

# Northumbria Research Link

Citation: Ansley, Les, Lambert, Mike I., Scharbort, Elske, St Clair Gibson, Alan and Noakes, Timothy (2004) Regulation of pacing strategies during successive 4-km time trials. *Medicine & Science in Sports & Exercise*, 36 (10). pp. 1819-1825. ISSN 0195-9131

Published by: Lippincott Williams & Wilkins

URL: <http://dx.doi.org/10.1249/01.MSS.0000142409.70181....>  
<<http://dx.doi.org/10.1249/01.MSS.0000142409.70181.9D>>

This version was downloaded from Northumbria Research Link:  
<https://nrl.northumbria.ac.uk/id/eprint/2140/>

Northumbria University has developed Northumbria Research Link (NRL) to enable users to access the University's research output. Copyright © and moral rights for items on NRL are retained by the individual author(s) and/or other copyright owners. Single copies of full items can be reproduced, displayed or performed, and given to third parties in any format or medium for personal research or study, educational, or not-for-profit purposes without prior permission or charge, provided the authors, title and full bibliographic details are given, as well as a hyperlink and/or URL to the original metadata page. The content must not be changed in any way. Full items must not be sold commercially in any format or medium without formal permission of the copyright holder. The full policy is available online: <http://nrl.northumbria.ac.uk/policies.html>

This document may differ from the final, published version of the research and has been made available online in accordance with publisher policies. To read and/or cite from the published version of the research, please visit the publisher's website (a subscription may be required.)



**Northumbria  
University**  
NEWCASTLE



**UniversityLibrary**

**Title:** Neural regulation of pacing strategies during successive 4 km time trials

**Authors:** Les Ansley, Elske Scharbort, Alan St Clair Gibson, Mike I Lambert,  
Timothy D Noakes

MRC/UCT Research Unit for Exercise Science and Sports Medicine and Discovery  
Health Chair of Exercise Science and Sports Medicine, Department of Human  
Biology, University of Cape Town and Sports Science Institute of South Africa, South  
Africa.

**Running title:** Neural regulation of pacing strategies

**Corresponding author:**

Les Nobbs

Sports Science Institute of South Africa

PO Box 115, Newlands

Cape Town, 7725

South Africa

**Fax:** +27 21 686 7330

**Phone:** +27 21 650 4559

**Email:** lnobbs@sports.uct.ac.za

## ABSTRACT

**Purpose:** Athletes adopt a pacing strategy to delay fatigue and optimise athletic performance. However, many current theories of the regulation of muscle function during exercise do not adequately explain all observed features of such pacing strategies. We studied power output, oxygen consumption and muscle recruitment strategies during successive 4km cycling time trials to determine whether alterations in muscle recruitment by the central nervous system could explain the observed pacing strategies. **Methods:** Seven, highly trained cyclists performed three consecutive 4 km time trial intervals, each separated by 17 minutes. Subjects were instructed to perform each trial in the fastest time possible, but were given no feedback other than distance covered. Integrated electromyographic (iEMG) readings were measured at peak power output and for 90 s before the end of each trial. **Results:** Subjects reach a  $VO_2$ max in each interval. Time taken to complete the first and third intervals was similar. Peak power output was highest in the first interval but average power output, oxygen consumption, heart rate and post-exercise plasma lactate concentrations were not different between intervals. Power output and iEMG activity rose similarly during the final 60 s in all intervals but were not different between trials. **Conclusion:** The similar pacing strategies in successive intervals and the parallel increase in iEMG and power output towards the end of each interval suggests that these pacing strategies could not have been controlled by peripheral mechanisms. Rather, these findings are compatible with the action of a centrally-regulated anticipatory mechanism that alters the number of motor units that are recruited and de-recruited during exercise based upon peripheral feedback or anticipatory feed-forward.

Key words: Fatigue, central, peripheral, lactate, electromyography, muscle recruitment

## INTRODUCTION

**Paragraph number 1** The capacity to produce and maintain power output decreases rapidly during bouts of high intensity exercise (2,30). Therefore, in order to sustain a high power output in events lasting longer than several seconds, athletes must adopt a pacing strategy to delay fatigue and optimise performance. The typical pacing strategy comprises several stages: a short initial period of high power output followed by a sharp reduction to a power output, which is usually maintained until the final period of the exercise bout when power output may again increase (8).

**Paragraph number 2** It is usually believed that these changes in power output are determined by the development of a critically low muscle pH that impairs skeletal muscle contractile function (16,17), so-called ‘peripheral fatigue’. However, this peripheral model of fatigue does not satisfactorily explain all the known features of exercise fatigue (13,19,20,28), most notably the phenomenon of the “lactate paradox” of high altitude. This paradox occurs during peak exercise at extreme altitude and in which exercise terminates at low blood lactate concentrations, often no higher than resting concentrations (10,26).

**Paragraph number 3** Foster *et al* (5) have proposed the alternate theory in which the pacing strategy is under central neural control. Indeed this theory that the central nervous system regulates athletic performance was first proposed over a century ago (29). But, the complexity of quantifying any central neural regulation of exercise performance has influenced researchers to focus on the peripheral, skeletal muscular determinants of fatigue and exercise performance with, until recently, less attention on any central component, (6,13,20,25).

