Re-examination of the incidence of exercise-induced hypoxaemia in highly trained subjects

Dale D. Brown PhD, Ronald G. Knowlton* PhD, Parviz B. Sanjabit MD and Brad T. Szurgot* MS

Department of Health, Physical Education, Recreation and Dance, Illinois State University, Normal, Illinois;

*Department of Physical Education, Southern Illinois University, Carbondale, Illinois; †Herrin Hospital, Herrin, Illinois; and Carbondale Clinic and Southern Illinois University School of Medicine, Carbondale, Illinois, USA

The purpose of this study was to examine the occurrence of exercise-induced hypoxaemia (EIH) during maximal exercise in highly trained athletes. Eleven trained cyclists (mean(s.d.) age 23(3.5) years; mean(s.d.) $\dot{V}O_{2\max}$ 66.9(4.8) ml kg⁻¹ min⁻¹) performed a continuous, multistage (270 kpm min⁻¹) cycle ergometer test to exhaustion. Measurements of arterial oxygen-haemoglobin saturation (%HbO₂) were obtained simultaneously at rest, every 2 min during exercise, and at maximum exercise capacity from arterial blood sampling (%SaO₂) and ear oximetry (%SpO₂). Exercise induced hypoxaemia (%HbO₂ ≤ 91%) was present in 64% of the athletes examined when EIH was determined using pulse oximetry, whereas none of the subjects exhibited EIH when %HbO₂ was determined using arterial blood. At rest the values for %HbO₂ were similar with mean(s.d.) %SaO₂ being 97.3(0.6)% and mean(s.d.) %SpO₂ being 96.5(1.6)%. During exercise, statistically significant differences were found for %HbO₂ between arterial blood and ear oximetry at the 6-min, 8-min, and maximal exercise sampling times (repeated measures analysis of variance, $P < 0.05$). The results indicate that ear oximetry overestimates the incidence of EIH and underestimates the oxyhaemoglobin saturation in highly trained cyclists during exercise in comparison with those measurements made from arterial blood.

Keywords: Arterial blood, desaturation, exercise, hypoxaemia, trained athletes

It has been postulated that there are two physiological extremes that exist which can limit maximal exercise performance¹. In the untrained individual, exercise performance becomes limited due to the 'weak links' of the oxygen transport system and oxidative capacity of the muscular system^{1,2}. Whereas, in the highly trained individual, the rate limiting step becomes the anatomical and physiological constraints of the respiratory system resulting in 'exercise-induced hypoxaemia' (EIH)^{1,2}. Numerous investigations exist reporting arterial oxygen-haemo-

globin desaturation in highly trained subjects during maximal exercise³⁻¹⁰. When using pulse ear oximetry, it has been reported that the incidence of oxygen-haemoglobin desaturation (%HbO₂ ≤ 91%) during maximal exercise occurs in approximately 50% of the highly trained athletes examined⁷. It is interesting to note, however, that there has been no scientific study which has examined the occurrence of EIH in highly trained athletes when saturation is determined using invasive procedures (arterial blood) rather than non-invasive ear oximetry analysis. Therefore, it was the purpose of this study to examine the occurrence of arterial oxygen-haemoglobin desaturation during maximal exercise in highly trained athletes.

Materials and methods

Eleven healthy, trained cyclists of mean(s.d.) age 23.0(3.5) years served as subjects after signing an institutionally approved informed consent. Each subject had a minimum of one year's experience as a licensed (United States Cycling Federation) competitive cyclist. Each subject reported to the laboratory 12 h after eating for data collection. Baseline ventilatory and metabolic measurements were collected following 20 min of supine rest. Subsequently, an arterial catheter (Arrow 1.25 inch, 20Fr) was placed into either the right or left radial artery using the procedure described by Wasserman *et al.*¹¹. The ear probe for the Biox IIa oximeter (Ohmeda, Boulder, Colorado, USA) and interface cables were secured to the subject so as to minimize movement artefact as determined by laboratory experience with the instrumentation. Resting arterial blood samples and ear oximetry readings were obtained simultaneously 15 min after the catheterization procedures.

