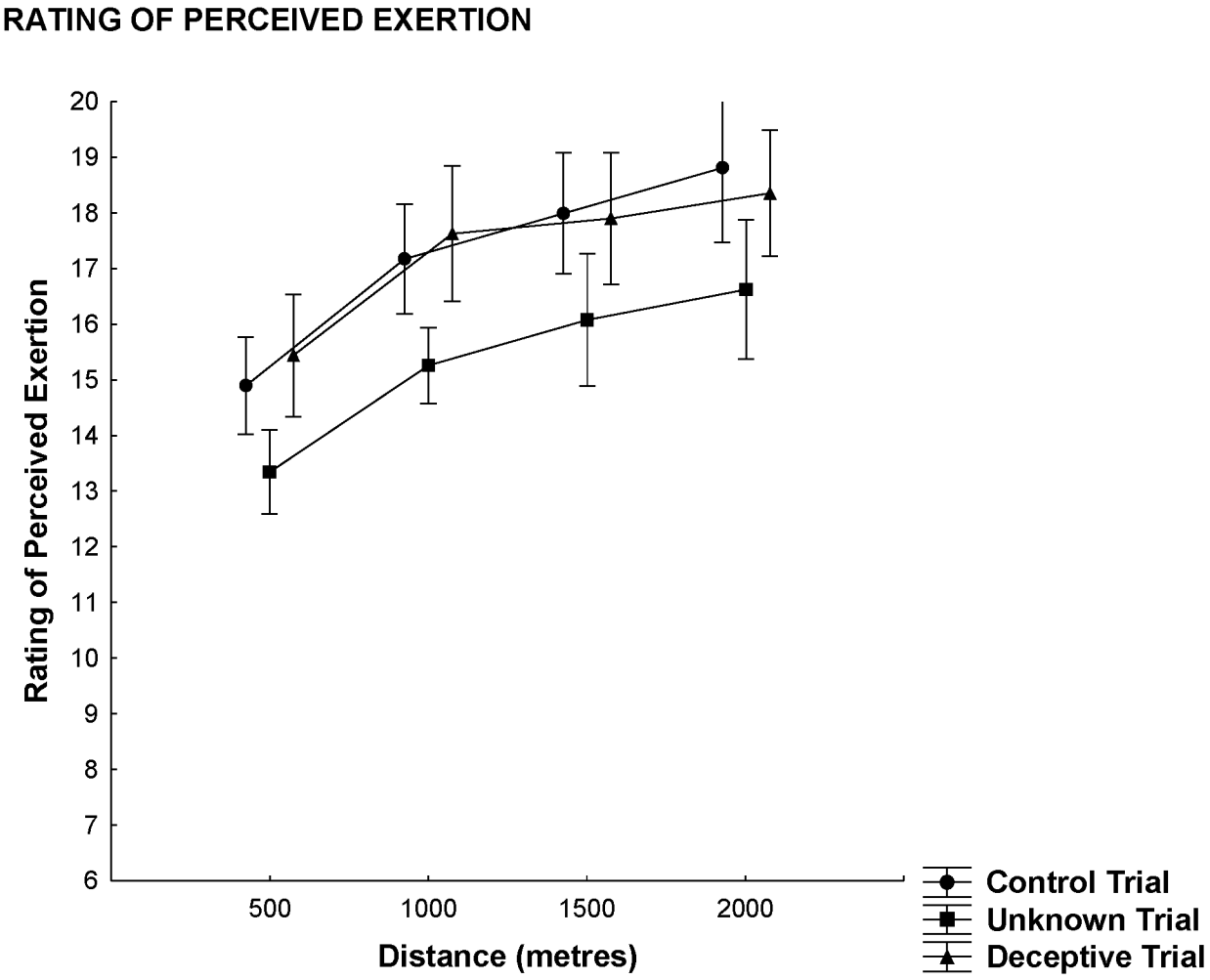

Figure 6: Mean Central RPE at each distance interval for each condition.

Table III: Tukey post hoc analysis of RPE data at 500m intervals for each condition.

Measure	CT 500m (14,91)	CT 1000m (17,18)	CT 1500m (18,00)	CT 2000m (18,82)	UT 500m (13,36)	UT 1000m (15,27)	UT 1500m (16,09)	UT 2000m (16,64)	DT 500m (15,46)	DT 1000m (17,64)	DT 1500m (17,91)	DT 2000m (18,36)
CT 500m		0,000135	0,000123	0,000123	0,010666	0,998576	0,129967	0,002527	0,960885	0,000123	0,000123	0,000123
CT 1000m	0,000135		0,633684	0,005221	0,000123	0,000599	0,213978	0,960885	0,002527	0,990157	0,781003	0,129967
CT 1500m	0,000123	0,633684		0,633684	0,000123	0,000123	0,000599	0,040649	0,000123	0,998576	1,000000	0,998576
CT 2000m	0,000123	0,005221	0,633684		0,000123	0,000123	0,000123	0,000154	0,000123	0,129967	0,475464	0,990157
UT 500m	0,010666	0,000123	0,000123	0,000123		0,000599	0,000123	0,000123	0,000201	0,000123	0,000123	0,000123
UT 1000m	0,998576	0,000599	0,000123	0,000123	0,000599		0,633684	0,040649	0,999999	0,000127	0,000123	0,000123
UT 1500m	0,129967	0,213978	0,000599	0,000123	0,000123	0,633684		0,960885	0,893618	0,010666	0,001192	0,000135
UT 2000m	0,002527	0,960885	0,040649	0,000154	0,000123	0,040649	0,960885		0,129967	0,330498	0,074512	0,002527
DT 500m	0,960885	0,002527	0,000123	0,000123	0,000201	0,999999	0,893618	0,129967		0,000154	0,000124	0,000123
DT 1000m	0,000123	0,990157	0,998576	0,129967	0,000123	0,000127	0,010666	0,330498	0,000154		0,999908	0,781003
DT 1500m	0,000123	0,781003	1,000000	0,475464	0,000123	0,000123	0,001192	0,074512	0,000124	0,999908		0,990157
DT 2000m	0,000123	0,129967	0,998576	0,990157	0,000123	0,000123	0,000135	0,002527	0,000123	0,781003	0,990157	

Approximate Probabilities for Post Hoc Tests Error: Within MSE = ,84545, df = 60,000. Mean RPE presented in brackets, significance highlighted in red.

Within Conditions

The control condition exhibited a low RPE at the 500m interval (14.91 ±1.30). All other intervals were significantly (p<0.05; d = 2.22, 2.67 and 2.80 respectively) higher than the 500m interval. Furthermore, the 1000m interval (17.18 ±1.47) was significantly (p<0.05; d = 0.93) lower than the 2000m interval (18.82 ±1.99). Notably, the 2000m interval of the control trial had the highest RPE value of all the intervals in all trials. A gradual increase in RPE was seen over the course of the unknown trial, with the highest value occurring at the 2000m interval. The 500m interval (13.36 ±1.12) was rated the lowest of all conditions and was significantly (p<0.05; d = 1.79, 1.86 and 2.14 respectively) lower than the other intervals of the unknown trial. The deceptive condition had a 500m interval of 15.45 ±1.63 which was significantly (p<0.05; d = 1.27, 1.45 and 1.75 respectively) lower than all the other intervals of the condition. Additionally, the 1000m interval was significantly different from the 2000m interval (p<0.05; d = 0.41).

Between Conditions

At the 500m interval, the unknown trial was perceived as significantly (p<0.05; d = 1.28 and 1.50 respectively) less effort than the control and deceptive trial. The subsequent intervals followed the same pattern as the first. The 1000m interval of the unknown trial was again significantly (p<0.05; d = 1.51 and 1.62 respectively) lower than the control and deceptive trials. The 1500m interval of the unknown trial was significantly (p<0.05; d = 1.13 and 1.03 respectively) lower than the control and deceptive trials. The 2000m interval of the unknown trial was also significantly (p<0.05; d = 1.13 and 0.97 respectively) lower than the control and deceptive conditions. The highest value was recorded at the 2000m interval of the control trial (18.82 ±1.99).

HEART RATE

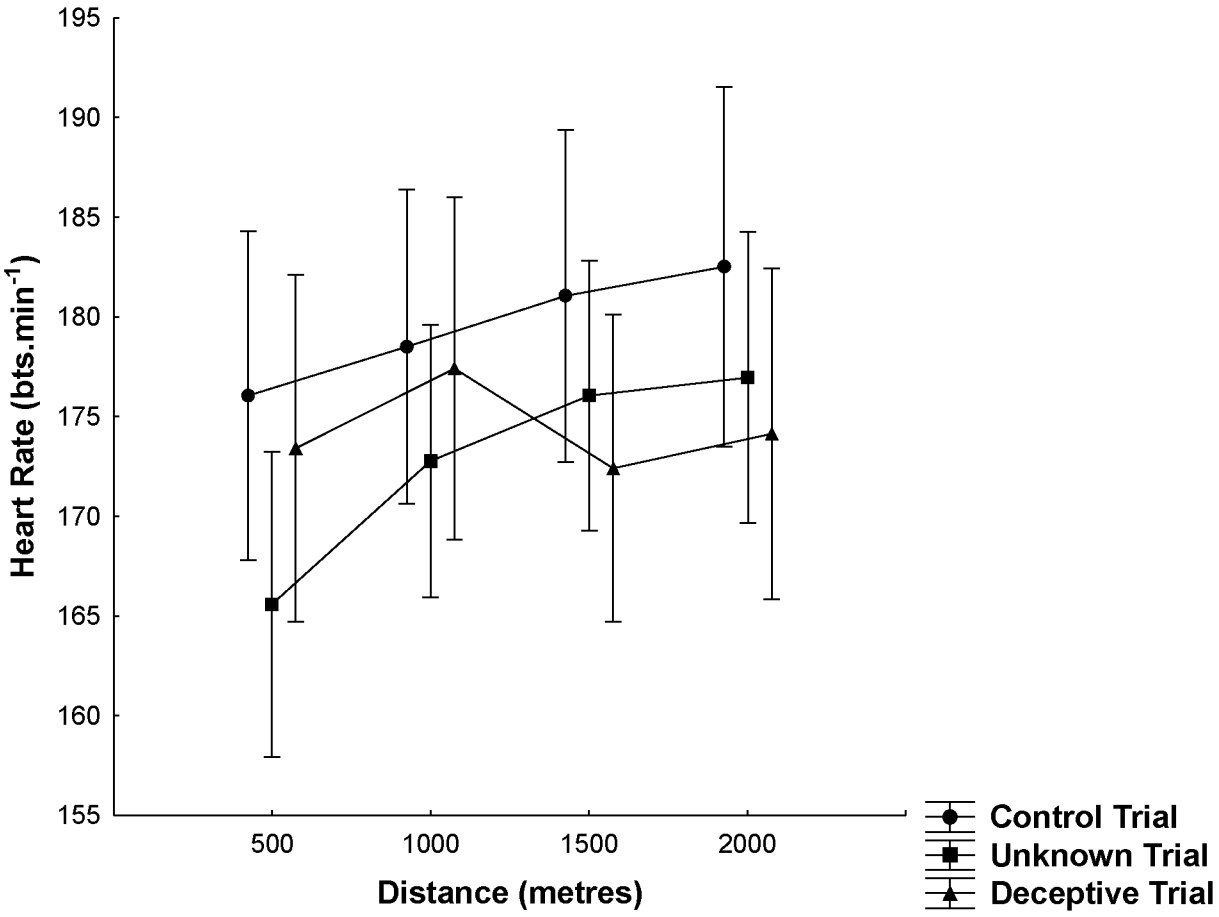

Figure 7: Mean heart rate at each distance interval for each condition.

Table III: Tukey post hoc analysis of heart rate data for each interval of each condition.

Measure	CT 500m (176,09)	CT 1000m (178,55)	CT 1500m (181,09)	CT 2000m (182,55)	UT 500m (165,64)	UT 1000m (172,82)	UT 1500m (176,09)	UT 2000m (177,00)	DT 500m (173,45)	DT 1000m (177,45)	DT 1500m (172,45)	DT 2000m (174,18)
CT 500m		0,584854	0,002599	0,000142	0,000123	0,175627	1,000000	0,999647	0,475831	0,987117	0,084112	0,870668
CT 1000m	0,584854		0,529974	0,036508	0,000123	0,000390	0,584854	0,966889	0,002021	0,998067	0,000195	0,014611
CT 1500m	0,002599	0,529974		0,978792	0,000123	0,000123	0,002599	0,029226	0,000123	0,084112	0,000123	0,000126
CT 2000m	0,000142	0,036508	0,978792		0,000123	0,000123	0,000142	0,000588	0,000123	0,002021	0,000123	0,000123
UT 500m	0,000123	0,000123	0,000123	0,000123		0,000124	0,000123	0,000123	0,000123	0,000123	0,000128	0,000123
UT 1000m	0,175627	0,000390	0,000123	0,000123	0,000124		0,175627	0,023300	0,999990	0,007053	1,000000	0,987117
UT 1500m	1,000000	0,584854	0,002599	0,000142	0,000123	0,175627		0,999647	0,475831	0,987117	0,084112	0,870668
UT 2000m	0,999647	0,966889	0,029226	0,000588	0,000123	0,023300	0,999647		0,102106	1,000000	0,009020	0,373250
DT 500m	0,475831	0,002021	0,000123	0,000123	0,000123	0,999990	0,475831	0,102106		0,036508	0,999141	0,999960
DT 1000m	0,987117	0,998067	0,084112	0,002021	0,000123	0,007053	0,987117	1,000000	0,036508		0,002599	0,175627
DT 1500m	0,084112	0,000195	0,000123	0,000123	0,000128	1,000000	0,084112	0,009020	0,999141	0,002599		0,929437
DT 2000m	0,870668	0,014611	0,000126	0,000123	0,000123	0,987117	0,870668	0,373250	0,999960	0,175627	0,929437	

Approximate Probabilities for Post Hoc Tests Error: Within MSE = 7,1136, df = 60,000. Mean heart rate (bts.min^{-1}) presented in brackets, significance highlighted in red.

Within Conditions

During the control trial, heart rate progressively increased at each interval. At the 500m interval (176.10 ±12.26 bts.min^{-1}), heart rate was significantly lower than the 1500m (181.10 ±12.40 bts.min^{-1}) and 2000m (182.55 ±13.42 bts.min^{-1}) intervals (p<0.05; d = 0.40 and 0.50 respectively). Furthermore, the 1000m (178.55 ±11.70 bts.min^{-1}) interval was significantly lower than the 2000m trial (p<0.05; d = 0.32). The highest mean heart rate of any interval was obtained in the control condition at the 2000m interval. The unknown trial had the lowest mean heart rate of any interval. An increase in heart rate was seen over the course of the trial, similar to that of the control trial, albeit with lower values. The 500m interval (165.64 ±11.39 bts.min^{-1}) was significantly lower than all the other intervals (p<0.05; d = 0.67, 0.97 and 1.02 respectively). The 1000m interval (172.82 ±10.07 bts.min^{-1}) was also significantly (p<0.05; 0.40) lower than the 2000m interval (177.00 ±10.85 bts.min^{-1}). The deceptive condition resulted in erratic heart rate changes at each interval, with only the 1000m interval (177.45 ± bts.min^{-1}) being significantly higher (p<0.05; d = 0.41) than the 1500m interval (172.45 ±11.47 bts.min^{-1}).