**Paragraph number 4** Interval training, in which athletes work repeatedly at near maximum effort with short periods of recovery between exercise bouts, represents one

of the most intense forms of physical activity. Since the activity is repetitive, athletes must adopt a pacing strategy within the first interval to ensure that they can produce repetitive intervals with similar total work output. In order to determine any contribution of the central nervous system to the regulation of this pacing strategy, we measured the work output and associated changes in physiological, integrated electromyographic (iEMG) and metabolic parameters in consecutive 4 km cycling intervals performed by highly trained cyclists in the laboratory. We theorised that central regulation would produce pacing strategies that were more similar across intervals and in which iEMG activity would follow the changes in power output. In contrast, peripheral regulation would produce pacing strategies that were less similar across intervals; in which changes in power output would mirror changes in blood lactate concentrations, and in which power output would steadily decline towards the latter stages of the exercise bout despite a progressive increase in iEMG activity. In addition, a high proportion of all available motor units would be recruited during each interval.

## **METHODS**

### *Subjects*

**Paragraph number 5** Seven highly trained, competitive male cyclists were selected to participate in this study. These subjects were chosen because they were well trained, accustomed to high intensity exercise and familiar with laboratory testing since they frequently participated in previous trials in this laboratory. At the time of investigation, they were cycling 400-800 km.wk<sup>-1</sup>. The study was conducted with the approval of the Ethics in Human Research Committee of the Faculty of Health Sciences at the University of Cape Town; prior to the trial all subjects were informed of the nature of the investigation, after which they gave written informed consent. Subject characteristics are described in Table 1.

### *Kingcycle ergometry system*

**Paragraph number 6** Each subject completed two trials in the laboratory. On the first occasion maximal power output and maximal oxygen consumption (VO<sub>2</sub>max) was measured. On the second occasion seven days later, subjects performed three consecutive, 4-km time-trials (TT) with a 17-minute rest between each. All tests were conducted on a Kingcycle ergometry system (Kingcycle Ltd, High Wycombe, U.K.) that allowed each cyclist to ride his own bicycle in the laboratory. Previous studies have shown that performance tests conducted on these ergometers have good reliability when compared to standard laboratory ergometers (14,21,22).

### *VO<sub>2</sub>max test*

**Paragraph number 7** On their first visit to the laboratory, cyclists completed the maximal incremental exercise test (MIE) to exhaustion during which oxygen consumption (VO<sub>2</sub>), power output and heart rate (HR) were recorded for the duration

of the test. After a 10-15 min warm-up at a self-selected intensity, the test commenced at a workload of 200 W. Thereafter the workload increased by 20 W.min<sup>-1</sup> until the subject could no longer maintain the required power output. The same investigators gave verbal encouragement throughout all maximal tests. Peak power output (PPO) was defined as the highest power output attained during the VO<sub>2</sub>max test. Subjects were requested to remain in a seated position for the duration of the MIE and the time trials.

**Paragraph number 8** For the measurement of VO<sub>2</sub> during the MIE and the 4-km time trials, subjects wore a mask covering the nose and mouth; the expired air passed through an on-line computer system attached to an automated gas analyzer (Oxycon model Alpha, Mijnhardt, The Netherlands). Before each test, the gas analyzer was calibrated with a Hans Rudolph 5530, 3-L syringe and a span gas mixture. Analyzer outputs were processed by a computer that calculated VO<sub>2</sub> and carbon dioxide production using conventional equations (12). VO<sub>2</sub>max was defined as the highest VO<sub>2</sub> measured during the MIE.

**Paragraph number 9** Heart rate (HR) was recorded every 10 seconds using a Polar Accurex Plus heart rate monitor (Polar Electro.Kempele, Finland). The data were downloaded via an interface into hrm files after completion of the trial. Maximum HR was taken as the highest HR recorded at any stage during the test.

### *Time-trials*

**Paragraph number 10** Within seven days of the MIE each subject returned to the laboratory at the same time of day to perform three consecutive 4-km time trials. Successive time trials were separated by approximately 17 min. Subjects were requested to perform the same type of training for the duration of the trial and to refrain from heavy physical exercise for 24 hours before the TT testing.

**Paragraph number 11** After a self-selected 10-15 min warm-up, the first TT started after a 2 min countdown during which the cyclists maintained a cycling speed of 35-40 km.hr<sup>-1</sup>. This speed had to remain constant until the start of the time trial. After each time trial, the subjects rested for 10 min after which they performed a 5 min warm-up before commencing the 2 min countdown. Subjects were told to perform each time trial in the fastest time possible but in the knowledge that three trials were to be performed. The elapsed distance was the only feedback given to the subjects during the time trials. They were not informed of completion times until after the final time trial. Power output, HR and VO<sub>2</sub> measurements were recorded continuously during the trials. A fan was positioned in front of the subjects during their time trials and during the 10 min rest interval, subjects were allowed to drink water *ad libitum*.

**Paragraph number 12** Before the MIE and time trials a 20-gauge Jelco cannula (Critikon, Halfway House, South Africa) was inserted into an antecubital forearm vein for blood sampling. Exactly 3 min (15) after both the VO<sub>2</sub>max test and each TT, blood was drawn and placed into tubes containing potassium oxalate and sodium fluoride for the measurement of plasma lactate concentration. The blood samples were kept on ice until centrifuged at 3 000 x g for 10 min at 4°C and the plasma stored at -20°C until later analysis. Plasma lactate concentrations were determined by spectrophotometric (Beckman Spectrophotometer -M35) enzymatic assays using the conventional assay (Lactate PAP, bioMérieux Kit, Marcey l'Etoile, France).

#### *Isometric testing of skeletal muscle function*

**Paragraph number 13** Immediately before the MIE test and time trials, each subject's peak isokinetic force was measured on a Kin-Com isokinetic dynamometer (Chattanooga Group Inc., USA). The peak force produced by the quadriceps muscles

was tested isometrically at an angle of 60°, with full knee extension being the 0° reference.