Experimental session

Each subject performed a multistage cycle ergometer aerobic capacity ($\dot{V}O_{2\max}$) test at an initial work rate of 135 W with a 45 W increase every minute until exhaustion. A 'true' test of $\dot{V}O_{2\max}$ was considered to be achieved if each subject met two of the following four criteria: a plateau in $\dot{V}O_2$ with increased exercise workload; a heart rate of 10% more or less than the

Address for correspondence: Dr Dale D. Brown, Department of Health, Physical Education, Recreation and Dance, Horton Fieldhouse, Room 214, Illinois State University, Normal, Illinois 61761, USA

© 1993 Butterworth-Heinemann Ltd
0306-3674/93/030167-04

Table 1. General characteristics of the subjects

Subject number	Age (years)	Height (cm)	Weight (kg)	$\dot{V}O_{2\max}$ (ml kg ⁻¹ min ⁻¹)	RER	Max HR (beats min ⁻¹)	Max VE (l min ⁻¹)	Lactate (mmol)
1	28	179.1	70.0	—*	—*	191	122.0	9.3
2	18	177.8	80.5	65.5	1.20	189	190.6	10.3
3	28	182.9	86.4	62.4	1.12	194	176.4	8.3
4	23	182.9	77.3	72.7	1.13	184	210.7	8.5
5	22	177.8	75.0	65.7	1.15	186	166.0	9.3
6	22	191.0	81.8	68.0	1.24	188	199.3	9.2
7	24	180.3	84.5	61.9	1.02	195	152.5	12.2
8	20	190.5	79.5	74.3	1.16	181	150.7	9.7
9	20	170.2	56.8	73.4	1.15	208	169.3	9.9
10	19	180.3	70.5	66.2	1.26	192	134.1	9.5
11	26	177.8	81.2	58.9	1.19	185	173.2	6.3
Mean(s.d.)	22.7(3.5)	181.0(5.9)	76.7(8.4)	66.9(5.2)	1.16(0.07)	190.3(7.3)	167.7(26.9)	9.3(1.4)

*Data unavailable. RER, respiratory exchange ratio; max HR, maximum heart rate; max VE, maximum ventilation

age-predicted maximum; a respiratory exchange ratio greater than 1.0; and a peak whole blood lactate in excess of 8.0 mmol^{6,7}. As indicated in *Table 1*, each subject achieved a 'true' test of maximal aerobic capacity.

Before starting to exercise the cyclists were instructed to remain seated throughout the entire cycle exercise test to avoid extraneous movements to the ear oximeter probe. The Monark cycle ergometer (Varberg, Sweden) was modified with toeclips, cycling seat and handle bars so as to simulate the cyclist's typical racing bicycle. After calibration with standardized reference gases, metabolic and ventilatory data (Quinton Q-Plex, Seattle, Washington, USA) were monitored continuously throughout the cycle ergometer test, using open circuit spirometry. Oxyhaemoglobin saturations for arterial blood (%SaO₂) and ear oximetry (%SpO₂) were simultaneously obtained every 2 min throughout the cycle exercise test and at maximal exercise capacity. The arterial blood samples were stored in an ice bath and subsequently analysed within 30 min of the time of withdrawal¹² for arterial pH, partial pressure of carbon dioxide and oxygen using a Corning 170 Blood Gas Analyzer (Ciba Corning, Medfield, Massachusetts, USA). The arterial oxygen-haemoglobin saturation was calculated automatically by the blood gas analyser. No corrections were made for changes in blood temperature, since it has been shown that short duration, high-intensity exercise produces a minimal increase in core temperature (approximately 0.5°C)^{13,14} with these temperature changes having an insignificant effect on the determination of arterial oxygen-haemoglobin saturation¹⁰. Calibration of the blood gas analyser was performed using precision grade reference tank gases and commercially prepared precision grade calibration buffer standards. Blood lactate concentration was determined using a Yellow Springs Lactate Analyzer (Yellow Springs, Ohio, USA) following standardized techniques for calibration.

Statistics

Analysis of variance for a repeated measures design was used to test for statistical differences, followed by

post-hoc analysis (APP-STAT; Statsoft, 1986, Tulsa, Oklahoma, USA). Statistical significance was accepted at a probability of less than 0.05.

