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# Effects of Acute Hypoxia on the Determination of Anaerobic Threshold Using the Heart Rate-Work Rate Relationships During Incremental Exercise Tests

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## Summary

Anaerobic threshold which describes the onset of systematic increase in blood lactate concentration is a widely used concept in clinical and sports medicine. A deflection point between heart rate-work rate has been introduced to determine the anaerobic threshold non-invasively. However, some researchers have consistently reported a heart rate deflection at higher work rates, while others have not. The present study was designed to investigate whether the heart rate deflection point accurately predicts the anaerobic threshold under the condition of acute hypoxia. Eight untrained males performed two incremental exercise tests using an electromagnetically braked cycle ergometer: one breathing room air and one breathing 12 % $O_2$. The anaerobic threshold was estimated using the V-slope method and determined from the increase in blood lactate and the decrease in standard bicarbonate concentration. This threshold was also estimated by in the heart rate-work rate relationship. Not all subjects exhibited a heart rate deflection. Only two subjects in the control and four subjects in the hypoxia groups showed a heart rate deflection. Additionally, the heart rate deflection point overestimated the anaerobic threshold. In conclusion, the heart rate deflection point was not an accurate predictor of anaerobic threshold and acute hypoxia did not systematically affect the heart rate-work rate relationships.

**Key words**

Exercise test • Hypoxia • Heart rate deflection point • Anaerobic threshold

## Introduction

During muscular exercise performance, the onset of systematic increase in blood lactate concentration which is called anaerobic threshold (Wasserman *et al.* 1990) is a widely used criterion in clinical medicine and sports science. The anaerobic threshold can be used in the assessment of aerobic fitness of subjects (Spurway 1992, Wasserman *et al.* 1994), establishing intensity of exercise (Whipp 1996), optimum training work rate in subjects with different fitness levels (Belman and Gaesser 1991, Casaburi *et al.* 1995) and even preoperative evaluation of patients undergoing major surgery (Older *et al.* 1993).

Accurate determination of anaerobic threshold requires frequent arterial (Yoshida *et al.* 1981) or arterialized (McLoughlin *et al.* 1992) blood withdrawals while pedaling on an ergometer at steadily increasing workloads. Despite some controversy, many investigators showed that the anaerobic threshold during exercise can be accurately estimated non-invasively by methods based on ventilatory and pulmonary gas exchange indices



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(Beaver *et al.* 1986, Whipp *et al.* 1986, Wasserman *et al.* 1994, Thin *et al.* 2002).

In addition, Conconi *et al.* (1982) introduced a simple non-invasive method for estimating the anaerobic threshold based on heart rate-work rate relationship during exercise performance. During exercise, the heart rate generally increases in a direct relationship to the energy expended until the point where the heart rate begins to increase out of proportion to the work rate, which is described as the deflection point. These authors proposed that the observed deviation from linearity in the heart rate-work rate relationship during exercise coincided with the anaerobic threshold (Conconi *et al.* 1982).

The physiological mechanisms of the deflection point in heart rate-work rate relationship are not yet completely clarified and it has been suggested that it is an artefact produced through execution of the incremental test protocol rather than a physiological phenomenon.

During progressively increasing work rate exercise tests, when the exercise intensity increases from aerobic to anaerobic intensity, anaerobic metabolic products could affect the heart rate-work rate relationship and result in a deflection point. The results of the studies based upon heart rate-work rate relationship are controversial; some studies showed a good relationship between the heart rate deflection point and anaerobic threshold (Ballarin *et al.* 1989, Conconi *et al.* 1996) while some authors did not confirm such a significant relationship (Jones and Doust 1997, Bodner and Rhodes 2000) during exercise.

The aim of this study was to assess the validity of the deflection point in heart rate-work rate relationship as an index for estimating the anaerobic threshold under conditions of acute hypoxia which is known to have a considerable influence on aerobic and anaerobic metabolism during exercise tests.

## Methods

Eight healthy untrained male subjects participated after providing written informed consent approved by the institutional ethics committee. The subjects mean age, weight and height (± S.D.) were 22.3±4.3 years, 75.6±10.5 kg and 179.5±6.6 cm, respectively.

Each subject performed two incremental exercise tests to exhaustion (Whipp *et al.* 1981) on a computer controlled, electromagnetically braked cycle ergometer (Lode, Excalibur) while breathing either room air (control) or air with 12 % $O_2$ (hypoxia) on different days. The exercise test consisted of an initial period of 4 min of cycling at 20 W as a warm-up, followed by the work rate being increased by 15 W/min to the limit of the subject's tolerance. The subjects were required to maintain a constant pedaling frequency within the range of 60-80 rpm. For the hypoxia study, the warm-up period with breathing room air was extended for a further 4 min, during which subjects breathed the hypoxic gas, and the ramp phase of the test started with hypoxic gas breathing as in the normoxia study.