Between Conditions

Mean reference heart rate for each of the conditions was between 65 and 70 bts.min^{-1}. At the 500m interval, heart rate during the unknown trial was significantly lower than during the control or deceptive trial (p<0.05; d = 0.88 and 0.64 respectively). A similar trend occurred at the 1000m interval, where both the control and deceptive trial was significantly higher than the unknown trial (p<0.05; d = 0.52 and 0.40 respectively). However, at the 1500m interval, a change is seen and the control trial heart rate is significantly higher than both the unknown and deceptive trials (p<0.05; d = 0.44 and 0.72 respectively). The same effect occurred at the 2000m interval where heart rate in the control was significantly higher than in the unknown and deceptive conditions (p<0.05; d = 0.46 and 0.65 respectively).

POWER OUTPUT

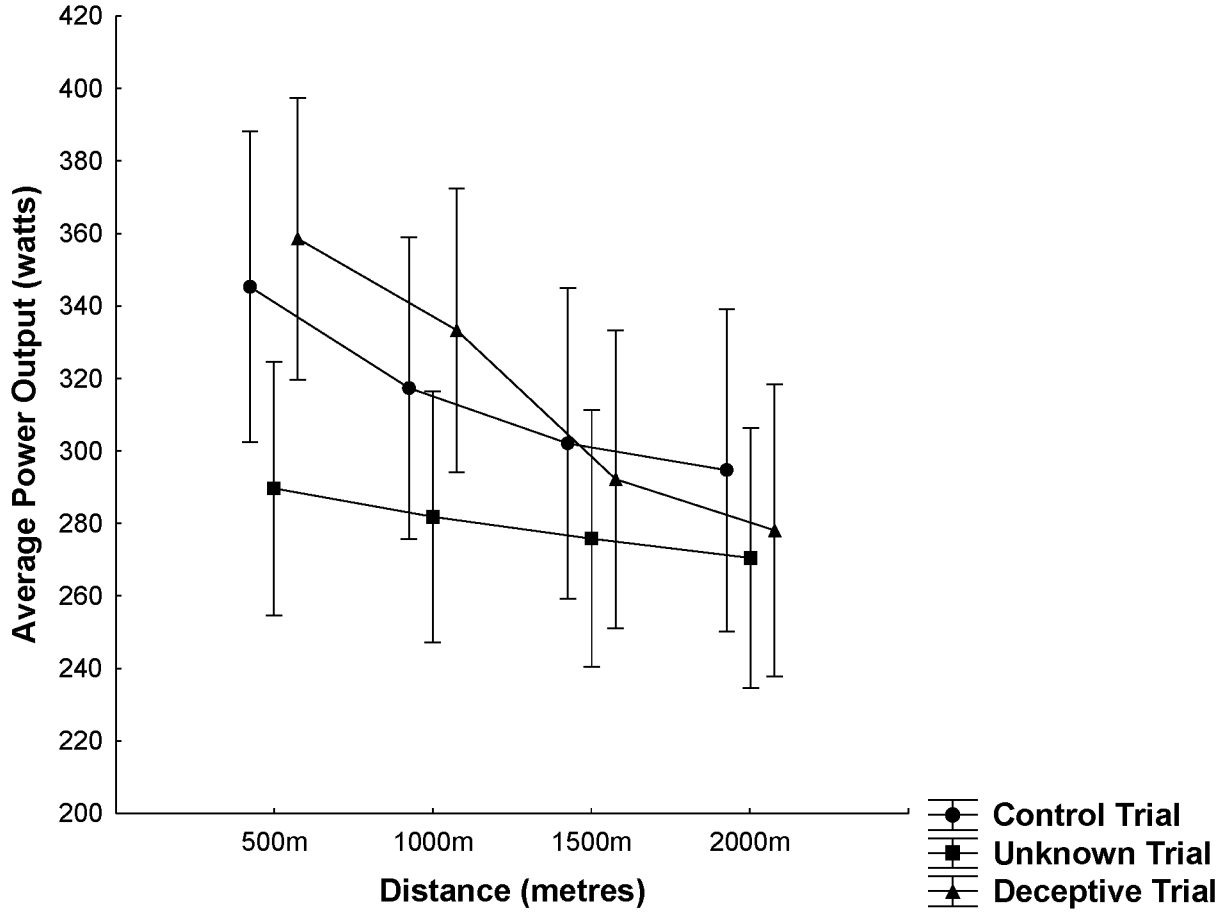

Figure 8: Mean power output over each distance interval for each condition.

Throughout all trials, power output decreased as distance increased, however the magnitude of the decrease was different for each condition and interval. It is important to recognize the limitation of the power output measurement. Each power output measurement is the average of the entire trial up to that point, rather than the average of each 500m interval.

Table V: Tukey post hoc analysis of mean power output data at each distance interval for each condition.

Measure	CT 500m (345,36)	CT 1000m (317,55)	CT 1500m (302,27)	CT 2000m (294,91)	UT 500m (289,91)	UT 1000m (282,09)	UT 1500m (276,09)	UT 2000m (270,73)	DT 500m (358,64)	DT 1000m (333,45)	DT 1500m (292,45)	DT 2000m (278,36)
CT 500m		0,000123	0,000123	0,000123	0,000123	0,000123	0,000123	0,000123	0,040941	0,101905	0,000123	0,000123
CT 1000m	0,000123		0,008957	0,000130	0,000123	0,000123	0,000123	0,000123	0,000123	0,005352	0,000123	0,000123
CT 1500m	0,000123	0,008957		0,737283	0,076177	0,000218	0,000123	0,000123	0,000123	0,000123	0,319051	0,000124
CT 2000m	0,000123	0,000130	0,737283		0,974755	0,056187	0,000512	0,000124	0,000123	0,000123	0,999958	0,003181
UT 500m	0,000123	0,000123	0,076177	0,974755		0,660402	0,027542	0,000402	0,000123	0,000123	0,999940	0,127379
UT 1000m	0,000123	0,000123	0,000218	0,056187	0,660402		0,912666	0,141864	0,000123	0,000123	0,245207	0,997746
UT 1500m	0,000123	0,000123	0,000123	0,000512	0,027542	0,912666		0,958209	0,000123	0,000123	0,003691	0,999981
UT 2000m	0,000123	0,000123	0,000123	0,000124	0,000402	0,141864	0,958209		0,000123	0,000123	0,000141	0,691856
DT 500m	0,040941	0,000123	0,000123	0,000123	0,000123	0,000123	0,000123	0,000123		0,000123	0,000123	0,000123
DT 1000m	0,101905	0,005352	0,000123	0,000123	0,000123	0,000123	0,000123	0,000123	0,000123		0,000123	0,000123
DT 1500m	0,000123	0,000123	0,319051	0,999958	0,999940	0,245207	0,003691	0,000141	0,000123	0,000123		0,022475
DT 2000m	0,000123	0,000123	0,000124	0,003181	0,127379	0,997746	0,999981	0,691856	0,000123	0,000123	0,022475	

Approximate Probabilities for Post Hoc Tests Error: Within MSE = 80,218, df = 60,000. Mean power output (watts) presented in brackets, significance highlighted in red.

Within Conditions

The control condition saw a high average power output at the 500m interval (345.36 ±63.64 watts). This was significantly higher than all other intervals in the control condition (p<0.05; d = 0.44, 0.68 and 0.78 respectively). The 1500m and 2000m splits also had significantly lower average power output compared to the 1000m interval (p<0.05; d = 0.24 and 0.35 respectively). The power output of the 500m interval (289.91 ±51.94 watts) of the unknown trial was significantly higher than that of the 1000m, 1500m and 2000m intervals (p<0.05; d = 0.15, 2.18 and 0.37 respectively). The 2000m interval (270.73 ±53.25) of the unknown trial saw the lowest average power output of all trials and intervals. In contrast, the deceptive trial saw the highest average power output of all trials and intervals at the 500m mark (358.64 ±57.70 watts). This value was significantly higher than all other intervals in this condition (p<0.05; d = 0.44, 1.11 and 1.37 respectively). The 2000m interval was also significantly lower than the 1000m interval (p<0.05; d = 0.93).

Between Conditions

Power output at the 500m interval was highest in the deceptive trial, until the 1500m interval where it dropped below that of the control trial. This resulted in the control trial having a higher power output at the 2000m interval. Interestingly, power output of the unknown trial was lower than the control and deceptive trial at all intervals. At the 500m interval, power output was significantly higher in the control and deceptive conditions compared to the unknown condition (p<0.05; d = 0.96 and 1.25 respectively). A similar effect occurred at the 1000m interval where the unknown condition had a significantly lower power output than the control and deceptive conditions (p<0.05; d = 0.62 and 0.94). However, the control trial was also significantly lower than the deceptive trial at the 1000m interval (p<0.05; d = 0.27). Again, at the 1500m mark, the control and deceptive trials were significantly higher than the unknown trial (p<0.05; d = 0.45 and 0.29). Finally, at the 2000m interval both the deceptive and unknown trials had significantly lower power output compared to the control trial (p<0.05; d = 0.40 and 0.26).

MUSCLE ACTIVITY: MEDIAL DELTOID

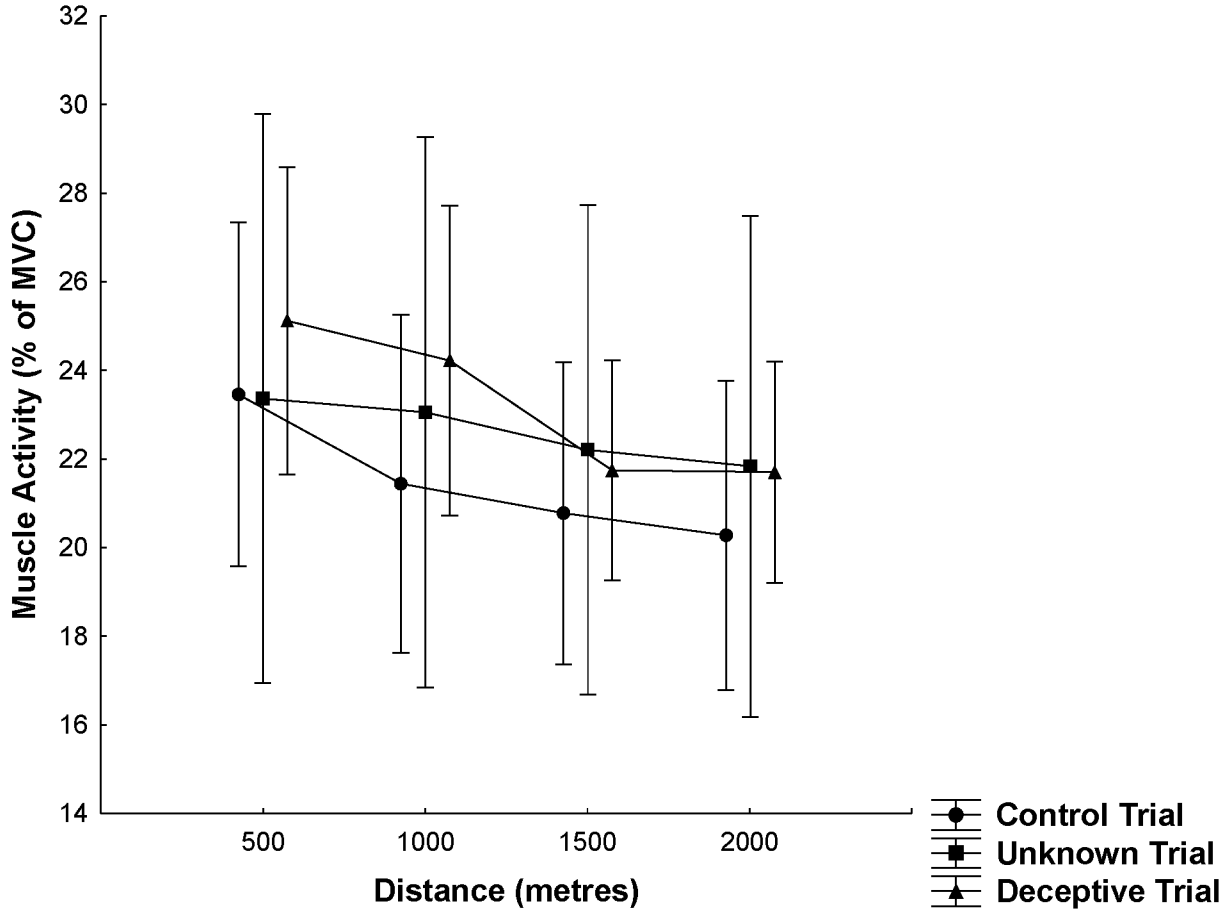

Figure 9: Mean muscle activity of the medial deltoid at each distance interval for each condition.

Table VI: Tukey post hoc analysis of mean medial deltoid muscle activity at each distance interval for each condition.

Measure	CT 500m (23,46)	CT 1000m (21,46)	CT 1500m (20,79)	CT 2000m (20,29)	UT 500m (23,37)	UT 1000m (23,06)	UT 1500m (22,22)	UT 2000m (21,85)	DT 500m (25,13)	DT 1000m (24,23)	DT 1500m (21,76)	DT 2000m (21,72)
CT 500m		0,040645	0,001232	0,000164	1,000000	0,999912	0,586351	0,205923	0,174755	0,972888	0,147266	0,127799
CT 1000m	0,040645		0,990858	0,682083	0,061639	0,212610	0,972888	0,999930	0,000124	0,000730	0,999995	0,999999
CT 1500m	0,001232	0,990858		0,999257	0,002064	0,010904	0,377621	0,797572	0,000123	0,000129	0,875183	0,900403
CT 2000m	0,000164	0,682083	0,999257		0,000201	0,000730	0,059183	0,256014	0,000123	0,000123	0,340130	0,377621
UT 500m	1,000000	0,061639	0,002064	0,000201		0,999993	0,692396	0,279722	0,123265	0,940544	0,205923	0,180689
UT 1000m	0,999912	0,212610	0,010904	0,000730	0,999993		0,944618	0,618688	0,031310	0,682083	0,510844	0,468451
UT 1500m	0,586351	0,972888	0,377621	0,059183	0,692396	0,944618		0,999956	0,000389	0,040645	0,999633	0,999257
UT 2000m	0,205923	0,999930	0,797572	0,256014	0,279722	0,618688	0,999956		0,000142	0,006106	1,000000	1,000000
DT 500m	0,174755	0,000124	0,000123	0,000123	0,123265	0,031310	0,000389	0,000142		0,917059	0,000133	0,000130
DT 1000m	0,972888	0,000730	0,000129	0,000123	0,940544	0,682083	0,040645	0,006106	0,917059		0,003738	0,003071
DT 1500m	0,147266	0,999995	0,875183	0,340130	0,205923	0,510844	0,999633	1,000000	0,000133	0,003738		1,000000
DT 2000m	0,127799	0,999999	0,900403	0,377621	0,180689	0,468451	0,999257	1,000000	0,000130	0,003071	1,000000	

Approximate Probabilities for Post Hoc Tests Error: Within MSE = 1,8352, df = 60,000. Mean muscle activity (%) presented in brackets, significance highlighted in red.