**Paragraph number 14** Subjects performed four 50%, two 70%, one 90% and one 100% familiarization trial of 5 seconds each as a warm-up. After this warm-up each subject performed four 5 second maximal voluntary contractions (MVC) with a 5 second rest between trials. The EMG activity coinciding with the peak torque of the second effort was used to normalise the EMG values recorded during the time trials (11). The subjects were verbally encouraged during the tests to exert a maximal effort.

#### *Electromyographic activity*

**Paragraph number 15** Muscle recruitment was assessed during the isometric test, as well as during the TTs by measuring EMG activity of the rectus femoris muscle. Electrodes (Thought Technology Triode™ MIEPO1-00, Montreal, Canada) with a bandwidth of 20-500 Hz and sensitivity of < 0.08 µV were attached to the subject's lower limb prior to the start of all testing. The skin overlying the rectus femoris muscle was firstly shaven, after which the outer layer of epidermal cells was abraded with sandpaper and the dirt removed from the skin using an alcohol swab. The triode electrode was placed in the middle of the subject's rectus femoris muscle, secured with self-adherent wrap (Coban 3M, 1582, St. Paul, MN, USA), and linked via a fibre-optic cable to a Flexcomp/DSP EMG apparatus (Thought Technology Montreal, Canada) and host computer. EMG activity was recorded for 5 s during the isokinetic test. During the time trials EMG activity was recorded from 60 to 80 s to coincide with the maximal power output and in 10 s intervals between 200 s to exhaustion during the TTs.

**Paragraph number 16** A toggle switch was activated at the beginning of each test to mark the start point of the test procedure, with each activity being sampled at a 1984 Hz capture rate. A 50 Hz line filter was applied to the raw EMG data to prevent any external interference from electrical sources. The EMG signals from the electrode were band-pass filtered (20-500 Hz) and amplified using standard differential amplifiers (Thought Technology, Montreal, Canada; common mode rejection ratio > 130 dB at 1 kHz, input impedance = 1 million MegOhms, adjustable gain up to 1600). The raw EMG signals were subsequently full wave rectified, movement artefact removed using a high-pass second order Butterworth filter with a cut off frequency of 15 Hz and then smoothed with a low-pass second-order Butterworth filter with a cut-off frequency of 5 Hz. This was performed using MATLAB™ gait analysis software. The integrated EMG data (IEMG) were used for subsequent analyses.

#### *EMG data analysis*

**Paragraph number 17** All EMG data were normalised by dividing the value at each time point during the cycle trial by the EMG value obtained during the MVC performed before the start of the performance test. Further normalisation of the EMG recorded over the final 90 s of exercise was performed against the mean EMG recorded over the period 60 to 80 s. These data are expressed as a percentage of the mean EMG during the peak power output.

#### *Statistical analysis*

**Paragraph number 18** Data for HR, VO<sub>2</sub> and power output were averaged over 30 s from 0 to 300 s. EMG data were compared every 10 s from 200 s to fatigue. An analysis of variance was used to assess differences between and within the trials. Once main effects were identified individual differences between the means were

located using Tukey's HSD *post hoc* procedure. Significance was accepted at  $P < 0.05$ . All data are expressed as mean  $\pm$  standard deviation (S.D.).

## RESULTS

### *Maximum values of VO<sub>2</sub>, HR and plasma lactate concentrations*

**Paragraph number 19** The VO<sub>2</sub>max attained during maximal incremental exercise (MIE) was similar to the highest VO<sub>2</sub> measured during each of the 4 km time trials (MIE 71.4 ± 2.3; TT1 70.2 ± 4.1; TT2 70.7 ± 5.3; TT3 69.8 ± 4.2 ml•kg<sup>-1</sup>•min<sup>-1</sup>) (Figure 1a). Maximum heart rate reached during MIE (193 ± 7 beats•min<sup>-1</sup>) was significantly higher than during TT1 (186 ± 6 beats•min<sup>-1</sup>) and TT2 (187 ± 6 beats•min<sup>-1</sup>) but was not different from TT3 (189 ± 7 beats•min<sup>-1</sup>) (Figure 1b). Post exercise plasma lactate concentrations were similar for all the time trials (TT1 12.7 ± 2.0; TT2 12.8 ± 2.5; TT3 14.0 ± 0.4 mmol•L<sup>-1</sup>) and the concentrations were not different from those observed after the incremental maximal exercise test (14.5 ± 0.8 mmol•L<sup>-1</sup>) (Figure 1c).

### *Peak and average power outputs*

**Paragraph number 20** The peak power outputs, reached at 60 seconds (Figure 2a), were significantly higher in the first interval (TT1 556 ± 51 W; TT2 504 ± 44 W; TT3 506 ± 41 W; p < 0.05). However, the average power outputs for each interval were not different (TT1 447 ± 30 W; TT2 425 ± 33 W; TT3 434 ± 33 W) (Figure 2b). Nor was the time taken to complete the first interval (284 ± 8 s) and the average velocity (51 ± 1 km•h<sup>-1</sup>) significantly faster than for the third interval (287 ± 9 s and 50 ± 2 km•h<sup>-1</sup>) although it was faster than the second interval (290 ± 9 s and 50 ± 2 km•h<sup>-1</sup>). Completion times were not different between the second and the third interval (Figure 2c). In all trials power output declined significantly from 60 s (Figure 3a) after which it reached a plateau. However, in the second and third intervals the

initial increase in power output during the first 30 seconds of exercise was less than in the first trial. Furthermore, power output rose over the final minute of exercise in the second and third trial (Figure 3a and 4a).