During the exercise test, the subjects breathed through a mouthpiece attached to a low dead-space, low-resistance turbine volume transducer (Alpha Technologies, VMM) for continuous measurement of inspired and expired volumes and flows. The dead space and resistance of the system were 90 ml and < 0.7 cm $H_2O$/l/s at 2 l/s.

The calibration of the flow signal was performed immediately before each experiment by using a 3-liter syringe. Respired air was drawn from the mouthpiece continuously and sampled by a quadrupole mass spectrometer (CaSE, QP9000) for continuous monitoring of $O_2$, $CO_2$ and $N_2$ concentrations in respired air; precision-analyzed gas mixtures whose concentrations spanned the range of interest were used for calibration immediately prior to each test. The heart rate was derived beat by beat from the R-R interval of a standard six-lead ECG (Quinton 5000). Arterial $O_2$ saturation was monitored continuously throughout the test by using pulse oximetry (Ohmeda, 3700).

Following analogue-to-digital conversion, the electrical signals from these devices were sampled every 20 ms and processed on-line by a digital computer for the computation and display, breath-by-breath, of minute ventilation ($V_E$ BTPS), $O_2$ uptake ($VO_2$ STPD), $CO_2$ output ($VCO_2$ STPD), respiratory exchange ratio (R), as previously described by Beaver *et al.* (1981) and Jenkins *et al.* (1989).

Prior to each protocol, a 20-gauge catheter was inserted into a vein on the dorsum of the left hand, warmed for at least 20 min by enclosure in a large heating pad so as to "arterialize" the blood (McLoughlin *et al.* 1992). Once the subject was seated on the cycle ergometer, the temperature of the hand was maintained by placing it on a heating pad (located on a small

platform immediately adjacent to the left handlebar) and also heating it from above with an infra-red lamp.

Blood was sampled at rest and after 3 min of cycling at 20 W. During the ramp phase, samples were taken at approximately 1-min intervals. Following expulsion of any air bubbles, syringes were immediately capped and stored in ice. Blood was centrifuged at 3000 rpm for 10 min, serum was separated from red blood cells and stored at –18 ºC for lactate analysis.

At each sampling point, the catheter was flushed clean and two 3 ml samples were withdrawn into heparinized syringes: one for blood gas and acid-base analysis (Instrumentation Laboratories, Model 1306), and the other for lactate analysis (Analox, GM7 Microstat). Lactate concentrations were assessed by a method previously described (Olsen 1971).

The $VO_2$ peak was taken as the highest oxygen attained at the end of the ramp test. Anaerobic threshold was estimated non-invasively by the "V-slope" technique of Beaver *et al.* (1986) and determined from the arterialized blood samples (Fig. 1). In addition, the heart rate in response to the progressively increasing work rate exercise tests was plotted to estimate the anaerobic threshold.

The paired t-test was used to evaluate the statistical significance of differences between mean control and hypoxia responses, ± S.D. The significance level was set at $P<0.05$.

## Results

The estimated anaerobic threshold using the V-slope break-point is associated with the onset of the systematic increase in blood lactate concentration and a decrease in standard bicarbonate concentration in both control and hypoxia studies (Fig. 1).

Hypoxia resulted in a marked reduction in $VO_2$ at the anaerobic threshold (Figs 1 and 3). The $VO_2$ at anaerobic threshold was 1.69±0.27 l/min for the hypoxia study and 1.88±0.33 l/min for the control study (Table 1). However, we have found that the aerobic to anaerobic metabolic transitions corresponded to exercise intensity 57.8±6 % of maximal $VO_2$ for the control and 63.0±2 % of maximal $VO_2$ for the hypoxia studies.

As expected, both the peak $VO_2$ and the peak work rate were also reduced significantly during the hypoxia study (2.66±0.44 l/min and 206±25 W) compared to the control study (3.34± 0.72 l/min and 262±32 W) (Table 1).