Results

When using ear oximetry, it has been reported that the incidence of oxyhaemoglobin desaturation (as defined by: %HbO₂ < 91%) in highly trained athletes during maximal exercise occurs in approximately 50% of those examined⁷. Applying that criterion to the results of the present study, seven of 11 subjects (64%) would be classified as having arterial oxyhaemoglobin desaturation when desaturation is determined through non-invasive ear oximetry. In contrast, none of the subjects would have been classified as having arterial oxyhaemoglobin desaturation when %HbO₂ was determined using arterial blood sampling.

The %SpO₂ and %SaO₂ values were similar under resting conditions with the mean(s.d.) values being 96.5(1.6)% and 97.3(0.6)%, respectively. However, statistical analysis revealed that there was a significant difference in %HbO₂ determinations for arterial blood and ear oximetry at 6-min, 8-min, and maximal exercise sampling times (*Table 2* and *Figure 1*), with these differences becoming prevalent at an exercise intensity of greater than 73% $\dot{V}O_{2\max}$.

Discussion

Although previous research using pulse oximetry indicates that approximately 50% of endurance trained athletes develop EIH (%HbO₂ < 91%), the findings of the present study using arterial blood %SaO₂ failed to find EIH in any of the endurance trained athletes examined. However, when using pulse ear oximetry, 64% of the athletes examined exhibited EIH. The %SaO₂ responses observed in the present study are similar to the data reported by others who have investigated arterial desaturation in highly trained individuals. Dempsey *et al.*³ and Hopkins and McKenzie⁴, using arterial blood sampling, have shown reductions in %SaO₂ from resting values of approximately 5% (%SaO₂ at maximum exercise = 92%) during maximal treadmill exercise using highly trained athletes. In this study the

Table 2. Absolute and change from rest values for percentage oxygen-haemoglobin saturation during the cycle ergometer test

Sampling time (min)	Oxygen uptake (% of max $\dot{V}O_2$)	Absolute value* (% SO_2)		Change from rest*† (% SO_2)	
		Ear oximeter	Arterial blood samples	Ear oximeter	Arterial blood samples
Rest	8.9	96.5(1.6)	97.3(0.6)	—	—
2	37.3	95.8(1.3)	96.7(0.8)	-0.6(1.6)	-0.6(1.0)
4	57.1	94.9(1.6)	96.2(1.6)	-1.6(2.3)	-1.1(1.6)
6	73.4	93.6(2.2)‡	95.9(1.5)	-2.8(3.0)	-1.3(1.3)
8	89.8	92.8(2.2)‡	95.6(1.5)	-3.5(3.3)	-1.7(1.2)
Maximal exercise	99.6	89.9(2.0)‡	95.0(1.7)	-6.6(2.9)	-2.3(1.5)

*Values are mean(s.d.); †Exercise % SO_2 minus resting % SO_2 ; ‡Statistically different ($P < 0.05$)

% SpO_2 decreased by 2.3%, from 97.3% at rest to 95.0% during maximal exercise. However, it is interesting to note that none of the studies in the scientific literature that have examined invasively determined arterial blood desaturation in highly trained subjects has found % SpO_2 reductions near the magnitude recorded by non-invasive ear oximetry^{7,9,15}. Oxygen-haemoglobin saturation determined from ear oximetry (% SpO_2) will typically decrease 8–10% from rest, with the % SpO_2 values ranging between 84% and 91%^{9,15}. Similar changes were observed in the present study with the % SpO_2 at maximal exercise being decreased by approximately 7% from resting conditions.

Previous investigations have shown that the use of ear oximetry is valid and reliable for determining % HbO_2 in subjects who are pulmonary patients, smokers, and 'normal', 'healthy' individuals, at rest, under normoxic and hypoxic conditions^{16–21}. A study by Martin *et al.*²² found that pulse, ear and finger oximeters are valid at monitoring arterial oxyhaemoglobin saturations in highly trained athletes during exercise. Yet, in that study, data were presented as the mean difference between % SpO_2 and % HbO_2 (i.e. bias) rather than actual oxyhaemoglobin saturation, therefore it is not possible to relate those findings to the incidence of EIH in highly trained athletes. Additionally, the pulse oximeter used in the validation study by Martin *et al.*²² was the newest model pulse ear oximeter, whereas, an older model ear oximeter was used in the original study which

showed the incidence of desaturation to be approximately 50% of the subjects examined⁸.