Within Conditions

A decrease in medial deltoid muscle activity occurred over the course of all the trials. The control trial saw a steady decrease from the 500m interval (23.46 ±5.77%), which was significantly higher than the 1000m, 1500m and 2000m intervals (p<0.05; d = 0.35, 0.49 and 0.58 respectively). The 2000m interval (20.29 ±5.19%) of the control saw the lowest medial deltoid muscle activity of any interval and trial. The unknown trial showed a non-significant decrease over the course of the trial, while the deceptive trial exhibited a significant (p<0.05; d = 0.55) decrease between the 1000m (24.23 ±5.20%) and 1500m (21.75 ±3.68%) intervals. The 1500m and 2000m intervals were both significantly lower than the 500m and 1000m intervals (p<0.05; d = 0.76, 0.76, 0.55 and 0.56 respectively) in the deceptive trial.

Between Conditions

At the 500m mark, the deceptive condition (25.13 ±5.15%) exhibits a higher muscle activity then both the control (23.46 ±5.77%) and unknown (23.37 ±9.55%) conditions. The 1000m interval of the control trial had a significantly (p<0.05; d = 0.51) lower muscle activity compared to the deceptive trial. At the 1500m and 2000m intervals, deltoid muscle activity continued to decrease, although this decrease was not significant. The deceptive trial saw a significant (p<0.05; d = 0.55) reduction in deltoid muscle activity from the 1000m to the 1500m interval, thereafter remaining the same.

ERECTOR SPINAE

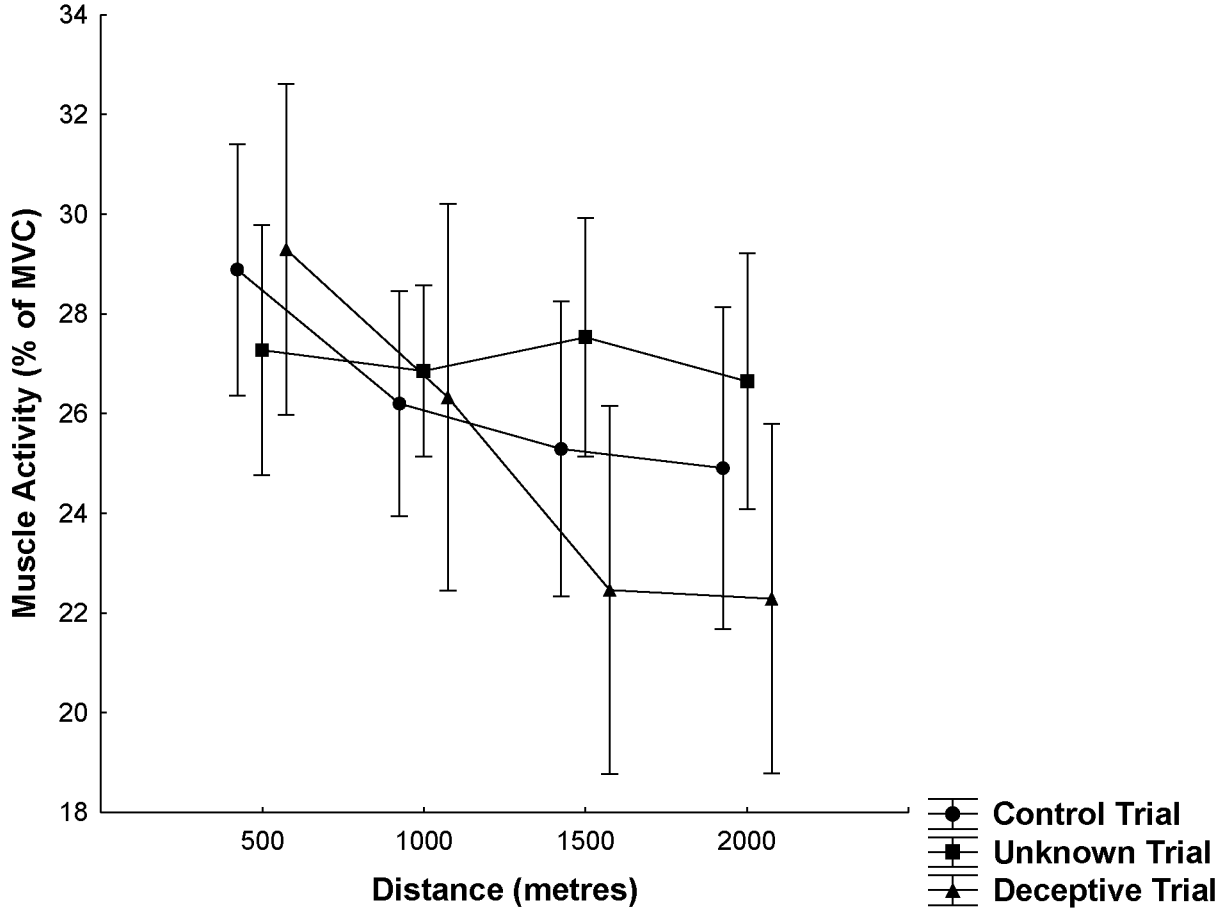

Figure 10: Mean muscle activity of the erector spinae at each distance interval for each condition.

Table VII: Tukey post hoc analysis of mean erector spinae muscle activity at each distance interval for each condition.

Measure	CT 500m (28,90)	CT 1000m (26,22)	CT 1500m (25,31)	CT 2000m (24,93)	UT 500m (27,29)	UT 1000m (26,87)	UT 1500m (27,55)	UT 2000m (26,66)	DT 500m (29,31)	DT 1000m (26,35)	DT 1500m (22,48)	DT 2000m (22,31)
CT 500m		0,064447	0,002385	0,000561	0,703213	0,361725	0,875068	0,226197	0,999996	0,095217	0,000123	0,000123
CT 1000m	0,064447		0,992595	0,905960	0,972983	0,999618	0,888969	0,999992	0,015953	1,000000	0,001330	0,000699
CT 1500m	0,002385	0,992595		0,999998	0,395986	0,738578	0,226197	0,875068	0,000512	0,979092	0,040156	0,022112
CT 2000m	0,000561	0,905960	0,999998		0,163350	0,424487	0,078564	0,598225	0,000184	0,838899	0,130581	0,078564
UT 500m	0,703213	0,972983	0,395986	0,163350		0,999996	1,000000	0,999745	0,368456	0,989794	0,000130	0,000126
UT 1000m	0,361725	0,999618	0,738578	0,424487	0,999996		0,999505	1,000000	0,133941	0,999953	0,000181	0,000147
UT 1500m	0,875068	0,888969	0,226197	0,078564	1,000000	0,999505		0,994254	0,575154	0,940873	0,000125	0,000123
UT 2000m	0,226197	0,999992	0,875068	0,598225	0,999745	1,000000	0,994254		0,072222	1,000000	0,000287	0,000192
DT 500m	0,999996	0,015953	0,000512	0,000184	0,368456	0,133941	0,575154	0,072222		0,025134	0,000123	0,000123
DT 1000m	0,095217	1,000000	0,979092	0,838899	0,989794	0,999953	0,940873	1,000000	0,025134		0,000823	0,000456
DT 1500m	0,000123	0,001330	0,040156	0,130581	0,000130	0,000181	0,000125	0,000287	0,000123	0,000823		1,000000
DT 2000m	0,000123	0,000699	0,022112	0,078564	0,000126	0,000147	0,000123	0,000192	0,000123	0,000456	1,000000	

Approximate Probabilities for Post Hoc Tests Error: Within MSE = 3,6250, df = 60,000. Mean muscle activity (%) presented in brackets, significance highlighted in red.

Within Conditions

The control condition saw a progressive decrease in erector spinae muscle activity over the course of the trial. The 500m interval muscle activity (28.90 ±3.75%) was significantly (p<0.05; d = 0.88 and 0.92 respectively) higher than the 1500m (25.31 ±4.40%) and 2000m (24.93 ±4.80%) intervals. There was steady erector spinae muscle activity during the course of the unknown trial. The deceptive trial exhibited a significant (p<0.05; d = 0.55 and 1.31 respectively) decrease from the 500m (29.31 ±4.94%) interval to the 1000m (26.35 ±5.76%) and a similar (p<0.05; d = 0.68) decrease at the 1500m (22.48 ±5.48%) interval. The 500m and 1000m intervals were also significantly (p<0.05; d = 1.38 and 0.69 respectively) higher than the 2000m (22.31 ±5.21%). The 1500m and 2000m intervals showed similar muscle activity in the deceptive trial.

Between Conditions

The highest muscle activity was found at the 500m (29.31 ±4.94%) of the deceptive trial, while the lowest was recorded at 2000m (22.31 ±5.21%) during the deceptive condition. Additionally, the 1500m interval of the deceptive (22.48 ±5.48%) trial showed significantly (p<0.05; d = 0.57 and 1.10 respectively) lower muscle activity than the 1500m intervals of the control (25.31 ±4.40%) and unknown (27.55 ±3.56%) conditions.

BICEPS FEMORIS

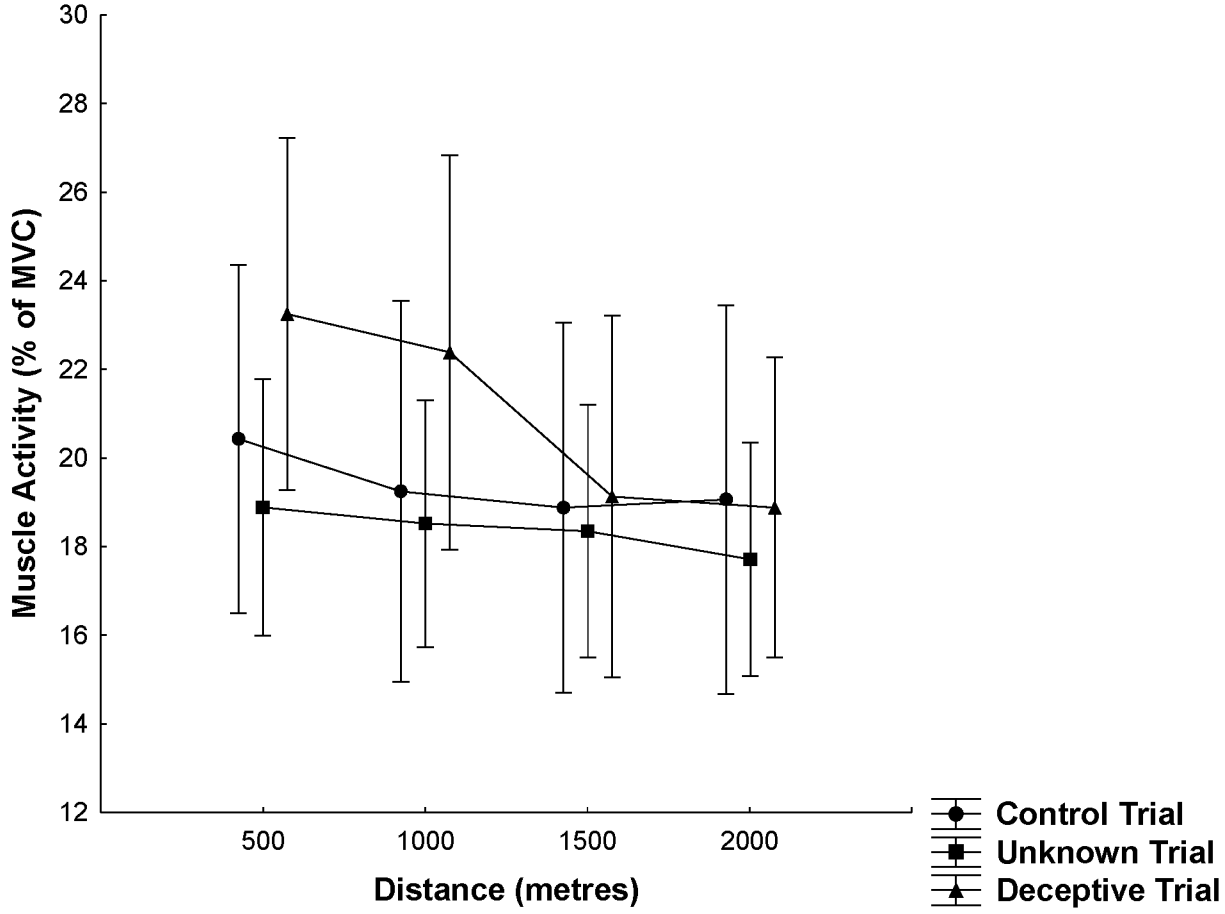

Figure 11: Mean muscle activity of the biceps femoris at each distance interval for each condition.

Table VIII: Tukey post hoc analysis of mean biceps femoris muscle activity at each distance interval for each condition.

Measure	CT 500m (20,44)	CT 1000m (19,26)	CT 1500m (18,89)	CT 2000m (19,07)	UT 500m (18,90)	UT 1000m (18,53)	UT 1500m (18,36)	UT 2000m (17,72)	DT 500m (23,26)	DT 1000m (22,39)	DT 1500m (19,14)	DT 2000m (18,89)
CT 500m		0,910212	0,649320	0,800173	0,654212	0,334725	0,216131	0,027129	0,018162	0,297631	0,844265	0,649320
CT 1000m	0,910212		0,999998	1,000000	0,999998	0,997960	0,987090	0,661528	0,000199	0,004992	1,000000	0,999998
CT 1500m	0,649320	0,999998		1,000000	1,000000	0,999998	0,999882	0,916446	0,000133	0,001066	1,000000	1,000000
CT 2000m	0,800173	1,000000	1,000000		1,000000	0,999867	0,998122	0,810081	0,000151	0,002329	1,000000	1,000000
UT 500m	0,654212	0,999998	1,000000	1,000000		0,999998	0,999869	0,913995	0,000133	0,001090	1,000000	1,000000
UT 1000m	0,334725	0,997960	0,999998	0,999867	0,999998		1,000000	0,994507	0,000124	0,000286	0,999613	0,999998
UT 1500m	0,216131	0,987090	0,999882	0,998122	0,999869	1,000000		0,999399	0,000123	0,000186	0,995983	0,999882
UT 2000m	0,027129	0,661528	0,916446	0,810081	0,913995	0,994507	0,999399		0,000123	0,000125	0,761867	0,916446
DT 500m	0,018162	0,000199	0,000133	0,000151	0,000133	0,000124	0,000123	0,000123		0,990574	0,000163	0,000133
DT 1000m	0,297631	0,004992	0,001066	0,002329	0,001090	0,000286	0,000186	0,000125	0,990574		0,003050	0,001066
DT 1500m	0,844265	1,000000	1,000000	1,000000	1,000000	0,999613	0,995983	0,761867	0,000163	0,003050		1,000000
DT 2000m	0,649320	0,999998	1,000000	1,000000	1,000000	0,999998	0,999882	0,916446	0,000133	0,001066	1,000000	

Approximate Probabilities for Post Hoc Tests Error: Within MSE = 3,0846, df = 60,000. Mean muscle activity (%) presented in brackets, significance highlighted in red.