#### *VO<sub>2</sub> and heart rate*

**Paragraph number 21** The rate of oxygen consumption was similar for all intervals after the first minute (Figure 3b) and did not increase significantly over the duration of the interval. Likewise heart rate did not vary between intervals, but it did increase significantly from the 30<sup>th</sup> to the 60<sup>th</sup> second ( $p < 0.05$ ), after which it did not increase significantly (Figure 3c).

#### *iEMG*

**Paragraph number 22** The iEMG recorded during the period of maximal power output during the time trials was less than 25% of iEMG activity during the MVC (TT1  $24.2 \pm 3.5$ ; TT1  $21.1 \pm 4.2\%$ ; TT3  $23.7 \pm 3.8\%$ ). There was no difference in iEMG between intervals (Figure 4c). However in all trials iEMG tracked changes in power with the highest values being measured at 60 and 300 seconds when power output was also highest (Figure 4a). Furthermore, iEMG did not increase sequentially across successive intervals and was not higher at 300 seconds than at 60 seconds in any interval.

## **DISCUSSION**

*Paragraph number 23* The first important finding of this study was that subjects adopted a similar pacing strategy during each of the three consecutive 4 km intervals (time trials) (Figures 2 and 3) even though they received no external feedback regarding their cycling speeds, power output, duration or heart rates. Distance elapsed was the only external feedback provided.

*Paragraph number 24* The pacing strategy adopted was such that the peak power output was reached after 60 seconds where after there was a steep decline in power output, more pronounced in the first interval, so that at 120 seconds, power outputs were the same as in the first 30 seconds. Thereafter power outputs fell only slightly to 240 seconds where after they rose again reaching outputs at 300 seconds that approached those reached after 60 seconds in the second and third trials, but which were lower than the peak values achieved in Trial 1 (Figure 3).

*Paragraph number 25* Indeed there was clear evidence of a learning effect since power outputs were higher at 30, 60 and 90 seconds in the first interval, than in the second and third intervals. Thereafter power outputs were essentially the same in all three trials. Thus, the pacing strategies were essentially reproducible throughout the second and third trials. This suggest that subjects may have altered their pacing strategies in the second and third intervals, on the basis of what they learned in the first trial. As a result, performance in the second and third trials was identical. Importantly there was no evidence for the development of a progressive fatigue since power output progressively increased over the last 60 seconds of the final two trials (Figure 4a). Presumably if the faster performance in the first interval had been attempted in the second and third intervals, there might have been the development of a progressive fatigue with a progressive reduction in power output. Rather the finding

that the average power output was the same in all three intervals suggests that these cyclists adopted an ideal pacing strategy for the optimum completion of the total work bout.

**Paragraph number 26** Indeed the second important finding of the study was that the power output increased during the final 60 seconds of each time trial, a phenomenon that has also been noted in a longer duration time trials lasting 60 minutes which included six one minute sprint intervals (13). These authors also found an increase in iEMG activity and power output to near initial values during the final sprint, following progressive declines in the intervening sprints. If the power output during the time trials had been regulated by peripheral fatigue mechanisms, an increased central neural recruitment of additional motor units would be expected to occur (4,7,9,27) in an attempt to compensate for the reduced power output from the fatiguing motor units. This would be shown as a progressive increase in iEMG activity and an irreversibly falling power output. But this did not occur in this study since power output and iEMG changed in parallel during the final 90 seconds of exercise (Figure 4). More significantly, iEMG were not progressively higher in successive time trials as would be expected if peripheral fatigue caused a progressive impairment of the power output of individual motor units, requiring the recruitment of progressively more motor units to compensate for the reduced power output of the fatiguing units.

**Paragraph number 27** In addition less than a quarter of the available motor units in the studied muscles were recruited during the time trials, a finding that is consistent with previous observations (13,24,25) but is incompatible with the peripheral fatigue model. For the peripheral model predicts that there must be near total motor unit

recruitment at exhaustion so that fatigue results from peripheral metabolite-induced modulation of the contractile activity of all the recruited motor units.

**Paragraph number 28** Indeed it is difficult to imagine how peripheral metabolite changes in the skeletal muscles could explain the subtle changes in pacing during the three different intervals. First it seems unlikely that identical metabolite concentrations in the active muscles would explain the identical pacing strategies in the second and third trials. For example, it would seem unlikely that full metabolic recovery could have occurred within 17 minutes after the subjects had already completed 10 minutes of exercise at, or close to,  $VO_2\text{max}$ . That the plasma lactate concentrations were similar at the end of each interval was unexpected (Figure 1c) and might indicate that a component of the pacing strategy is to complete exercise with specific terminal plasma (and muscle) lactate concentrations (5).

**Paragraph number 29** Second, since each interval was performed at close to  $VO_2\text{max}$ , there must have been a progressive increase in plasma and muscle lactate concentrations during each interval. If a progressive rise in the muscle lactate concentrations impairs exercise performance, then the athletes should have shown a pacing strategy that produced a maximum power output in the first seconds of exercise with a progressive fall thereafter with the lowest power outputs in the last seconds of each interval, most especially in the last interval. As already described, this did not occur (Figure 3a).

**Paragraph number 30** Rather, the most plausible explanation is that the pacing strategy results from changes in the number of motor units that are alternatively recruited and de-recruited during different stages of exercise. This would occur if a subconscious “controller” determines the overall pacing strategy during exercise by matching the rate of energy expenditure and the current energy reserves with the

predicted energy cost of the exercise (28), but within the physiological capacity of the individual. Ulmer (28) was perhaps the first to propose that the optimal metabolic rate during exercise is maintained by the action of a programmer that takes cognisance of the duration or distance, or both, of the planned effort and calculates the optimum pacing strategy on the basis of previous experience. This pre-emptive mechanism would not only pattern a single time trial, but would be responsible for the pacing strategy adopted in the consecutive trials that comprise the entire exercise bout.