The reason for the significant differences in oxyhaemoglobin saturation determinations between ear oximetry and arterial blood at the higher exercise intensities is not known, however, there are a number of possible explanations for the observed differences. It has been speculated that a reduction in ear perfusion during exercise may be a factor in the difference²³. Although the Biox Ila is equipped with a low perfusion indicator there were no indications from the oximeter that a reduction in perfusion occurred during exercise in any of the subjects in the present study. However, the sensitivity of the low perfusion indicator to respond to perfusion limitations during exercise in highly trained subjects is unknown. Dempsey *et al.*³ showed that in highly trained runners during maximal exercise there is a substantial decrease in mean erythrocyte capillary transit time by comparison with 'average' individuals. The coupled effects of exercise perfusion alterations and an increased rate of blood flow in highly trained subjects during maximal exercise may result in an inability of the oximeter to measure oxygen-haemoglobin saturation of these subjects accurately at high power outputs. Research has indicated that the improper attachment of the oximeter ear probe contributes to the inaccuracy of estimating arterial oxygen-haemoglobin saturation¹⁸. The extent to which movement artefact contributed to the significantly reduced estimates of % HbO_2 in the ear oximeter is unknown. Even though precautions to prevent extraneous movements of the ear probe were taken as directed by the manufacturer, the exercise requirements may have interacted with the ear oximeter thereby contributing to the resulting difference.

The presence of elevated levels of carboxyhaemoglobin (HbCO) have been shown to alter the ability of oximeters to estimate the oxyhaemoglobin saturation^{5,20}. Likewise, calculated oxyhaemoglobin saturations from arterial blood samples may not provide a true reflection of actual oxyhaemoglobin saturation since calculated values do not account for HbCO binding. Although HbCO levels were not measured in the present study, there was no reason to expect elevated arterial HbCO, since none of the subjects were smokers. Had an elevated HbCO been present in these subjects it would be expected that a

Figure 1. Oxygen-haemoglobin saturation during exercise for arterial blood samples and ear oximetry. ○, arterial blood; ●, ear oximetry; *, significantly different

consistent difference in saturation would have been present between the ear oximeter and arterial blood, irrespective of workload.

Although newer pulse oximeter technology is available, numerous scientific investigations are still being completed using the older oximeter technology^{7, 24, 25}. Given the lack of scientific validation studies for the use of older oximeters in exercising highly trained athletes and the significant differences observed for %HbO₂ between non-invasive and invasive methods evidenced in the present study, it would appear that validation of ear oximetry for determining arterial oxygen-haemoglobin saturation in highly trained individuals is strongly warranted. Co-oximeter analysis of arterial blood, which provides a detailed analysis of total haemoglobin and its subfractions (oxyhaemoglobin, methaemoglobin and carboxyhaemoglobin), would be appropriate to ensure the accuracy of reporting the incidence of oxyhaemoglobin desaturation in highly trained athletes during maximal exercise.

This study found that ear oximetry overestimates the incidence of EIH and underestimates the oxyhaemoglobin saturation in highly trained cyclists during exercise in comparison to those measurements made from arterial blood. Although ear oximetry can provide qualitative analysis for arterial blood oxygenation, the validity and accuracy of this technology used with highly trained individuals during maximal exercise remains in question and requires further examination.

Acknowledgements

Work was done at the Coal Miner's Respiratory Disease Clinic/Cardiopulmonary Laboratory of Herrin Hospital, Herrin, Illinois, USA.

The authors would like to thank Deb Van Tassel and Regina Minton for their technical support in the collection of data for this study. Additional thanks are given to the administration, specifically Joe Hutchcraft, and the staff of the Cardiopulmonary Laboratory and the Respiratory Therapy Department of the Herrin Hospital for their support throughout this project. The authors would also like to extend a note of thanks to Dr John T. Mouw for his assistance in the statistical design for this study.