Within Conditions

The control trial exhibited a non-significant decrease in muscle activity until the 1500m interval, after which an increase occurred at the 2000m interval. Muscle activity during the unknown condition remained constant up to the 1500m interval with a decrease at 2000m interval. The deceptive trial resulted in high muscle activity during the 500m and 1000m intervals. A significant decrease then followed. The 500m interval (23.25 ±5.90%) was significantly (p<0.05; d = 0.69 and 0.80) higher than the 1500m interval (19.14 ±6.06%) and the 2000m interval (18.89 ±5.03%) during the deceptive trial. The 1000m interval (22.39 ±6.62%) was also significantly (p<0.05; d = 0.51 and 0.60) higher than the 1500m and 2000m intervals.

Between Conditions

The highest muscle activity of the biceps femoris was found at the 500m interval of the deceptive trial (23.25 ±5.90%), while the lowest was seen at the 2000m interval of the unknown condition (17.72 ±3.91%). The 500m interval of the deceptive trial was significantly (p<0.05; d = 0.48 and 0.84 respectively) higher than the 500m interval of the control trial (20.44 ±5.85%) and the 500m interval of the unknown condition (18.90 ±4.31%). Furthermore, the 1000m interval of the deceptive trial (22.39 ±6.62%) was significantly (p<0.05; d = 0.48 and 0.70 respectively) higher than the 1000m interval of the control trial (19.25 ±6.39%) and the 1000m interval of the unknown the trial (18.53 ±4.14%).

QUADRICEPS FEMORIS

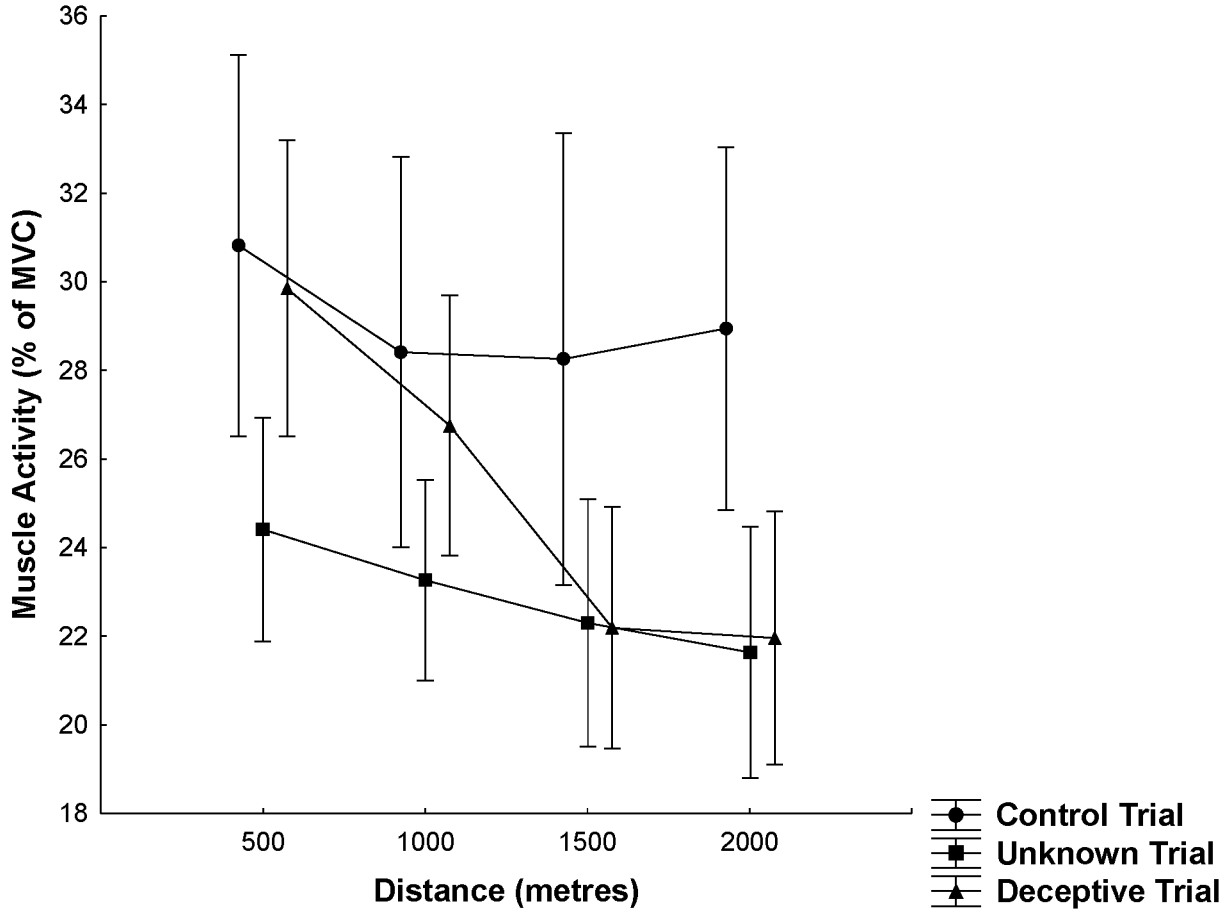

Figure 12: Mean muscle activity of the quadriceps femoris at each distance interval for each condition.

Table IIII: Tukey post hoc analysis of mean muscle activity of quadriceps femoris at each distance interval for each condition.

Measure	CT 500m (30,84)	CT 1000m (28,43)	CT 1500m (28,27)	CT 2000m (28,96)	UT 500m (24,43)	UT 1000m (23,28)	UT 1500m (22,32)	UT 2000m (21,66)	DT 500m (29,86)	DT 1000m (26,77)	DT 1500m (22,21)	DT 2000m (21,98)
CT 500m		0,165627	0,108629	0,516284	0,000123	0,000123	0,000123	0,000123	0,989189	0,000550	0,000123	0,000123
CT 1000m	0,165627		1,000000	0,999955	0,000682	0,000125	0,000123	0,000123	0,846125	0,694705	0,000123	0,000123
CT 1500m	0,108629	1,000000		0,999479	0,001193	0,000127	0,000123	0,000123	0,743259	0,806776	0,000123	0,000123
CT 2000m	0,516284	0,999955	0,999479		0,000168	0,000123	0,000123	0,000123	0,994295	0,280764	0,000123	0,000123
UT 500m	0,000123	0,000682	0,001193	0,000168		0,962811	0,334659	0,058101	0,000123	0,194818	0,264066	0,150505
UT 1000m	0,000123	0,000125	0,000127	0,000123	0,962811		0,989973	0,715857	0,000123	0,004668	0,976911	0,913841
UT 1500m	0,000123	0,000123	0,000123	0,000123	0,334659	0,989973		0,999644	0,000123	0,000190	1,000000	1,000000
UT 2000m	0,000123	0,000123	0,000123	0,000123	0,058101	0,715857	0,999644		0,000123	0,000125	0,999938	1,000000
DT 500m	0,989189	0,846125	0,743259	0,994295	0,000123	0,000123	0,000123	0,000123		0,020166	0,000123	0,000123
DT 1000m	0,000550	0,694705	0,806776	0,280764	0,194818	0,004668	0,000190	0,000125	0,020166		0,000163	0,000136
DT 1500m	0,000123	0,000123	0,000123	0,000123	0,264066	0,976911	1,000000	0,999938	0,000123	0,000163		1,000000
DT 2000m	0,000123	0,000123	0,000123	0,000123	0,150505	0,913841	1,000000	1,000000	0,000123	0,000136	1,000000	

Approximate Probabilities for Post Hoc Tests Error: Within MSE = 3,7823, df = 60,000. Mean muscle activity (%) presented in brackets, significance highlighted in red.

Within Conditions

Muscle activity of the quadriceps femoris decreased over the course of the control trial until the 2000m interval at which point an increase was seen. The unknown trial had a non-significant decline in muscle activity over the duration, while the deceptive trial showed a decrease in muscle activity up to the 1500m interval after which it stabilised. The 500m interval of the deceptive condition (29.86 ±4.97%) was significantly (p<0.05; d = 0.66, 1.69 and 1.71 respectively) higher than the 1000m interval (26.77 ±4.37%), 1500m interval (22.21 ±4.06%) and 2000m interval (21.98 ±4.24%). The 1000m interval was also significantly (p<0.05; d = 1.08 and 1.11) higher than the 1500m and 2000m intervals.

Between Conditions

The highest muscle activity of the quadriceps femoris was found in the 500m interval of the control trial (30.84 ±6.40%), while the lowest was found at the 2000m interval of the unknown condition (21.65 ±4.21%). The 500m interval of the unknown trial (24.43 ±3.75%) was significantly (p<0.05; d = 1.22 and 1.23 respectively) lower than the 500m intervals of the control (30.84 ±6.40%) and deceptive (29.86 ±4.97%) trials. The 1000m interval of the unknown trial (23.28 ±3.37%) was also significantly (p<0.05; d = 0.99 and 0.89) lower than the 1000m intervals of the control (28.43 ±6.55%) and deceptive (26.77 ±4.37%) trials. At the 1500m interval, the control trial (28.27 ±7.59%) was significantly (p<0.05; d = 0.97 and 0.10) higher than the 1500m intervals of the unknown (22.32 ±4.16%) and deceptive (22.21 ±4.06%) trials. The 2000m interval of the control trial (28.96 ±6.09%) was also significantly (p<0.05; d = 1.40 and 1.33) higher than the 2000m intervals of the unknown (21.65 ±4.21%) and the deceptive (21.98 ±4.24%) trials.

INDIVIDUAL DATA

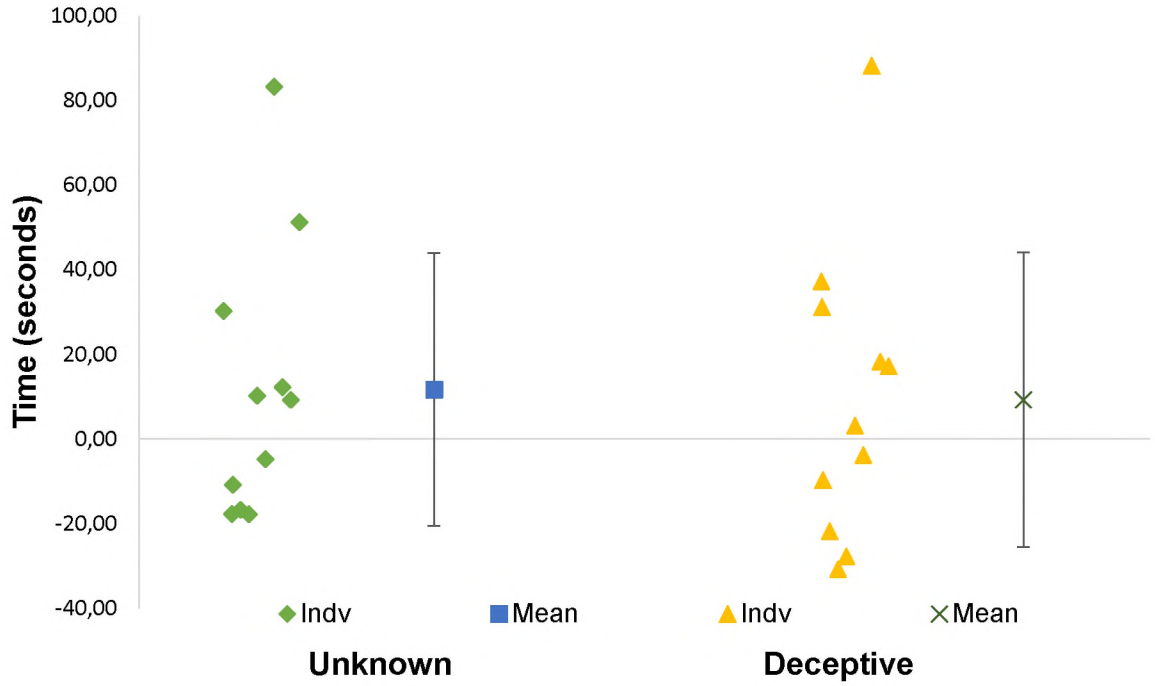

Figure 13: Individual and mean performance times of the unknown and deceptive conditions relative to the mean control trial performance time (depicted by the horizontal axis).

It is often assumed that the mean change in a variable, following an exercise intervention, represents the typical response for most individuals. However, it is more common for individuals to respond to exercise interventions in a variety of manners (Mann, Lamberts and Lambert, 2014). Indeed, as is evident from Figure 4 (page 39) and Figure 13 (page 64), on average, the control trial was faster than both the deceptive and unknown trials. Although not the focus of this study, it was interesting to note the variation between the responses of participants which should be investigated in future studies. Large differences in responses have been found between individuals following standardized training programmes (Hautala *et al.*, 2006). The variation in individual responses around the mean indicates the large role of genetic factors, particularly since participants were following the same training programme at the time of testing. Furthermore, it seems the variance in responses is dependent on the variable which is being measured (Mann *et al.*, 2014). It is also recognised that if "non-responders" were training more, their response may be

different. Thus, the dose-response relationship should also be investigated (Montero and Lundby, 2017).

Summary of Key Findings

Overall, both the unknown and deceptive trials were significantly (p<0.05) slower than the control trial, however the deceptive trial was the fastest of all trials at the 1000m distance. The unknown trial was slowest trial overall. Muscle activity, RPE, heart rate and power output were significantly (p<0.05) lower in the unknown trial compared to the control and deceptive trial. The deceptive trial revealed a significant (p<0.05) reduction in performance time, heart rate, power output and muscle activity after the 1000m interval. The first 500m interval had the fastest performance and highest power output in all trials. Additionally, in all trials, RPE was highest at the end of the 2000m, the highest reading being obtained at the end of the control trial. During the first 1000m of the deceptive trial, the dependant variables were similar to those in the control trial. Once the deception was revealed, there was a significant (p<0.05) reduction in the muscle activity, heart rate and power output.

CHAPTER V

DISCUSSION

GENERAL FINDINGS

Anthropometric Data

Participants had an average stature of 180cm (Table, page 38), which is 10cm lower than elite male oarsmen (Hagerman, 1984). It is well established that taller individuals tend to have an advantage when rowing (Hagerman, 1984; Shephard, 1998). This was an expected finding as participants were not of the elite calibre. It is interesting to note that the coefficient of variation was 5.46%, while for mass it was 11.61% (Table, page 38) i.e. stature among participants was similar, while body mass was less similar. This may indicate that stature is a more important indicator of rowing potential than mass.

Mean BMI was 24.49 $kg.m^{-2}$, which is similar to the value obtained by Maestu *et al.* (1999) of 22.83 $kg.m^{-2}$ during a study of collegiate rowers. This is within the healthy body mass index range of 18.5 – 24.9 $kg.m^{-2}$ as outlined in the World Health Organisation guidelines.

IDENTIFYING PACING STRATEGIES

The current study comprised three trials consisting of the same distance (2000m row) where only the nature of the information provided before and during each trial differed. It was hypothesised that this varied information would result in different pacing strategies resulting in different responses in the dependant variables. These methods were modelled on previous studies examining pacing strategies and deception (Baden *et al.*, 2005; Billaut *et al.*, 2011; Elliott, 2014; Eston *et al.*, 2012). While some of these studies focused on pacing during repeated sprints, the current study was concerned with continuous exercise.