**Paragraph number 31** The third noteworthy finding of the study was that despite the different power output profiles (Figure 3a) between the first and third intervals, the average velocities were quite similar as were the finishing times. Foster et al (5) have postulated the presence of a central mechanism governing muscle recruitment that is influenced by afferent sensory feedback of pH levels in the exercising muscle. They suggest that athletes learn to ‘sense’ the intramuscular pH and adapt their workload on the basis of this feedback so that a critically low muscle pH is never approached.

**Paragraph number 32** The proposed mechanistic functioning of this feedback system involves the detection of pH changes by nociceptors or chemoreceptors that transmit an inhibitory signal to the central nervous system, which is perceived as a “sensation”. As a result, the motor cortex decreases efferent neural drive directed to those motor units in the exercising muscles from which the afferent signals were received. The degree of reduction in efferent activity is proportional to the magnitude of afferent feedback activity. Therefore, a centrally directed pacing strategy is adopted, which is fine-tuned in the motor cortex in response to this peripheral feedback.

**Paragraph number 33** The lag phase inherent in this model would explain the power output profile observed in the first time trial in which there was an initial very rapid increase in power output with a disproportionately large percentage of the total work

performed in the initial stages of exercise. However, in the third time trial the initial spike in power output is dampened, which is consistent with the peripheral monitoring theory proposed by these workers (5) since it is unlikely that complete intramuscular biochemical homeostasis, would have been restored during the rest periods between trials (3). Alternatively this modification could have resulted from changes in feedforward control as a result of information acquired during the first interval. Although muscle pH and lactate may act as the monitored chemicals, neither would appear to be limiting factors according to those studies that have dissociated blood lactate concentrations and pH from changes in performance (23) by altering blood lactate and pH during stochastic 40 km time trials without equivalent changes in performance. Similarly Medbø and Sejersted (15) concluded that blood lactate concentrations do not limit physical performance during exhaustive exercise.

***Paragraph number 34*** In summary, this study shows that the pacing strategies adopted by athletes during 3 successive 4km cycling time trials that each elicited a  $\text{VO}_2$  max response were remarkably similar, despite the absence of external feedback to the subjects other than distance covered. Importantly, power outputs and iEMG activities rose progressively and similarly from 210 – 300 seconds of exercise in all three intervals. The finding that the pacing strategies were remarkable similar, that iEMG activities were not progressively greater in successive intervals and that only about 25% of available motor units were recruited at the point of peak power output, suggests the presence of a centrally-determined, anticipatory regulation of the adopted pacing strategies. Such regulation would result from a centrally-determined recruitment and de-recruitment of more or less motor units during the exercise bout.

The extent to which the pacing strategy is influenced by feedback from metabolic events in the active muscles could not be determined by this study.

## **ACKNOWLEDGEMENTS**

Funding for this study was provided by the Beatrix Waddell Scholarship Fund, the Lowenstein Scholarship Trust and the Harry Crossley Staff Research Fund, all of the University of Cape Town; the National Research Foundation and the Medical Research Council of South Africa; and Discovery Health Pty Ltd.

## REFERENCES

1. Andersen, K. L. The cardiovascular system in exercise. In: *Exercise Physiology*. Falls, H. B. (Eds.) New York: Academic Press, 1968, pp. 79-128.
2. Bar-Or, O. The Wingate anaerobic test. An update on methodology, reliability and validity. *Sports Med.* 4:381-394, 1987.
3. Busse, M. W. and N. Maassen. Effect of consecutive exercise bouts on plasma potassium concentration during exercise and recovery. *Med. Sci. Sports Exerc.* 21:489-493, 1989.
4. Crenshaw, A. G., S. Karlsson, B. Gerdle, and J. Friden. Differential responses in intramuscular pressure and EMG fatigue indicators during low- vs. high-level isometric contractions to fatigue. *Acta Physiol. Scand.* 160:353-361, 1997.
5. Foster, C., M. Schrage, A. C. Snyder, and N. N. Thompson. Pacing strategy and athletic performance. *Sports Med.* 17:77-85, 1994.
6. Gandevia, S. C. Spinal and supraspinal factors in human muscle fatigue. *Physiol. Rev.* 81:1725-1789, 2001.
7. Gerdle, B., S. Karlsson, A. G. Crenshaw, and J. Friden. The relationships between EMG and muscle morphology throughout sustained static knee extension at two submaximal force levels. *Acta Physiol. Scand.* 160:341-351, 1997.
8. Hagerman, F. C. Applied physiology of rowing. *Sports Med.* 1:303-326, 1984.
9. Hakkinen, K. and P. V. Komi. Electromyographic and mechanical characteristics of human skeletal muscle during fatigue under voluntary and reflex conditions. *Electroencephalogr. Clin. Neurophysiol.* 55:436-444, 1983.

