THE IMPACT OF CORE TEMPERATURE CORRECTIONS ON EXERCISE-INDUCED HYPOXEMIA

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DECLARATION

I declare that this thesis contains no material which has been accepted for the award of any other degree or diploma in any University or tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person except where due reference has been made in the text.

I give consent to this copy of my thesis, when deposited in the University library, being available for loan and photocopying

Nicholas Jon Shipp Date

DEDICATION

This thesis is dedicated to my family and friends and, especially, to my future wife Belinda. Your support throughout its making has been invaluable and I look forward to our life ahead.

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PUBLICATIONS AND PRESENTATIONS

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Presentations-

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ABSTRACT

The primary purpose of this doctoral dissertation was to investigate the effect of body temperature responses at physiologically relevant sites during an incremental exercise test on the phenomenon of exercise-induced hypoxemia (EIH). This phenomenon has been considered as an important limitation to physical performance with a prevalence of ~50 % in trained male athletes, but described in both sexes, across the range of both age and physical fitness in more recent literature. Previously this phenomenon has been described as a decrement in both arterial oxygen partial pressure (PaO_2) and oxy-haemoglobin saturation (SaO_2 or SpO_2) with, particularly important for PaO_2, a lack of or inappropriate correction made for the change in body temperature during intense exercise.

The initial study of this thesis determined the thermal response within the body at physiologically relevant sites measured simultaneously during an incremental exercise test. The results demonstrated the inadequacy of rectal temperature as an indicator of the acute temperature changes occurring during an incremental exercise test due to its slow response rate and relative thermal inertia. Radial arterial blood and oesophageal temperatures were shown to behave almost identically during the exercise test, albeit with an offset of approximately 1.3ºC, and were considered much more appropriate and relevant indicators of thermal changes during exercise. As an extension of the initial work active muscle temperature (*vastus lateralis*) was measured during the

exercise test, demonstrating a significantly lower resting temperature than the oft-reported “core” temperatures (rectal and oesophageal) as well as a significantly greater increase in temperature in comparison to all other measurement sites. Overall, the results of this first study indicated that the physiologically relevant temperatures measured at the oesophageal and muscle sites differed markedly to the outdated rectal temperature measurement site and should be used as measures of thermal response when evaluating oxygen loading (oesophageal) or unloading (active muscle).

Utilising the definition of EIH as a decrease in PaO_2 of ≥ 10 mmHg, the effect of temperature correcting PaO_2 was evaluated in the second study. Arterial blood gases measured simultaneously to the temperature measurements during the incremental exercise test were adjusted for the temperature changes at each site (every 1ºC increase in temperature will increase a PaO_2 value by ~5 mmHg). Whilst uncorrected PaO_2 values indicated an almost 100% prevalence of EIH in this group, oesophageal temperature corrected PaO_2 values decreased this prevalence to ~50% while muscle temperature corrections resolved all cases of EIH and demonstrated an HYPEROXAEMIA (i.e. the reverse of the well-established phenomenon) in the majority of subjects. Further investigation of arterial oxygen content during the exercise test indicates that there is no disruption in the delivery of oxygen to the active muscles and therefore any performance decrement should be attributed to another mechanism.

Whilst the phenomenon of EIH is determined by the definition applied and the use of temperature corrections in the case of PaO_2, its reproducibility in a test-retest situation had not previously been determined. Utilising a subset of

previously tested subjects, the reproducibility of both temperature and PaO_2 were determined with results indicating that the blood gas response was highly reproducible, especially the minimum PaO_2 value noted during each exercise test. However, comparing a more statistically relevant definition of a change in PaO_2 of ± 2 standard deviations from the mean resting PaO_2 to the previous delimiter of 10 mmHg indicated a lesser reproducibility of the prevalence of EIH.

In summary, this thesis exposes the inadequacies of previous research into EIH with regard to the expected reproducibility of the phenomenon and the need to correctly adjust PaO_2 values for exercise-induce hyperthermia as well as demonstrating the difference in thermal responses to acute exercise in physiologically significant areas of the body. Furthermore, previously described correlations between the change in PaO_2 and $\dot{V}O_2$ max were not evident in the subjects tested within this thesis, nor was there any indication of a diffusion limitation based on reduced pulmonary capillary transit time (by association with $\dot{V}O_2$ max) or pulmonary oedema (rebuked by a rapid return of PaO_2 to above resting levels following exercise cessation).