Control Trial

The control trial exhibited a reverse J-shaped pacing strategy consistent with literature (Garland, 2005; Abbiss and Laursen, 2008; Brown *et al.*, 2010; Muehlbauer *et al.*, 2010). Characterised by the first and last quarters of the race being significantly faster

than the second and third quarters, the control trial exhibited this pattern, with the first 500m being significantly faster than the 1000m and 1500m (Figure 5, page 40) (Abbiss and Laursen, 2008). The 2000m interval was faster than the 1500m, although this was not significant. It is possible this is due to the different capabilities of the rowers in the present study as Garland (2005) found that rowers of different calibre may pace themselves differently. Another possibility is that more regular intervals could have been more sensitive to detect subtle changes in performance in this study. Nonetheless, the pattern clearly corresponds to those reported in previous studies as the reverse J-shaped pacing profile. This finding also agrees with pacing research in other sports such as cycling (Albertus *et al.*, 2005) and running (Noakes, Lambert and Hauman, 2009). This parallel is due to the similar nature of the exercise mode of rowing, cycling and running as they were all self-paced, head-to-head competitions during which the individual with the shortest time to completion wins (Muehlbauer *et al.*, 2010). Thus, the type of pacing strategy used during the control condition was determined by the known length of the exercise bout. Though they received no distance or time feedback, the rowers had performed regular 2000 m trials during training and had a good idea of the requirements, thereby eliminating the need for feedback and drawing on previous knowledge of the same exercise bout (Tucker, 2009).

This pacing pattern is contrary to the even pacing profile which has been suggested to be more beneficial from an efficiency and hydrodynamic point of view (Kleshnev, 2000). Furthermore, even pacing is supported by several mathematical and power models (Billat *et al.*, 1999; Foster *et al.*, 2003). It is common opinion that even distribution of boat speed is the most optimal pacing profile for rowing (Abbiss and Laursen, 2008). However, this is not the pacing strategy most rowers and coaches employ, because there are other factors which superimpose influence over the previous one (Kleshnev, 2000). For example, it is tactically and physiologically advantageous to perform the initial part of the race fast (Shephard, 1998).

Seventy to eighty percent of energy utilised during a 2000m rowing bout is obtained from oxygen dependant metabolism (Kleshnev, 2000; Secher and Volianitis, 2007). While it is known that neither energy system functions in isolation, it is beneficial for rowers to start the race fast as this allows oxygen independent metabolism to be utilised which provides more power required to start the boat moving and reach

maximum speed quickly (Secher and Volianitis, 2007). This strategy allows rowers to utilise oxygen independent pathways at the end of the race as well since stores will be replenished by this stage (Secher and Volianitis, 2007). However, the start should not be too fast otherwise rowers must tolerate high lactate concentrations and oxygen debt (Kleshnev, 2000). The second factor is psychological. It is advantageous for rowers to begin the race at a fast speed as this allows them to monitor and react to trailing competitors during the race (Shephard, 1998; Kleshnev, 2000).

The increase in speed over the final 500m lends support to the anticipatory regulation of exercise intensity (St Clair Gibson *et al.*, 2006; Tucker, 2009). This 'end-spurt', as it is commonly referred to, suggests that an energetic reserve is maintained during exercise to prevent premature exhaustion as well as catastrophic failure of homeostasis (Foster *et al.*, 2003). The end-spurt is particularly advantageous in securing a finishing position, which is crucial to success in rowing (Muehlbauer *et al.*, 2010). Therefore, withholding some energetic resources for later in the race may be advantageous in sports such as rowing (Muehlbauer *et al.*, 2010).

A decreasing muscle activation would be expected due to the increasing demands being placed on the musculoskeletal system (Peltonen *et al.*, 1997). While muscle activation was initially high, significant decreases occurred in the medial deltoid and erector spinae muscles (Figure 9, page 52; Figure 10, page 55). In these measures, muscle activity at the 500m interval was significantly higher than all other intervals. This indicates that these muscles were not responsible for the end-spurt observed. Additionally, research has shown that rowers utilise different muscle groups when rowing maximally as opposed to sub-maximally which may be a reason for the differences found between muscles (Gerzevic *et al.*, 2011).

The EMG activity results show that two muscles (biceps femoris and quadriceps femoris) contributed to the end-spurt while two (medial deltoid and erector spinae) did not, possibly due to the upper limb muscles being fatigued. Thus, it is inaccurate to conclude that power output and performance were affected by an increase in muscle activity or whether muscle activity may have decreased with a concomitant increase in performance. A study by Hettinga *et al.* (2006) found that EMG of the vastus lateralis and biceps femoris muscles increased despite a corresponding decline in power output during a 4000m cycling time trial on an ergometer. Similarly, EMG activity of

the biceps femoris and quadriceps femoris exhibited a parabolic profile over the course of the trial in the present study (Figure 11, page 58; Figure 12, page 61). While no intervals were significantly different in these measures, this result supports the central regulation of exercise as research has shown variations in power output correspond to changes in muscle activity (Tucker *et al.*, 2004). Billaut *et al.* (2011) suggest higher levels of muscle activation result in greater mechanical output, while St Clair Gibson *et al.* (2001) found that reductions in muscle activity were linked to decreased power output during high intensity cycling. Due to greater stimulation from the central nervous system, more muscle fibres are recruited, increasing power output (Enoka, 1996). Therefore, if regulation of exercise were purely due to peripheral feedback, it could not allow an end-spurt since it could not foresee the extent of the fatigue at the finish of the end-spurt (Marino, 2014).

Muscle activity increased in the biceps femoris and rectus femoris muscles after the 1500m interval. Hettinga *et al.* suggest fatigue and resulting variations in pacing strategy are not simply dictated by central regulation, but also by physiological changes to the muscle itself (Hettinga *et al.*, 2006). Ansley *et al.* (2004) found that maximal power output was obtained 60s into a 4000m cycling time trial, after which there was a decline in power during the middle of the trial. This corresponds somewhat to the results of the present study since the highest power output was seen at 500m. Dissimilarly, Ansley *et al.* (2004) found an increase in the final seconds of the trial. It is likely that the measurement error during the collection of the power output data meant that the end-spurt was not observed during the current study. It is also plausible that the measurement intervals of the current protocol were too large to observe the end-spurt in power output.

Ansley *et al.* (2004) conclude from power output and EMG measures that, rather than peripheral fatigue being the limit to performance, a subconscious "controller" determines pacing strategy. Approximately a quarter of motor units were recruited during the control trial indicating changes in the motor units which are recruited and de-recruited during different stages of exercise. The results of the control trial in the study by Ansley *et al.*, (2004) confirm this conclusion since muscle activity measures were never greater than 33% of MVC. This is incompatible with the peripheral fatigue model, which predicts that motor unit recruitment would increase with increasing fatigue (Ansley *et al.*, 2004).

RPE was significantly lower at the 500m intervals compared to all other intervals, consistent with the literature (Foster *et al.*, 2009; Swart *et al.*, 2009). Swart *et al.* (2009) found that RPE values typically rise as a function of exercise duration. It is proposed that the rising RPE produced during the exercise bout serves as a protective mechanism to prevent an exercise intensity which could potentially threaten homeostasis (Swart *et al.*, 2009, Tucker, 2009). This occurrence is most likely due to the athletes having performed the same exercise bout several times. Therefore, they could push themselves to the limits of their capabilities having reliable knowledge of how long the exercise bout would be from previous experience (Tucker and Noakes, 2009). Similar results were found by Foster *et al.* (2009) during varying lengths of cycling time trials where RPE values peaked at the end of each trial regardless of distance. As with several other studies examining RPE, values peaked at the end of the trial, specifically in the final 2000m of the control condition (Joseph *et al.*, 2008; Swart *et al.*, 2009; Billaut *et al.*, 2011). The mean RPE value of 19, corresponding to the verbal anchor "extremely hard", at the end of the trial indicates that participants had most likely exerted themselves maximally by this point. This is supported by the high heart rate (183 bts.min^{-1}) readings measured concurrently.

It is well established that RPE increases linearly as a function of the proportion of the exercise bout completed and this appears to hold true in the control trial (de Koning *et al.*, 2011). RPE at the 500m interval was significantly lower than all other intervals, while the 1000m interval was significantly lower than the 2000m interval indicating a progressive increase over the course of the trial (Figure 6, page 43). There is a mismatch between RPE and power output, which Marino (2014) attributes to the fact that physiological feedback during the early stages of exercise is difficult to achieve. It is possible that some forms of physiological feedback (such as blood pH) may take time to alter and be integrated by the CNS thereby causing the lag effect (Billaut *et al.*, 2011).

Unknown Trial

The unknown trial exhibited what might be defined as an even pacing strategy by Abbiss and Laursen (2008). It is characterised by a relatively constant pace throughout the exercise event and is supported by several mathematical and power models (Billat

et al., 1999; Foster *et al.*, 2003). The theoretical basis for this type of pacing strategy is that athletes are able to exercise at an intensity slightly below the fatigue threshold, preventing the accumulation of deleterious metabolites (Abbiss and Laursen, 2008). This allows the athlete to continue exercising for prolonged periods without a substantial drop in power output. As an example, performance time increased over the course of the unknown trial (Figure 5, page 40). This increase was not significant, but was approximately 1 second per 500m interval. According to the anticipatory feedback model, during exercise of unknown duration, athletes become more economical by reducing work rate thereby saving physiological resources, simultaneously resulting in sub-optimal performance (Tucker, 2009). The slow initial work rate in conjunction with a slow rate of RPE increase ensures that a reserve is maintained. This results in physiological variables such as heart rate and power output remaining relatively consistent over the course of the exercise bout (Baden *et al.,* 2005; Coquart and Garcin, 2008). This study saw similar results with reduction in variables such as performance, heart rate, and power output and muscle activity.

Billaut *et al.* (2011) found similar results, where participants' muscle activity was reduced during repeated sprints of unknown number, thereby causing reduced power output. Power output in the unknown trial in this study was initially reduced, followed by a progressive decline over the course of the condition. The decline can be explained by an extension of the anticipatory regulation hypothesis proposed by Tucker (2009). It is likely that during exercise of unknown duration, as the exercising time increases, athletes will downregulate further due to the uncertainty associated with the exercise bout.

Muscle activity tended to decrease over the course of the trial in a manner similar to that of power output, however no significant differences were found. It has been shown that muscle activity and power output have a close association (Enoka, 1996; St Clair Gibson, 2001). These findings support the central regulation of exercise hypothesis proposed by Noakes (2008), and extended by Tucker (2009), who argued that when a lack of end-point information is available, downregulation occurs in order to sustain exercise for prolonged periods. Furthermore, the failure to observe large changes suggests that athletes are able to monitor their energetic resources and expenditure in some way. Notably, it seems that this phenomenon is not limited to elite athletes, but also to the sub-elite. Thus, it has been hypothesised that this monitoring process

is an ability learnt relatively early on by athletes via experience (Foster *et al.*, 2003). It is conceivable that had participants performed the unknown trial multiple times, performance would have improved as experience and knowledge of the exercise bout grew.

Heart rate increased progressively during the unknown trial. Eston *et al.*, (2012) conducted a dual investigation in which both running and cycling were examined in a manner similar to the present study. Interestingly, both running and cycling saw a progressive increase in RPE and heart rate, almost identical to that of the current study. Since power output and performance time remained relatively constant, the increasing heart rate is attributed to cardiovascular and metabolic drift associated with the exercise (Coyle and Gonzalez-Alonso, 2001). Heart rate at the 500m interval was significantly lower than the 1000m, 1500m and 2000m intervals in the present study. Additionally, the recording at the 1000m interval was significantly lower than that noted at the 2000m interval (Figure 7, page 46).

The low heart rate value of 166 bts.min^{-1} at 500m and the comparatively low maximal value of 177 bts.min^{-1} indicate that participants were downregulating. Another study, in which participants were not told how long a running task would last, found that heart rate gradually increased over the course of the trial, as did RPE (Baden *et al.*, 2005). The pattern of RPE reported indicates that participants perceived the start of the trial as a lower intensity of effort. An investigation exploring exercise of unknown duration during 4000m time trial cycling also found an increase in RPE over the course of the unknown condition, however heart rate data was not presented (Williams *et al.*, 2012). The responses of heart rate and RPE support the findings that performance time, heart rate, power output and muscle activity of the erector spinae, biceps femoris and quadriceps femoris were also reduced.

The RPE values reported for this trial expressed a similar pattern to that of heart rate. In fact, exactly the same intervals were significantly different, namely the 500m interval had a significantly lower RPE than the 1000m, 1500m and 2000m intervals. The 1000m interval was also significantly lower than the 2000m interval. Reduced RPE and heart rate in the current study indicates anticipatory regulation as this allows the avoidance of premature fatigue due to the uncertainty in the end-point (Eston *et al.*, 2012). Furthermore, the mismatch between heart rate and power output responses

supports the anticipatory model of fatigue as explained by St Clair Gibson *et al.* (2006) and Marino (2014).

Williams *et al.* (2014) propose that when exercise duration is unknown, athletes tend to have a greater dependence on afferent feedback. Ultimately, it seems that when end-point information is unknown, it prevents athletes from successful exercise regulation and pacing by causing a mismatch between maximal RPE values and the end of exercise (Williams *et al.*, 2014).

Deceptive Trial: Complete Trial

The deceptive trial indicated no clear overall pattern. Therefore, it is proposed that a variable pacing strategy was adopted during this trial. Variable pacing strategies are characterised by effort and intensity fluctuations over the course of an exercise bout (Abbiss and Laursen, 2008). The fluctuations are usually in response to some changing condition during the race (e.g. geographical). However, in this case the reason for the fluctuation is probably the change in the end-point information as large differences were observed in the measures once the deception was revealed (Williams *et al.*, 2014).

According to the anticipatory feedback model, when actual exercise duration exceeds expected exercise duration, performance will be negatively affected (Tucker, 2009). Due to the expectation of a shorter exercise bout, factors such as initial work rate and rate of RPE increase will be selected at a pace too high to maintain for the actual exercise length (Tucker, 2009). Since participants thought that the trial would only be 1000m, the first two intervals of the deceptive trial indicated a high level of effort and intensity. This was observed in performance time (Figure 5, page 40), heart rate (Figure 7, page 46) and power output (Figure 8, page 49) as well as in the muscle activity of the medial deltoid (Figure 9, page 52), erector spinae (Figure 10, page 55), biceps femoris (Figure 11, page 58) and quadriceps femoris (Figure 12, page 61).

In all instances, there was a significant reduction in effort and intensity once the deception was revealed at the 1000m mark. This downregulation is indicative of the mismatch between the template RPE and the actual RPE of the participants. The results obtained during the deceptive condition are in agreement with those of Billaut

et al. (2011), who examined the effects of deception on short, repeated sprints in cyclists. The results indicated that when the cyclists were deceived and told they would be performing fewer sprints, muscle recruitment and power output were increased for those sprints. However, athletes then suddenly downregulated when they were told to complete more sprints than expected. Similarly, downregulation occurred in the study by Baden *et al.* (2004) once the deception was revealed, however power output and muscle activity were not measured and this was indicated by oxygen consumption. Eston *et al.* (2012) observed similar to results to those of Baden *et al.* where oxygen consumption decreased after the deception was revealed. This is attributed to greater economy of the athletes, since the intensity was controlled (Eston *et al.*, 2012). These results suggest that in anticipation of shorter exercise distance and duration, athletes enhance muscle recruitment, which causes a greater power output and performance for the first part of the trial. However, once the deception is revealed less muscle is recruited causing a reduction in performance indicative of down-regulation (Eston *et al.*, 2012).