10. Hochachka, P. W., C. L. Beatty, Y. Burelle, M. E. Trump, D. C. McKenzie, and G. O. Matheson. The lactate paradox in human high-altitude physiological performance. *News Physiol. Sci.* 17:122-126, 2002.
11. Hunter, A. M., A. St Clair Gibson, M. I. Lambert, and T. D. Noakes. Electromyographic (EMG) normalization method for cycle fatigue protocols. *Med. Sci. Sports Exerc.* 34:857-861, 2002.
12. Jones, N. L. Clinical exercise testing. Philadelphia: W.B.Saunders Co, 1987, pp. 292-300.
13. Kay, D., F. E. Marino, J. Cannon, A. St Clair Gibson, M. I. Lambert, and T. D. Noakes. Evidence for neuromuscular fatigue during high-intensity cycling in warm, humid conditions. *Eur. J. Appl. Physiol.* 84:115-121, 2001.
14. Lindsay, F. H., J. A. Hawley, K. H. Myburgh, H. H. Schomer, T. D. Noakes, and S. C. Dennis. Improved athletic performance in highly trained cyclists after interval training. *Med. Sci. Sports Exerc.* 28:1427-1434, 1996.
15. Medbø, J. I. and O. M. Sejersted. Acid-base and electrolyte balance after exhausting exercise in endurance-trained and sprint-trained subjects. *Acta Physiol. Scand.* 125:97-109, 1985.
16. Metzger, J. M. and R. H. Fitts. Role of intracellular pH in muscle fatigue. *J. Appl. Physiol.* 62:1392-1397, 1987.
17. Nakamaru, Y. and A. Schwartz. The influence of hydrogen ion concentration on calcium binding and release by skeletal muscle sarcoplasmic reticulum. *J. Gen. Physiol.* 59:22-32, 1972.
18. Noakes, T. D. Implications of exercise testing for prediction of athletic performance: a contemporary perspective. *Med. Sci. Sports Exerc.* 20:319-330, 1988.

19. Noakes, T. D. 1996 J.B. Wolfe Memorial Lecture. Challenging beliefs: ex Africa semper aliquid novi. *Med. Sci. Sports Exerc.* 29:571-590, 1997.
20. Noakes, T. D. Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scand. J. Med. Sci. Sports.* 10:123-145, 2000.
21. Palmer, G. S., S. C. Dennis, T. D. Noakes, and J. A. Hawley. Assessment of the reproducibility of performance testing on an air-braked cycle ergometer. *Int. J. Sports Med.* 17:293-298, 1996.
22. Schabort, E. J., J. A. Hawley, W. G. Hopkins, I. Mujika, and T. D. Noakes. A new reliable laboratory test of endurance performance for road cyclists. *Med. Sci. Sports Exerc.* 30:1744-1750, 1998.
23. Schabort, E. J., G. Wilson, and T. D. Noakes. Dose-related elevations in venous pH with citrate ingestion do not alter 40-km cycling time-trial performance. *Eur. J. Appl. Physiol.* 83:320-327, 2000.
24. Sjogaard, G. Force-velocity curve for bicycle work. In: *Biomechanics VI*. Asmussen, E. and Jorgensen, K. (Eds.) Baltimore: University Park Press, 1978, pp. 33-39.
25. St Clair Gibson, A., E. J. Schabort, and T. D. Noakes. Reduced neuromuscular activity and force generation during prolonged cycling. *Am. J. Physiol.* 281:R187-R196, 2001.
26. Sutton, J. R., J. T. Reeves, B.M. Groves, P. D. Wagner, J. K. Alexander, H. N. Hultgren, A. Cymerman, and C. S. Houston. Oxygen transport and cardiovascular function at extreme altitude: lessons from Operation Everest II. *Int. J. Sports Med.* 13 Suppl 1:S13-S18, 1992.

27. Taylor, A. D., R. Bronks, P. Smith, and B. Humphries. Myoelectric evidence of peripheral muscle fatigue during exercise in severe hypoxia: some references to m. vastus lateralis myosin heavy chain composition. *Eur. J. Appl. Physiol.* 75:151-159, 1997.
28. Ulmer, H. V. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia.* 52:416-420, 1996.
29. Waller, A. D. The sense of effort: an objective study. *Brain.* 14:179-249, 1896.
30. Withers, R. T., W. M. Sherman, D. G. Clark, P. C. Esselbach, S. R. Nolan, M. H. Mackay, and M. Brinkman. Muscle metabolism during 30, 60 and 90 s of maximal cycling on an air- braked ergometer [published errata appear in *Eur. J. Appl. Physiol.* 64:387, 1992 and 1992;64:485, 1992]. *Eur. J. Appl. Physiol.* 63:354-362, 1991.

## LEGEND TO FIGURES

**Figure 1** Mean maximum values of  $\text{VO}_2$ , heart rate and lactate concentrations attained during the maximal incremental exercise and three consecutive 4 km time trial intervals.

**Figure 2** Peak power output, mean power output and the performance time for the three consecutive 4 km time trial intervals.

$\lambda$  1<sup>st</sup> interval is significantly different from the 2<sup>nd</sup> and 3<sup>rd</sup> intervals ( $p < 0.05$ )

\* 2<sup>nd</sup> interval is significantly different from the 1<sup>st</sup> interval ( $p < 0.05$ )

**Figure 3** Power output,  $\text{VO}_2$  and heart rate for the duration of the consecutive 4 km time trial intervals.

<sup>a</sup> 1<sup>st</sup> interval is significantly different from the 2<sup>nd</sup> and 3<sup>rd</sup> intervals ( $p < 0.05$ )

<sup>b</sup> Significantly different from 60 s in the 1<sup>st</sup> interval ( $p < 0.05$ )

<sup>c</sup> Significantly different from 60 s in the 1<sup>st</sup> and 2<sup>nd</sup> intervals ( $p < 0.05$ )

<sup>d</sup> Significantly different from 60 s for all intervals ( $p < 0.05$ )

<sup>e</sup> Significantly different from the 2<sup>nd</sup> interval ( $p < 0.05$ )

**Figure 4** Absolute power, relative power and relative iEMG at 60, 210, 240, 270 and 300 s in each of the three consecutive 4 km time trials.