References

- 1 Dempsey JA. Is the lung built for exercise? *Med Sci Sports Exerc* 1986; **18**: 143–55.
- 2 Dempsey JA, Johnson BD, Saupe KW. Adaptations and limitations in the pulmonary system during exercise. *Chest* 1990; **97**: 81S–87S.
- 3 Dempsey JA, Hanson PG, Henderson KS. Exercise-induced arterial hypoxaemia in healthy human subjects at sea level. *J Physiol (Lond)* 1984; **355**: 161–75.
- 4 Hopkins SR, McKenzie DC. Hypoxic ventilatory response and arterial desaturation during heavy work. *J Appl Physiol* 1989; **67**: 1119–24.
- 5 Powers SK, Dodd S, Freeman J, Ayers GD, Samson H, McKnight T. Accuracy of pulse oximetry to estimate HbO₂ fraction of total Hb during exercise. *J Appl Physiol* 1989; **67**: 300–4.
- 6 Powers SK, Lawler J, Dempsey JA, Dodd S, Landry G. Effects of incomplete pulmonary gas exchange on $\dot{V}O_{2, \max}$. *J Appl Physiol* 1989; **66**: 2491–5.
- 7 Powers SK, Dodd S, Lawler J et al. Incidence of exercise induced hypoxemia in elite endurance athletes at sea level. *Eur J Appl Physiol* 1988; **58**: 298–302.
- 8 Powers SK, Williams J. Exercise-induced hypoxaemia in highly trained athletes. *Sports Med* 1987; **4**: 46–53.
- 9 Powers SK, Dodd S, Woodyard J, Beadle RE, Church G. Haemoglobin saturation during incremental arm and leg exercise. *Br J Sports Med* 1984; **18**: 212–16.
- 10 Smyth RJ, D'Urzo AD, Slutsky AS, Galko BM, Rebuck AS. Ear oximetry during combined hypoxia and exercise. *J Appl Physiol* 1986; **60**: 716–19.
- 11 Wasserman K, Hansen JE, Sue DY, Whipp BJ. *Principles of Exercise Testing and Interpretation*. Philadelphia, USA: Lea and Febiger, 1984.
- 12 Shapiro BA, Harrison RA, Watson JR. *Clinical Applications of Respiratory Gases*. Chicago, Illinois, USA: Year Book Medical, 1982.
- 13 Bergh U, Ekblom B. Physical performance and peak aerobic power output at different body temperatures. *J Appl Physiol* 1979; **46**: 885–9.
- 14 Saltin B, Gagge AP, Bergh U, Stolwijk JAJ. Body temperatures and sweating during exhaustive exercise. *J Appl Physiol* 1972; **32**: 635–43.
- 15 Williams JH, Powers SK, Stuart MK. Hemoglobin desaturation in highly trained athletes during heavy exercise. *Med Sci Sports Exerc* 1986; **18**: 168–73.
- 16 Escourrou PJL, Delaperche MF, Visseaux A. Reliability of pulse oximetry during exercise in pulmonary patients. *Chest* 1990; **97**: 635–8.
- 17 Cecil WT, Petterson MT, Lamoopun S, Rudolph CD. Clinical evaluation of the Biox IIA ear oximeter in the critical care environment. *Respir Care* 1985; **30**: 179–83.
- 18 Reis AL, Farrow JT, Clausen JL. Accuracy of two ear oximeters at rest and during exercise in pulmonary patients. *Am Rev Respir Dis* 1985; **132**: 685–9.
- 19 Chapman KR, D'Urzo A, Rebuck AS. The accuracy and response characteristics of a simplified ear oximeter. *Chest* 1983; **83**: 860–4.
- 20 Shippy MB, Petterson MT, Whitman RA, Shivers CR. A clinical evaluation of the BTI Biox II Ear Oximeter. *Respir Care* 1984; **29**: 730–5.
- 21 Fahey PJ, Gruber S, Siska D, Leach B. Clinical evaluation of a new ear oximeter. *Am Rev Respir Dis* 1983; **127** (Suppl): 129.
- 22 Martin D, Powers S, Cicale M, Collop N, Huang D, Criswell D. Validity of pulse oximetry during exercise in elite athletes. *J Appl Physiol* 1992; **72**: 455–8.
- 23 Hansen JE, Casaburi R. Validity of ear oximetry in clinical exercise testing. *Chest* 1987; **91**: 333–7.
- 24 Rhodes J, Coast JR, Crouse SF. Arterial desaturation in healthy untrained subjects. *Med Sci Sports Exerc* 1988; **20** (Suppl): 281.
- 25 McCusker M, Brilla LR. Exercise induced hypoxemia in female athletes. *Med Sci Sports Exerc* 1992; **24** (Suppl): 415.