It is hypothesised that in the scenario arising in the current study, participants began the exercise bout at an intensity which was too high due to the mismatch between the expected exercise length and the actual exercise length. This resulted in premature fatigue, indicated by a decline in power output which ultimately lead to a reduced performance. A decline in power output and force production is commonly observed during high intensity exercise, however the reasons for this decline are likely multifaceted and the possibility that a single reason exists to explain decreased performance is unlikely (Mauger, 2014; Peltonen *et al.*, 1997). However, it is probable that some "critical factor" causes an increased probability of premature fatigue. There are likely many of these "critical factors" which may result in decreased performance, some examples being elevated environmental temperature, athlete dehydration or (as in the current study) the mismatch between the expected exercise task and the actual exercise task.

During the deceptive trial, RPE was highest at the 1000m interval, just after the deception was revealed. This corresponds to a study conducted by Baden *et al.* (2005) who found that RPE was highest after participants were informed they would be continuing exercise longer than expected. Baden *et al.* attribute this finding to negative affective processes in response to an unexpected increase in exercise duration akin

to a sudden mismatch between the anticipated and required exercise duration. A similar study found that RPE increased following the reveal of the deception during both cycling and running, but subsequently decreased (Eston *et al.*, 2012). It has been demonstrated that individuals who are attempting to regulate conscious and subconscious emotions may incur a greater metabolic strain (Wilson *et al.*, 2012). Negative emotions about the deception which had taken place are likely to have been experienced by participants and this may have affected RPE reports (Baden *et al.*, 2005). A similar effect may have occurred during the deceptive trial of the current study as RPE was elevated after participants were told to continue for another 1000m. However, it must be noted that the increased RPE may also be due to an increase in physical exertion as power output at this point was also high (Figure 8, page 49). The RPE of the control trial followed a similar pattern to that of the deceptive trial, with the exception being that the deceptive trial was higher than the control trial at 1000m and *vice versa* at 2000m. It is likely that these results were observed due to the end-spurt phenomenon, since participants thought the exercise was ending at 1000m in the deceptive trial and so exerted themselves maximally at this point. This subjective measure of physical exertion indicates that participants were nearing exhaustion at the 1000m mark during the deceptive trial and 2000m mark during the control trial.

Swart *et al.* (2009) contend that the RPE template is not a fixed construct, but rather is dynamic and adaptable. This could explain the results of the deceptive trial, as the resulting mismatch between the actual RPE and template RPE would have caused alterations to the template RPE by athletes (Paterson and Marino, 2004). This alteration may have allowed athletes to continue exercising even though a seemingly maximal effort had already been produced. The change in the template RPE may have caused a decrease in neural drive, resulting in a reduced performance, but ultimately allowing exercise to continue.

Deceptive Trial: First 1000m vs Second 1000m

A comparison between the first and second half of the deceptive trial is necessary as results appear to partly mimic those in the control and unknown trials. To the author's knowledge, no study has investigated this factor of deception and corresponding literature is scarce.

The responses during the first 1000m of the trial was similar to the control trial. While there was no significant difference in performance time, the deceptive trial was marginally faster at 1000m compared to the control trial. Heart rate was similar to the control trial and was not significantly different at the 500m and 1000m intervals compared to the other deceptive trial intervals. Muscle activity was significantly higher in the medial deltoid at 500m as well as the biceps femoris at 500m and 1000m compared to the rest of the deceptive trial intervals. This suggests that the increased power output was due to both the upper and lower body muscles' engagement.

Additionally, it seems the upper body fatigues faster during rowing as the significant decline was only found at 500m in the medial deltoid. RPE values were not significantly different at 500m and 1000m, however the deceptive trial was rated slightly more difficult than the control trial at 1000m.These results indicate that the pattern observed in the control trial was followed closely by the first 1000m of the deceptive trial. This is to be expected as athletes knew (or thought they knew) the end-point of the exercise and so similar responses are anticipated.

The second 1000m produced similar responses to the unknown trial. This similarity is most likely because, following the deception, participants did not fully trust the investigator and downregulated in anticipation of further deception. Performance time was reduced after the deception was revealed and decreased below that of the unknown trial, being significantly slower at the 1500m interval compared to the first 1000m of the trial. While RPE was significantly higher at 1500m and 2000m compared to the start of the deceptive trial, the pattern was similar between the trials as both began levelling off as the trial progressed. Heart rate was not significantly different at any interval; however, it was higher than the unknown trial for the first 1000m and then dropped below corresponding values of the unknown trial after the deception was revealed. Power output fell throughout the deceptive trial, but was still significantly higher than the unknown trial at 1500m. There was no significant difference at 2000m indicating the magnitude of the reduction during the deceptive trial. Erector spinae muscle activity was significantly higher at 1500m and 2000m. This was the only significant difference in muscle activity and may indicate that at lower intensities, rowers recruit more core muscles. After the deception was revealed, results mimic those of the unknown trial during which exercise end-point was unknown. Thus, the

deceptive trial findings seem to be something of a combination of the control and unknown trials.

Some of the largest differences from the deceptive trial were found between the 1000m and 1500m intervals. Performance time, heart rate and power output decreased significantly between these intervals. Eston *et al.* (2012) suggest the reduction in effort may be linked to uncertainty in the end-point and stress due to possible further deception, causing athletes to subconsciously regulate physiological resources in order to conserve energy and enable completion of the exercise task. Indeed, this regulation seems to be centrally controlled as medial deltoid, erector spinae, biceps femoris and quadriceps femoris were all found to have a significant decrease in muscle activity between these intervals. While RPE did increase between 1000m and 1500m, this increase was not significant which contrasts with results presented by Baden *et al* (2005) and Eston *et al.* (2012) who found that RPE increased significantly after the deception was revealed during treadmill running and ergometer cycling. It is possible that the sub-elite athletes in the current study incurred a rate of RPE increase which was too great to allow observation of a significant increase at this interval i.e. RPE was already too high at 1000m for a significant increase to occur after this point. It is also plausible that the increase in RPE was not observed due to the interval lengths. Eston *et al.* (2012) note that the increase in RPE following the deception was short lived, therefore smaller intervals would be more sensitive to this.

EFFECTS BETWEEN TRIALS

The performance time of the control trial was significantly ($p<0.01$) faster than both the unknown and deceptive trial (Figure 4, page 39), which has been shown by others (Baden *et al.*, 2005; Billaut *et al.*, 2011; Mauger *et al.*, 2009; Mauger *et al.*, 2010). In these studies, correct feedback and end-point information were crucial to successful performance. In the control trial, participants were correctly informed of the distance to be completed, however it must be noted that distance feedback was not given. This result corresponds with the hypothesis of Tucker (2009) since the RPE template was not disrupted during the exercise bout, therefore permitting optimal performance.

The anticipatory feedback model proposes that when athletes expect exercise of a shorter duration, factors such as initial work rate and the rate of RPE increase will be elevated (Eston *et al.*, 2007; Tucker, 2009). However, this creates a mismatch between the actual exercise bout and the expected exercise bout, which results in decrements in performance since the athlete is not able to create an accurate template RPE (Tucker, 2009). It is likely that athletes will over exert themselves before the end of the exercise bout since exercise of a shorter duration is expected, ultimately resulting in a large reduction in intensity or complete cessation of exercise to prevent any catastrophic failure of homeostasis (Swart *et al.*, 2009). While the mean performance time for the deceptive trial was longer than that of the control trial, interval analysis show that the first 1000m of the deceptive trial was performed faster than the control trial (Figure 5, page 40). However, performance was significantly reduced at the 1500m and 2000m intervals compared to the same intervals of the control trial. The degree to which performance is affected depends on the athletes' ability to identify the discrepancy and complete the necessary changes to correct this discrepancy as well as the extent to which the over-exertion has occurred (Tucker, 2009).

At every 500m interval the unknown trial RPE values were significantly lower than the corresponding control and deceptive values (Table page 44), again indicating a perceived reduction of work rate by the athletes. Edwards and Polman (2013) note that when athletes are confronted with adverse, unfamiliar conditions (such as exercise with an unknown end-point) voluntary power output is reduced. The subconscious goal being the maintenance of a reserve in anticipation of the uncertainty of the end-point (Baden *et al.*, 2005; Williams *et al.*, 2014; Billaut *et al.*, 2011). Whether this reduction in workload was consciously or sub-consciously regulated is beyond the scope of this study, however it is likely that both elements play a vital role in this regulation (Edwards and Polman, 2013). Likewise, heart rate and power output were significantly higher in the control trial compared to the unknown trial. Reduced performance in the unknown trial was to be expected, since it has been found that athletes reduce work rate and utilise resources more economically when the end-point of exercise is unclear (Edwards and Polman, 2013). At the onset of exercise, a low work rate is adopted which produces a slow increase in RPE and therefore, allows the maintenance of a physiological reserve (Tucker, 2009). This corresponds to RPE values obtained during the unknown trial.

RPE during the deceptive trial followed a similar pattern to that of performance. At the 1000m interval, RPE was greatest in the deceptive trial. This reading was obtained just after participants had been informed that another 1000m was required to be completed. This may be considered a limitation and RPE should be obtained at more regular intervals, but particularly just before and just after the deception is revealed. This would enable conclusions to be drawn on the impact of deception on RPE. While the relationship between RPE and power output is complex, it is known that the gain of the relationship is altered depending on the duration of the exercise bout (Eston *et al.*, 2007). Since participants believed they were would be exercising for a shorter duration than they really were, RPE was allowed to increase at a rate which could not be maintained.

In a study conducted by Baden *et al.* (2005), RPE significantly increased in comparison to a control trial, after participants were told they would need to complete more exercise than was originally expected. While a similar result is depicted in Figure 6 (page 43), the difference was not significant compared to the control trial. Interestingly, the power output in the deceptive trial at the 1000m interval was significantly higher than corresponding interval of the control trial, while RPE was not significantly different at this interval. This indicates that while participants perceived to be working at a similar intensity, they were in fact, not. There are three possible explanations for this. The first is that, as proposed by Baden *et al.* (2005), some sort of affective or emotional processes may be contributing to the RPE value. For example, it is possible that some negative emotion about the deception which had taken place as well as the mismatch between the anticipated and actual exercise duration may have altered the RPE value of the participants. Secondly, this disparity could be due to the exercise intensity. Coquart *et al.* (2011) note that when high exercise intensities are utilised (over 80% of maximal treadmill running speed), deception has little or no effect on RPE. It is suggested that the afferent signals derived from such high intensity exercise may override any psychological influence of effort perception. A similar effect may have occurred in the current study as participants were exercising at a high intensity during both the control and deceptive trials. This is indicated by heart rate during the trials (Figure 7, page 46). The third possible explanation for this discrepancy is that, while participants had been exposed to the RPE scale before the testing, they may not have completely grasped the concept and

this may have led to incorrect values being reported (Coquart *et al.*, 2011). It has been suggested that combining the RPE scale with a subjective estimation of exhaustion time (Estimated Time Limit Scale) may provide a more complete understanding of perceptual responses during exercise (Coquart *et al.*, 2011).

Muscle activity of the quadriceps femoris and biceps femoris was lower compared to the control trial, however significant differences were only found in the quadriceps femoris muscle. Whether muscle activity increases or decreases in this kind of scenario is a subject of debate in the literature. Both increases (Billaut *et al.*, 2011) and decreases (Elliot, 2014) in muscle activation have been found to occur in studies of similar deception, where performance was improved. The fundamental factor that should be considered is overall performance as this determines the success of the athlete. However, muscle activity provides insights as to why performance may or may not be improved. For example, Elliot (2014) suggested that the attainment of improved performance may be coupled with a lower muscle activation. In contrast, Billaut *et al.* (2011) found higher muscle activity in trials where performance was best. These contrasting views may be due to a lack of similar research as the current study appears to offer evidence for both cases.

The medial deltoid exhibited muscle activity higher in the unknown trial than that of the control trial (where performance was best), while erector spinae muscle activity was highest in the unknown trial. Conversely, muscle activity was decreased in the quadriceps femoris and biceps femoris during the unknown trial compared to the control trial. These differences may be attributed to the use of different muscle groups when rowers are exerting themselves maximally. Rowers tend to use different muscles to varying degrees when rowing maximally as opposed to when rowing sub-maximally (Gerzevic, Strojnik and Jarm, 2011). These authors found changes in the vastus lateralis, latissimus dorsi, biceps brachii and rectus femoris muscles. As several of these muscles were not measured during the present study, it is difficult to draw direct comparisons. However, it supports the notion that different muscles may have been recruited during the trials, thereby causing discrepancies in the EMG results. Furthermore, EMG may respond differently depending on the cellular make of the muscle (Peltonen *et al.*, 1997). For example, muscle with predominantly fast twitch fibres may respond differently to those with predominantly slow twitch fibres (Peltonen *et al.*, 1997). However, individual muscle make-up and behaviour was beyond the

scope of this study. In the unknown trial, rowers were not fully exerting themselves and this may be another reason for the disparities found in the EMG data. Finally, the four muscles which were monitored in the current study are not solely responsible for force production during rowing. It is therefore, plausible that power output (and consequently, performance) may have been altered by increased or decreased activation of muscles which were not monitored. This factor may also explain possible disparities between the obtained EMG results and performance.

Muscle activity during the deceptive trial was varied amongst the different muscles possibly because rowers use different muscles groups when rowing at different intensities (Gerzevic *et al.*, 2011). In addition, different muscles groups may fatigue at different rates dependant on factors such as muscle fibre type (Peltonen *et al.*, 1997). Activity of the medial deltoid was greatest during the first 1000m of the deceptive trial (Figure 9, page 52). A significant decrease was then measured until the 2000m distance was completed. A similar muscle recruitment pattern was observed in the erector spinae muscle during the deceptive trial where the 500m and 1000m intervals had significantly higher muscle activity compared to the 1500m and 2000m intervals (Figure 10, page 55). Interestingly, muscle activity of the erector spinae during the final 1000m of the deceptive trial was significantly lower than that of the corresponding interval of the unknown trial. During the first 1000m of the deceptive trial, the biceps femoris muscle had a significantly higher muscle activity compared to corresponding intervals of both the control and unknown trials (Figure 11, page 58). Again, a significant reduction was observed after the deception was revealed at the 1000m mark. Finally, the quadriceps femoris muscle showed similar muscle activation to that of the control trial during the first 1000m (Figure 12, page 61). However, a significant reduction in muscle activity occurred over the course of the trial, except between the 1500m and 2000m intervals. A reduction in muscle activity following the deception may indicate a stronger presence of subconscious control (St Clair Gibson *et al.*, 2003). Due to the change in the exercise bout requirements, it is possible that participants relied more on subconscious processes and perceptions to become more economical and ensure task completion. This occurred by reducing muscle activation.