<sup>a</sup> 1<sup>st</sup> interval is significantly different from the 2<sup>nd</sup> and 3<sup>rd</sup> intervals ( $p < 0.05$ )

<sup>b</sup> Significantly different from 60 s in the 1<sup>st</sup> interval ( $p < 0.05$ )

<sup>d</sup> Significantly different from 60 s for all intervals ( $p < 0.05$ )

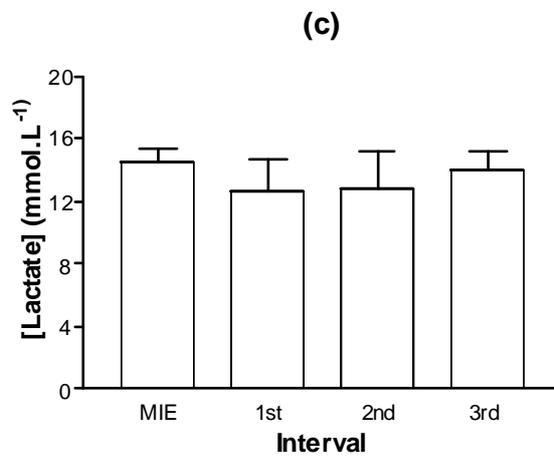
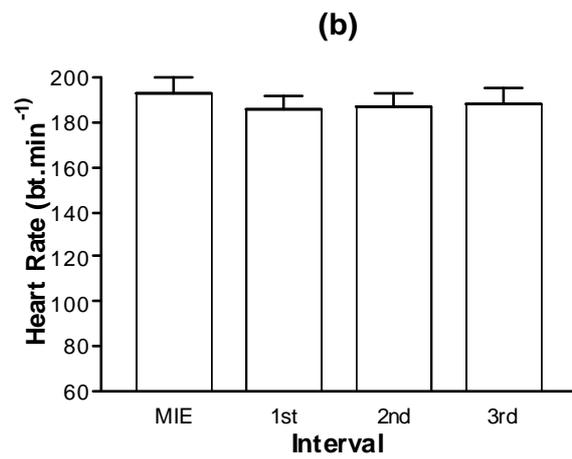
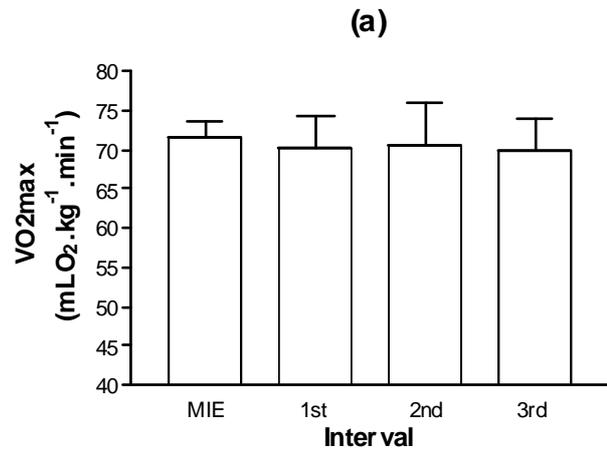
**Table 1** Subject characteristics and results from the maximal incremental exercise test (n=7)

Age (yr)	23.7 ± 4.4
Height (m)	1.81 ± 0.09
Mass (kg)	73.7 ± 9.2
VO <sub>2</sub> max (mL•kg <sup>-1</sup> •min <sup>-1</sup> )	71.4 ± 2.3
HRmax (beats•min <sup>-1</sup> )	193 ± 7
PPO (W)	463.6 ± 32.4
P:W (W•kg <sup>-1</sup> )	6.3 ± 0.6
La <sub>peak</sub> (mmol•L <sup>-1</sup> )	14.5 ± 0.8

Values are expressed as mean ± SD

VO <sub>2</sub> max	peak oxygen uptake
HRmax	maximum heart rate
PPO –	peak power output
P:W –	power to weight ratio
La <sub>peak</sub> –	peak blood lactate