The results of the muscle activity support the hypothesis that different muscle groups are recruited during different intensities of rowing (Gerzevic, Strojnik and Jarm, 2011). For example, during the unknown trial performance was reduced, while muscle activity

of the erector spinae was highest, particularly in the final 1000m. The same pattern as observed in the medial deltoid muscle over the last 1000m. Indeed, the control trial had a consistently lower muscle activity in the medial deltoid over the course of the entire trial, while performance was greatest. It is thus plausible that rowers utilise different muscle groups depending on fatigue and the requirements of the exercise bout. Tentatively, it is proposed that when rowing maximally (such as during the control and first 1000m of the deceptive trial), rowers recruit a larger lower limb muscle mass, while when rowing sub maximally (such as during the unknown and final 1000m of the deceptive trial) more upper body muscle mass is recruited. However further research into this area is required to provide more evidence for this conclusion.

Power output was also significantly lower in the unknown trial compared to the control and deceptive trials (Figure 8, page 49). There is a linear relationship between the force produced by a muscle and its electrical activity (Vidacek and Wishner, 1971). Therefore, a resultant reduction in power output is not surprising with a lower measured muscle activity in the unknown trial. As early as 1971, Videcek and Wishner recognised that expected task duration influences muscle activity. They proposed that in order to limit fatigue, an individual performs the task which is expected to be longer more economically in order to suppress feelings of fatigue.

Heart rate was significantly lower in the unknown trial compared to the control trial as well as the first 1000m of the deceptive trial (Figure 7, page 46). Baden *et al.* (2005) did not find a change in heart rate in the unknown trial and this may be due to differences in the exercise mode as well as intensity between the two studies. Athletes in the Baden *et al.* study became more economical and this is attributed to the theory of maintaining a reserve in anticipation of a longer exercise bout and greater physiological demand. Conversely, Eston *et al.* (2012) performed a nearly identical study to that of Baden *et al.* and found that heart rate was reduced in the unknown trial as in the present study. This supports the notion that exercise mode and intensity may be causing the disparity between results of these studies. Further research is required to conclude this reliably however.

Mean heart rate data of the control trial was highest at every interval over the trial, however during the first 1000m there was no significant difference between the deceptive trial and the control trial (Figure 6, page 43). Mean power output was

significantly higher in the deceptive trial compared to the control trial at this interval. The increased power output without a corresponding increase in heart rate is unexpected, but may be due to natural variations in heart rate from day to day, since the difference was small (±3-4 bts.min^{-1}). Heart rate decreased significantly in the deceptive trial after participants were informed they would be continuing for another 1000m. During this interval, the heart rate of the deceptive trial was significantly lower than the control trial. A similar observation was made with power output during this interval. At the 2000m mark, power output in the deceptive trial was also significantly lower than the control trial. This is indicative of the downregulation occurring following the participants being told they would need to continue exercise beyond what was originally expected. Over the course of the deceptive trial, power output showed a similar response pattern to that of heart rate. Power output was the greatest of all trials during the first 1000m after which it dropped below the control trial. Once again, this is indicative of the downregulation of participants after they were told that another 1000m would have to be completed. Consistent with previous literature, it seems that deception manipulating the knowledge of the end-point is expressed primarily through the subjective measure of RPE (Williams *et al.*, 2014). Furthermore, this reaction seems to only be realised after the announcement of a change in duration. Since physiological mechanisms such as heart rate were not radically different, it points toward the common theme of psychological emotion being a critical factor (Williams *et al.*, 2014).

Thus, in the unknown trial, a lower mechanical output profile was achieved by the rowers. This was due to a decreased neural drive, causing a lower muscle activation (in the biceps femoris and quadriceps femoris muscles, muscles of the lower limb). This resulted in a lower rating of perceived exertion and reduced heart rate. These factors were a product of an inferior pacing strategy, which ultimately caused suboptimal performance. For example, the highest erector spinae muscle activity (Figure 10, page 55) was at the 1500m and 2000m intervals of the deceptive trial, while performance at these intervals of the same trial was decreased.

A possible limitation to the present study was the motivation of the athletes who participated. According to Tucker and Collins (2012), athletes may possess intrinsic factors which predispose them to have greater motivation. Furthermore, it is well accepted that a high level of motivation results in superior performances (Billaut *et al.*,

2011; Tucker and Collins, 2012). While it is difficult to control a psychological factor such as this, it is conceivable that motivation may have impacted on the responses obtained from participants.

CHAPTER VI

SUMMARY, CONCLUSION AND RECOMMENDATIONS

The control trial exhibited a significantly (p<0.05) faster performance time than both the unknown and deceptive trials. The unknown trial (439.45 ±32.18s) began at a slower pace which was maintained. The deceptive trial (437.00 ±34.86s) had a fast pace until the 1000m distance after which there was a significant reduction in speed. The control trial (427.73 ±34.81s) exhibited the reverse J-shape pacing profile, where both the start and end were faster than the middle 1000m (Figure 5, page 40).

Within the control trial, muscle activity exhibited a similar pattern to that of performance. In addition, the highest RPE value (18.82) was obtained at the 2000m mark of the control trial. A similar pattern was observed with heart rate over the course of the trial which increased linearly with time, reaching a maximal value of 183 bts.min^{-1} at the end of the trial. Power output decreased by approximately 20 watts each 500m during the control trial (Figure 8, page 49).

In the unknown trial, heart rate increased over the course of the trial, but was significantly (p<0.05) lower than the control trial at each 500m interval (Figure 7, page 46). There was a consistent pace in power output and thus performance at each 500m interval (Figure 8, page 49). This pattern was also observed in the muscle activity variables. During the unknown trial, RPE was consistently the lowest of the three conditions (Figure 6, page 43).

No clear pattern was evident during the deceptive trial and this is indicative of a variable pacing strategy as it was characterised by fluctuations in effort and intensity. This was to be expected given the deceptive nature of the trial. The first 1000m of the deceptive condition indicated high effort and intensity as expressed in the performance time (Figure 5, page 40), power output (Figure 8, page 49) and muscle activity measures. There was a significant reduction in these variables when the deception was revealed. RPE increased over the course of the trial peaking (18.36) at the 2000m interval (Figure 6, page 43). Heart rate peaked at the 1000mm interval (177.45 bts.min^{-1}) after which there was a large decrease (Figure 7, page 46).

Between trials, power output, RPE and heart rate values were significantly (p<0.05) lower in the unknown trial compared to the deceptive and control trials. Only the quadriceps femoris muscle (Figure 12, page 61) had a significantly (p<0.05) lower muscle activity compared to the control trial. The medial deltoid (Figure 9, page 52) and erector spinae (Figure 10, page 55) muscles had higher muscle activity in the unknown trial. The unknown trial was characterised by a lower mechanical output with reduced muscle activity, particularly in muscles of the lower limb. This resulted in reduced heart rate and RPE scores and ultimately lead to suboptimal performance.

The rowers' perception of the deceptive trial followed the same pattern as the performance time. At the 1000m mark RPE was highest, thereafter falling below the control trial (Figure 6, page 43). Heart rate during the deceptive trial was similar to that of the control trial, however the control trial had a significantly (p<0.05) higher heart rate after the 1000m interval (Figure 7, page 46). Muscle activity during the deceptive trial was varied. The medial deltoid exhibited the highest muscle activity during the first 1000m of the deceptive trial (Figure 9, page 52). Following that there was a reduction in muscle activity. Erector spinae activity followed a similar pattern during the deceptive trial (Figure 10, page 55). The quadriceps (Figure 12, page 61) and biceps femoris (Figure 11, page 58) muscles had a high muscle activity for the first 1000m, but once the deception was revealed there was a significant reduction in muscle activity.

Conclusion

The results of this study indicate that accurate end-point information resulted in improved performance, while deceptive information produced reduced performance and a complete lack of end-point knowledge caused the worst performance. Furthermore, the reverse J-shape pacing profile was evident during the control trial. Training practices may be improved by deceiving athletes or by ensuring the endpoint is known, depending on which strategy positively affects performance. For example, this study found that knowledge of the endpoint improved performance, therefore training methods can be improved by ensuring that athletes regularly train the distance in which they will compete. Conversely, to prevent performance decrements, athletes

should always be informed of the distance to be covered before an event starts as when endpoint information is unknown or incorrect, performance is worse.

Recommendations

Future studies examining pacing strategies should take the following factors into consideration.

A larger sample size should be recruited to increase the statistical power of the study. Participants numbers were limited by the number of athletes in the Rhodes University Rowing Club. In order to maintain homogeneity, a further suggestion is that the ability and skill of the rowers be strictly controlled as rowers of different calibre and experience may pace themselves differently.

Future studies should measure several muscles from all muscle groups in the body, since rowers employ many muscles during a stroke and the utilisation of these muscles may change during an exercise bout. This would strengthen the validity of the study. An attempt was made to do this in the present study by measuring a muscle from each major muscle group, as capturing EMG data from more muscles was not feasible.

There are also inherent limitations associated with using EMG. For example, electrodes are required to be placed in precisely the same location for accurate results. An attempt was made to control for this by marking the areas where electrodes were placed. Additionally, EMG interference may also be caused by the dynamic nature of the exercise and excessive sweating of participants. This effect was minimised by securing the wires and electrodes with adhesive tape. However, if it is possible to circumvent the use of EMG in future studies this is advised, perhaps by using a neuromuscular performance test.

It is noted that laboratory time trials may not necessarily represent the optimal pacing strategy for a given event as factors such as environment and competitors may influence pacing. Furthermore, it is recommended that on-water rowing should be studied and compared to ergometer rowing as these two types of exercise may elicit different responses. On-water rowing during actual races would provide the most

accurate and reliable results. The laboratory scenario did not accurately represent some factors which may impact a rower during an on-water race. For example, psychological and environmental factors.

Statistical analysis could have been improved by using magnitude based inferences. This is a more contemporary approach to analysing results. Conventional statistics and effect sizes were included, however a stronger interpretation could have been made with magnitude based inferences. The smallest worthwhile change should have been calculated for all variables.

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APPENDICES

Appendix A: General Information

- Ethical Approval
- Pre-Protocol Information Letter
- Pre-Consent Form
- Confidentiality Declaration
- Post-Protocol Information Letter
- Post-Consent Form
- Pre-test Instructions to Participants

Appendix B: Data Collection

- Order of Procedures
- Rating of Perceived Exertion Scale and Explanation
- Data Collection Sheet

Appendix C: Ethical Application Forms

- Ethics Application

Appendix A: General Information

Human Kinetics and Ergonomics

Application for Ethical Approval

RHODES UNIVERSITY
Where leaders learn

Student Name: Dean Ferreira

Type of Research: MSc

Project Title: **The impact of pacing on the performance of experienced rowers during rowing ergometer**

Supervisor: Dr CJ Christie

Application received: 4 March 2015

Re-submission received: **7 April 2015**

Reference Number: **HKE-2015-3**

Approved	Approved, on condition that suggestions have been effected ✓	Request for rework and resubmission	Rejected

Signed

JE Viljoen

Chair: Human Kinetics and Ergonomics Ethics Committee

PRE-PROTOCOL INFORMATION LETTER

Department of Human Kinetics and Ergonomics

Cell: 083 559 6121 E-mail: g10f2864campus.ru.ac.za

Fax: (046) 603 8934

Dear:_____

Thank you for offering to participate in my Master of Science degree research project entitled:

The impact of pacing on the performance of experienced rowers during rowing ergometry.

The purpose of this study is to identify the physiological mechanisms pertaining to the regulation of the pacing strategy employed by rowers. An ergometer will be set up in one of the laboratories and trials will be performed, mimicking a performance assessment/race. The nature of the trials has been specifically designed to replicate those expected during rowing. Therefore, as a participant in this study, you will be

required to perform maximal ergometer rowing. The distance to be rowed will not exceed an average training session. However, in order to assess pacing strategies, deception is sometimes necessary. If deception does occur, it will only be because the information required cannot be obtained in any other manner.

Procedures

As a participant in this study, you will be required to perform a total of four sessions, of which three will be experimental trials and one an introductory session. All sessions will take place in the Human Kinetics and Ergonomics Department.

The first session will be an introductory and habituation session where you will receive an explanation of the testing procedures, requirements and aims of the project. You will then be asked to sign a consent form, indicating you are comfortable with the procedures and indicating your willingness to participate in the study. After the consent form has been signed, you will receive a participant code to ensure all data is kept confidential and anonymous. Demographic and anthropometric data will then be collected, such as your age, stature and mass. Following this, you will be habituated to the procedures, equipment and scales to be used during the experimental trials. Electromyography (EMG) electrodes will be placed on certain muscles and you will be allowed to row until you are comfortable with the equipment. The EMG equipment is not invasive and allows measurement of muscle activity from the surface of the skin. An explanation of the Borg Rating of Perceived Exertion scale will then also take place. This session should not take longer than 30 minutes.

After the introductory and habituation session, you will be required to complete three experimental sessions over the course of several days. During each session, one of 3 trials will be completed. You are required to wear the exact clothing/tri-suit as per a normal performance assessment, including shoes. Resting heart will be recorded, after which you will perform a light warm-up mimicking that before a race. The electrodes will then be placed on the specific muscles and once resting heart rate has

been reached, the trial will begin. During the trial, RPE ratings, heart rate, power output, time and EMG data will be collected at certain intervals.

Risks associated with the study:

1. **Muscle/Joint injury –** The study requires that you perform maximal ergometer rowing over a certain distance. This subjects the body to repetitive, high intensity muscle contractions, which may result in delayed onset muscle soreness (DOMS) for 4 – 7 days. If muscle fiber damage is severe, injury may occur however, the likelihood of severe muscle damage is extremely low as you are well trained rowers. To compensate for this, there will be at least a 7 day gap between each experimental trial. This will prevent DOMS affecting your performance and also reduce the risk of injury. If you are still experiencing DOMS following the rest period, additional rest days will be provided. The warm-up before the exercise will also serve to reduce the risk of injury. However, being an experienced rower, you and your body should be familiar with the type of exercise required. If you do feel pain or discomfort of any kind, at any point, please inform the researcher.

2. **Ergometer injury**

   The ergometer will be secured to the floor in order to prevent it slipping or moving which may cause injury. The drag factor will be set at 125, consistent with the training drag factor you use. In this way, you will not be straining your body any more than during a normal training session.

3. **Skin irritation**

   In order to ensure optimal contact during testing, the area of skin where the electrode is to be placed will be shaved. An alcohol swab will be used to prevent infection. An aqueous electrode conduction cream will then be placed on the skin to optimize the signal from the muscles. This cream may produce minor irritations on the skin, in which case the cream will be removed.

Benefits Associated with the Study

Participation in this study will provide you will knowledge about your anthropometrical and morphological data. You will be exposed to several procedures as well as equipment which will increase your understanding of the nature of sports science research, specifically in rowing. Additionally, you will gain greater comprehension of the impact of pacing strategies on performance and thus, may be able to improve your own performances.

Additional Information

You are free to withdraw from the study at any point. The information obtained during the sessions will be kept anonymous and will only be used for statistical purposes. Data will be kept in the Human Kinetics and Ergonomics Department and may be used for future research, however your information will always be kept confidential. You will receive feedback from the researcher once the study is complete.

Thank you for your time and for showing interest in this research. If you have any questions regarding any aspect of the investigation please contact me and I will happily answer any questions you may have.

Yours sincerely

Dean Ferreira

Human Kinetics and Ergonomics Department

083 559 6121

g10f2864@campus.ru.ac.za

PRE-TRIAL CONSENT FORM

Department of Human Kinetics and Ergonomics

Cell: 083 559 6121 Fax: (046) 603 8934 E-mail: g10f2864@campus.ru.ac.za

PARTICIPANT CONSENT FORM

I, _____, agree that I have been informed as far as possible, both verbally and in writing, of the procedures required in this research project entitled:

The impact of pacing on the performance of experienced rowers during rowing ergometry

I have read the information sheet and fully understand the testing procedures that will take place during this study. I understand that the aim of this study is to identify the nature of pacing strategies and that each condition will differ. I am aware that deception may need to be employed in this type of research. The risks and benefits associated with the study have also been brought to my attention. I have further been given the opportunity to ask questions or express my concerns regarding the testing procedures. By voluntarily consenting to participate in this research project I accept responsibility together with the Human Kinetics and Ergonomics Department, whereby should injury or accident occur as a result of the protocol being performed, the Human Kinetics Department will cover any fees incurred and provide support to ensure my rehabilitation. If however injury it is shown to be self-inflicted, or not directly related to the study, the department will waiver any recourse against the researchers of Rhodes University. I further understand that all information gained from this study will be treated confidentially and that my anonymity will be protected at all times. I am aware that data obtained from this study may be used and published for statistical and scientific purposes. I realize that if I experience distress at any time during the study, I may withdraw.

I have read and fully understood that above information, as well as the information in the letter accompanying this form.

I therefore consent to voluntarily participate in this study.

PARTICIPANT (OR LEGAL REPRESENTATIVE):

_____	_____	_____
(Print name)	(Signed)	(Date)

PERSON ADMINISTERING INFORMED CONSENT:

_____	_____	_____
(Print name)	(Signed)	(Date)

WITNESS:

_____ _____ _____

(Print name) (Signed) (Date)

WITNESS:

_____ _____ _____

(Print name) (Signed) (Date)

CONFIDENTIALITY DECLARATION

Department of Human Kinetics and Ergonomics

Cell: 083 559 6121 Fax: (046) 603 8934 E-mail: g10f2864@campus.ru.ac.za

DECLARATION

I, _____, as a participant in the study entitled:

The impact of pacing on the performance of experienced rowers during rowing ergometry

recognize my responsibility in maintaining the confidentiality of this trial and hereby agree to the following:

- I will not reveal the nature of this trial to any other participant recruited for this study, until all experimentation is complete.

- I acknowledge that revealing this information would render this study null and void.

- I agree that obtaining this information is vital to the scientific progress in the assessment of competitive sport, in this case, rowing, and failure to obtain valid and reliable data during this study may negatively impact the sport itself.

- If asked by fellow participants, I agree that I am permitted to share that the trial consisted of rowing a certain distance, however I am not permitted to share the exact distance completed in this trial.

- I agree that this confidentiality agreement shall be binding up to and including the moment at which I receive an email from the researcher thanking the sample for participation in the study and thus declaring the research complete.

PARTICIPANT (OR LEGAL REPRESENTATIVE):

_____	_____	_____
(Print name)	(Signed)	(Date)

PERSON ADMINISTERING CONFIDENTIALITY DECLARATION:

_____	_____	_____
(Print name)	(Signed)	(Date)

WITNESS:

_____	_____	_____
(Print name)	(Signed)	(Date)

WITNESS:

_____	_____	_____
(Print name)	(Signed)	(Date)

POST-PROTOCOL INFORMATION LETTER

Department of Human Kinetics and Ergonomics

Cell: 083 559 6121 E-mail: g10f2864campus.ru.ac.za

Fax: (046) 603 8934

Dear:_____

Thank you for participating in my Master of Science research project entitled:

The impact of pacing on the performance of experienced rowers during rowing ergometry

The aim of this study was to investigate the pacing strategies employed by experienced rowers and in so doing, elucidate some of the physiological mechanisms pertaining to the regulation of exercise. It has been hypothesized that when a person exercises, their subconscious brain integrates information of how the athlete is feeling in the present and how the athlete expects to feel later in the race. By doing this, the brain down regulates activity to ensure that the athlete will finish the race. In an attempt to explain the mechanisms involved in preventing premature fatigue, the Central Governor Hypothesis has been proposed. This theory posits that exercise is regulated via an anticipatory feedback system, which means that the brain is constantly interpreting information from the senses and effecting changes based on these sensations. This ensures athletes are able to maintain the highest possible work output without fatiguing before the end of the exercise task, which is key to successful performances. This model integrates previous experience of the exercise task (i.e. the more often the exercise bout is performed, the better the performance), anticipation of the exercise distance/duration as well as physiological feedback throughout the event. Consequently, the body integrates this information and regulates performance via the pacing strategy.

You were selected to participate in this study as you are an experienced rower, well familiarized with the nature of the sport. In order to assess the type of pacing strategy adopted, deception was used in the final two trials. The researcher could not have told you about this deception at the start of the experiment as this may have resulted in the adoption of other types of pacing strategies. Therefore, deception was necessary in order to determine whether your previous experience in the sport allowed the adoption of a more suitable pacing strategy when exercise end-point was unknown or incorrect.

Once again, thank you for participating in this investigation. Please feel free to contact me if you have any questions.

Yours sincerely

Dean Ferreira

Department of Human Kinetics and Ergonomics

083 559 6121

g10f2864@campus.ru.ac.za

POST-CONSENT FORM

Department of Human Kinetics and Ergonomics

Cell: 083 559 6121 Fax: (046) 603 8934 E-mail: g10f2864@campus.ru.ac.za

PARTICIPANT POST-CONSENT FORM

I, _____, having participated in the study entitled:

The impact of pacing on the performance of experienced rowers during rowing ergometry

am now fully aware that deception was employed in two of the three trials, in attempt to adequately assess whether pacing strategies were adopted by the rowers participating in the study. I am also aware that without the use of deception, pacing

could not have been accurately assessed, as knowledge of an exercise end-point, and in this case the total distance to be completed, would essentially alter the rower's performance. By voluntarily consenting to participate in this research I accept responsibility together with the Human Kinetics and Ergonomics Department, whereby should injury or accident occur as a result of the protocol being performed, the Human Kinetics Department will cover any fees incurred and provide support to ensure my rehabilitation. If however injury it is shown to be self-inflicted, or not directly related to the study, the department will waiver any recourse against the researchers of Rhodes University. I further understand that all information gained from this study will be treated confidentially and that my anonymity will be protected at all times. I am aware that data obtained from this study may be used and published for statistical and scientific purposes. I am also aware that if I experience distress at any time during the study, I can withdraw immediately; in which case my data obtained during the experimental trials may not be used.

I have read and fully understood the above information, as well as the information in the letter accompanying this form, and any questions I may have had, have been answered by the researcher to my satisfaction.

PARTICIPANT (OR LEGAL REPRESENTATIVE):

_____	_____	_____
(Print name)	(Signed)	(Date)

PERSON ADMINISTERING THE POST- INFORMED CONSENT:

_____	_____	_____
(Print name)	(Signed)	(Date)

WITNESS:

_____	_____	_____
(Print name)	(Signed)	(Date)

WITNESS:

_____	_____	_____
(Print name)	(Signed)	(Date)

PRE-TEST INSTRUCTIONS TO SUBJECTS

On the day of testing, please inform the investigator of any factors which you think may impact your performance or results, for example if you are feeling ill or have taken any medication. Please ensure you have informed the investigator of any recent injuries. Finally, please adhere to the following guidelines pertaining to the day before and the day of the trial:

24 hours before trial:

- Do not consume alcohol.
- Do not take any medication such as aspirin, panado etc.
- Do not participate in strenuous exercise of any kind.
- Try to get at least 8 hours of sleep.

On the day of the trial:

- Eat a large, balanced meal 2 hours before testing.
- After the meal, do not consume any more food until after the trial.
- Hydrate adequately throughout the day.
- Void faeces and urine before coming to the research venue.
- Bring a water bottle and sweat towel to the testing session.
- Wear appropriate rowing clothing.

In order to gain valid and reliable results, it is critical that these instructions are adhered to as closely as possible. If for any reason you cannot comply with the above instructions please inform the investigator as soon as possible. Your cooperation is greatly appreciated.

Appendix B: Data Collection

ORDER OF PROCEDURES

Session I: Introduction and Habituation

1. Welcome, introduction and explanation of study
2. Explanation of equipment, techniques and measures
3. Provide and explain letter of information and informed consent forms
4. Provide time for reading and questions
5. Familiarise participants to all equipment
6. Ensure consent forms have been signed
7. Obtain stature and mass measures
8. Ensure participants complete PAR-Q and additional information sheet
9. Provide pre-test instructions to participants.

Session II, III and IV: Experimental Trials

1. Welcome participant, ensure pre-test instructions were followed.
2. Attach heart rate monitor, record resting value
3. Prepare areas for electrode attachment (shave and clean area)
4. Attach electrodes firmly with Elastoplast
5. Ensure Datalogger is functioning correctly
6. Allow participant to warm-up (dynamic stretching and low intensity ergometer rowing)

7. Perform calibration of Datalogger.
8. Ensure heart rate is within 10 beats of resting value
9. Begin experimental trial
10. Record all measures as per methodology
11. End experimental trial
12. Remove equipment
13. Administer confidentiality declaration if relevant
14. Schedule next testing session

Explanation of Ratings of Perceived Exertion

The rating of perceived exertion scale is a subjective estimate of the intensity at which you feel you are exercising. The values range from 6 to 20. A value of 6 corresponds to a feeling of resting, standing still, while a value of 20 corresponds to maximal effort. The rating is central in nature implying that it is a measure of the cardiovascular system i.e. heart and lungs. Throughout the experimental tests, you will be required to give your RPE.

rating	description
6	NO EXERTION AT ALL
7	
8	EXTREMELY LIGHT
9	VERY LIGHT
10	
11	LIGHT
12	
13	SOMEWHAT HARD
14	
15	HARD (HEAVY)
16	
17	VERY HARD
18	
19	EXTREMELY HARD
20	MAXIMAL EXERTION

DATA COLLECTION SHEET

Demographic Information:

Name:_____ Age:_____

Participant Code:_____ Order of Testing:_____

Rowing History:

Number of Years Rowing (including school, club etc.):_____

Number of Regattas Participated in (total):_____

Anthropometric Data:

Stature (mm):_____ Mass (kg):_____

BMI:_____

EXPERIMENTAL DATA COLLECTION SHEET

Participant Code:_____ Experimental Test: CT / UT / DT

Heart Rate

Resting	500m	1000m	1500m	2000m

Ratings of Perceived Exertion

Resting	500m	1000m	1500m	2000m

Total Power Output

_____

Average Power Output

500m	1000m	1500m	2000m

Performance Time

500m	1000m	1500m	2000m

Appendix C: Ethics Application

Deception

Deception studies have grown in popularity over recent years as a means to assess pacing strategies (Ansley *et al.*, 2004; St Clair Gibson *et al.*, 2006; Billaut *et al.*, 2011; Stone *et al.*, 2014). While the deception of athletes is not to occur without extreme

caution, it is the most accurate means by which pacing strategies can be examined. In the current study, if participants were correctly informed of the exercise end-point, the results would be invalid as they would be able to anticipate the exercise requirements and thus adopt a typical pacing strategy. Therefore, if participants had complete knowledge of the hypotheses and methodology of this study, it would render the results null and void.

In the current study, several measures have been used to ensure the safety and well-being of the participants. The participants are experienced rowers meaning they have a good understanding of the nature and requirements of the sport. They are highly trained in rowing and will be somewhere nearing their peak fitness at the time of testing. The protocol design results in the same distance being completed in each trial. Furthermore, the distance is that of a typical rowing race and participants will have completed far greater distances during training. All the measures mentioned above aim to ensure that participants are fully able to complete all three trials without great difficulty and without a significant risk of injury.

Additionally, data collected from the deceptive trials will not be used until participants have been fully informed of the deception which has taken place. Following this, participants will be asked if their data may be used and if they agree, analyses will begin. This process will be completed by the use of a post-protocol information letter and a post-consent form.

The order of the information to be given to participants is:

1. A pre-protocol information letter will be given to potential participants if interest is shown in the study.
2. A verbal explanation of the research will then be provided to potential participants.
3. Participants will then be required to sign a pre-trial consent form in order to partake in the research.

4. Following the unknown and deceptive trials participants will be required to sign confidentiality declaration form in order to ensure that the nature of the deception is not shared between participants.
5. Once participants have completed all three experimental trials, a post-consent form will be signed.
6. A post-protocol information letter will be also be provided at this time.

Risks and Benefits of the Research

Participant risk associated with the study:

1. **Deception –** In order to examine the factors related to pacing, participants must be deceived as knowledge of the end-point of the exercise would alter the pacing strategy employed by the participants. Therefore, it is necessary that deception be used and this may put the participants at risk. However, several measures have been taken to reduce the level of this risk as much as possible.

2. **Muscle/Joint injury –** The study requires that participants perform maximal ergometer rowing over a certain distance. This subjects the body to repetitive, high intensity muscle contractions, which may result in delayed onset muscle soreness (DOMS) for 4 – 7 days. If muscle fiber damage is severe, injury may occur however, the likelihood of severe muscle damage is extremely low as the participants are well trained rowers. To compensate for this, there will be at least a 7 day gap between each experimental trial. This will prevent DOMS affecting their performance and also reduce the risk of injury. If the participants are still experiencing DOMS following the rest period, additional rest days will be provided. The warm-up before the exercise will also serve to reduce the risk of injury. However, being experienced rowers, the participants and their bodies should be familiar with the type of exercise required. A physical activity readiness questionnaire will also be completed by the participants. This will indicate whether the participant is at an increased risk for a musculoskeletal injury, in which the participant will be excluded from the study.

3. **Ergometer injury**

   The ergometer will be secured to the floor in order to prevent it slipping or moving which may cause injury. The drag factor will be set at 125, consistent with the training drag factor participants use during training. In this way, participants will not be straining their body any more than during a normal training session.

4. **Skin irritation**

   In order to ensure optimal contact during testing, the area of skin where the electrode is to be placed will be shaved. An alcohol swab will be used to prevent

infection. An aqueous electrode conduction cream will then be placed on the skin to optimize the signal from the muscles. This cream may produce minor irritations on the skin, in which case the cream will be removed.

Benefits Associated with the Study

Benefits to participants:

Participation in this study will provide the participants with knowledge about their anthropometrical and morphological data. They will be exposed to several procedures as well as equipment which will increase their understanding of the nature of sports science research, specifically in rowing. Additionally, the participants will gain greater comprehension of the impact of pacing strategies on performance and thus, may be able to improve their own performances.

Benefits to the knowledge base:

Pacing strategies are vital to optimal athletic performance. Scientific knowledge of the mechanisms and factors influencing the pacing strategy are limited and the concept is poorly understood. Thus, by studying the pacing strategies employed by rowers, it is possible that some of these factors may be elucidated. This may provide information which could inform coaches, athletes and fellow sports scientists on various aspects of optimal performance such as more suitable training programs and techniques. Knowledge of the results may be used to improve rowing performances by optimizing the athlete's use of resources and delaying the onset of fatigue during a race. Furthermore, this information could be relevant not only to the rowing community, but also to other continuous sports. With this knowledge, it may be possible to consistently improve athletic performances in several sports.