**Figure 1**



**Figure 2**

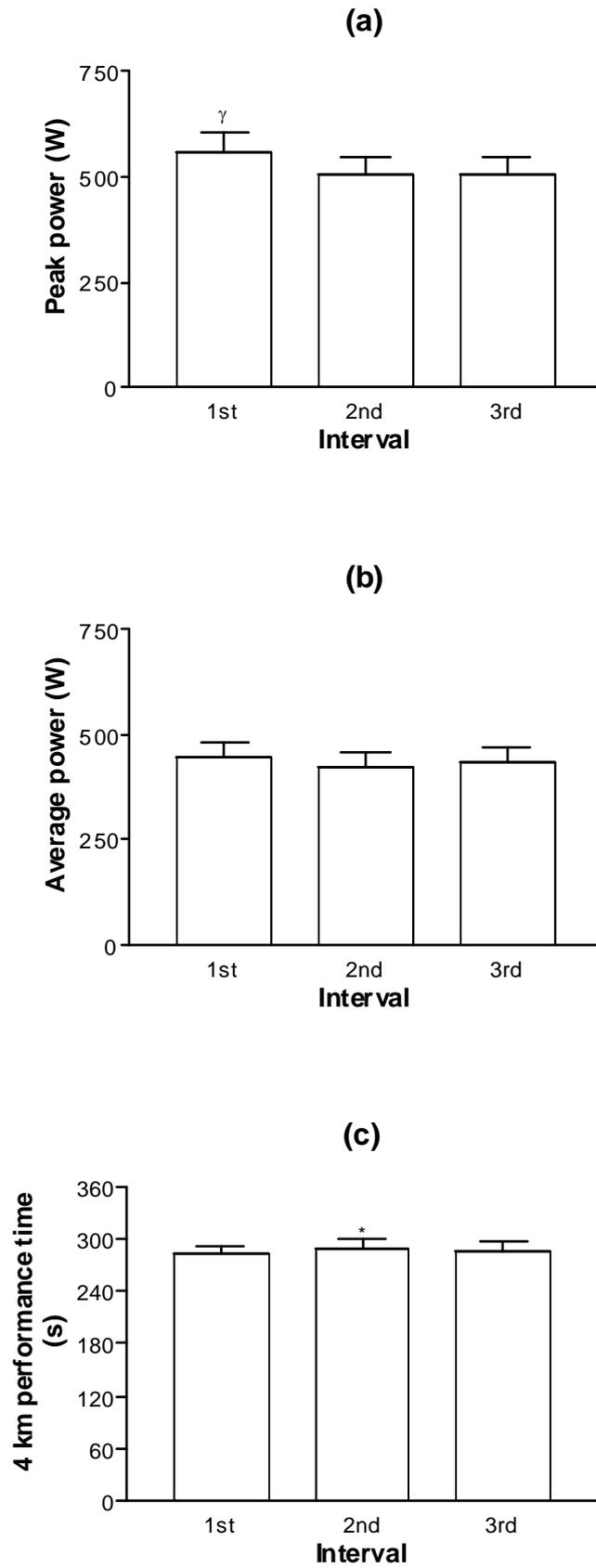


Figure 3

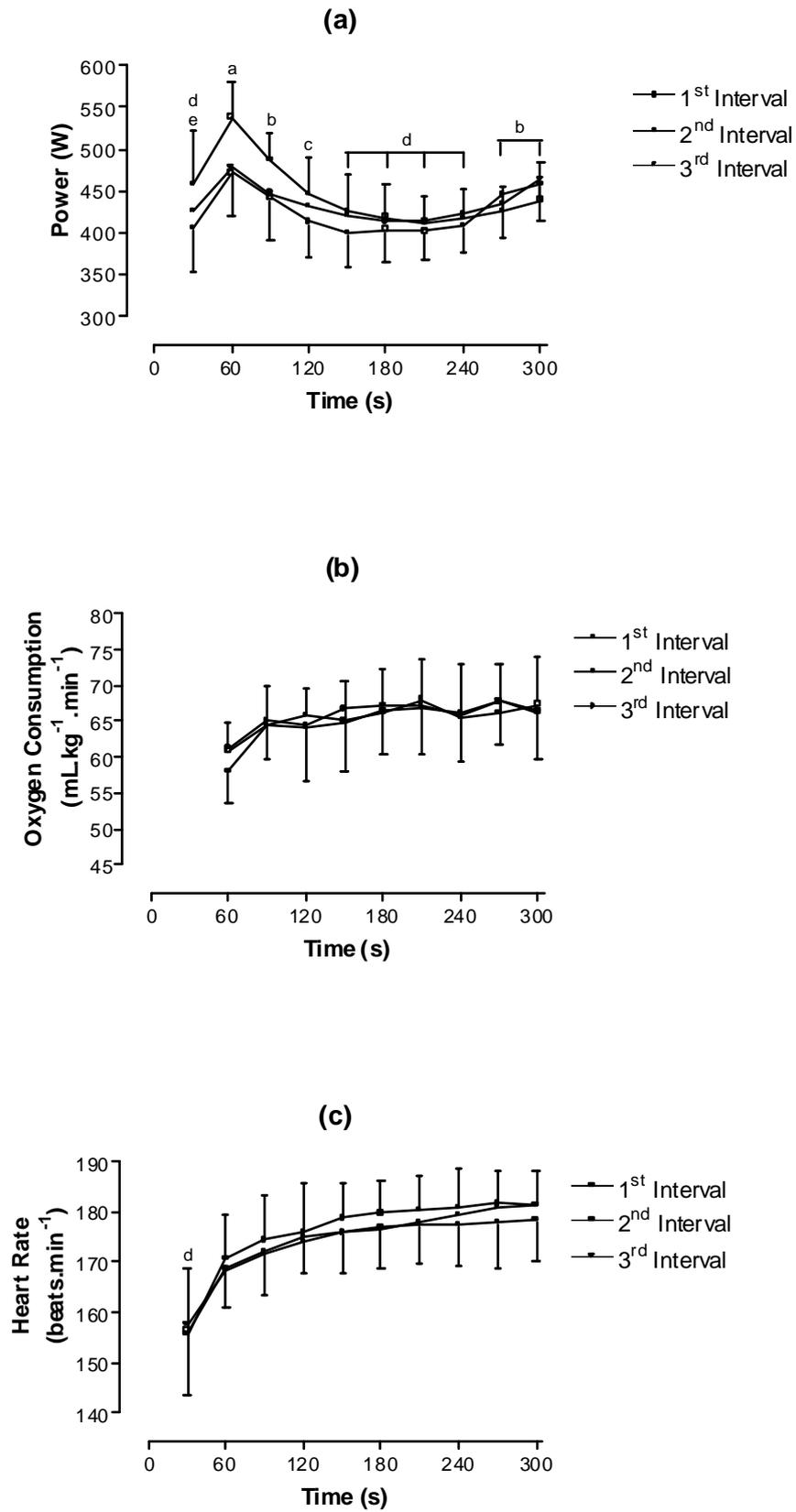


Figure 4