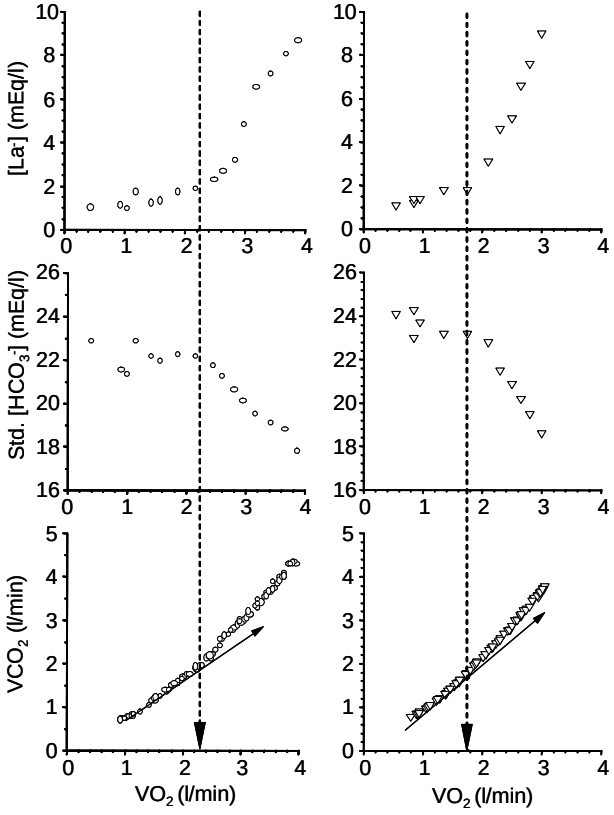


**Fig. 1.** V-slope estimation of anaerobic threshold with respect to the profiles of the measured lactate ([La]) and standard bicarbonate (std.[$HCO_3$]) during the incremental exercise with breathing room air (o) and breathing 12 % $O_2$ (∇) studies. The vertical dashed lines represent the V-slope break-point.

**Table 1.** Peak $O_2$ uptake ($VO_2$ peak), peak exercise performance (WR peak), anaerobic threshold ($\theta_{an}$) and the ratio of $VO_2$ at the anaerobic threshold to peak $O_2$ uptake ($\theta_{an}$ %) for control and hypoxia studies.

| | **$VO_2$ peak (l/min)** | **$\theta_{an}$ (l/min)** | **$\theta_{an}$ %** | **WR peak (W)** |
|---|---|---|---|---|
| *Control* | 3.22±0.6 | 1.84±0.3 | 57.8±6 | 261±33 |
| *Hypoxia* | 2.63±0.4* | 1.65±0.2* | 63.0±2 | 208±27* |

Data are Means ± S.D. *represent a significant difference ($p<0.05$) from the control study.

During the incremental exercise test, the deflection point in the heart rate-work rate relationships was not found in all subjects. In the control study, the heart rate increased linearly with increasing work rate until the exhaustion in six out of eight subjects despite the clear break point in V-slope relationships, e.g. R was found to be 0.996 (P<0.0001) in the subject depicted in Figure 2. In the hypoxia study, the deflection point in the heart rate-work rate relationships occurred in four subjects, i.e. the heart rate deflection occurred in two subjects where it was not seen in the control study and in two others where it also occurred in the control study.

In our studied group, the heart rate-work rate deflection point did not match with the anaerobic threshold in any of the subjects. The heart rate deflection point (i.e. two in the control and four in the hypoxia groups) occurred after the anaerobic threshold in all subjects (Fig. 3).

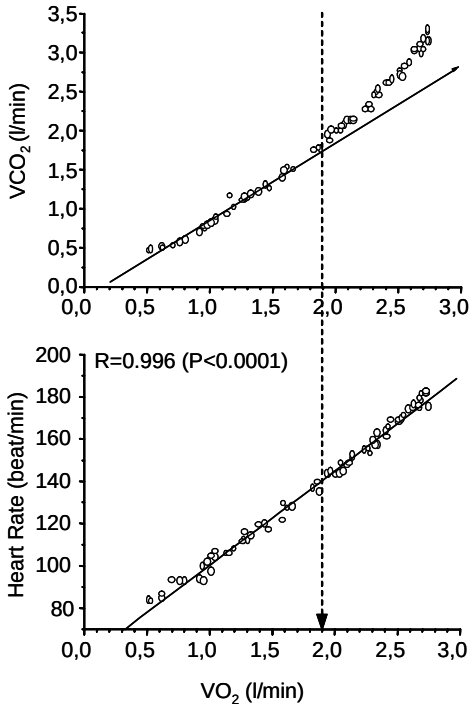


**Fig. 2.** $CO_2$ output-to-$O_2$ uptake ($VCO_2/VO_2$) relationships and heart rate in response to the incremental exercise test. The vertical dashed line represents the estimated anaerobic threshold. A linear heart rate-work rate relationship to the incremental exercise test (R=0.996, P<0.0001).

## Discussion

In the present study, we used the V-slope relationships to estimate the anaerobic threshold which is proposed as a more effective non-invasive method than the other conventional methods based on ventilatory and pulmonary gas exchange indices with the exception of the condition of altered body $CO_2$ stores prior to the exercise test (Ozcelik *et al.* 1999). This is known to be unaffected by ventilatory alterations (Beaver *et al.* 1986, Ozcelik and Colak 2002), since the V-slope method based on the determination of changes in $CO_2$ production to $O_2$ consumption ($VCO_2/VO_2$) ratio are due to aerobic and anaerobic metabolism. It has two linear slopes: a lower slope reflecting aerobic metabolism and an upper slope reflecting aerobic and also anaerobic metabolism (Beaver *et al.* 1986). The break point between these two slopes occurred at approximately 60 % of $VO_2$ peak and was associated with a systematic increase in blood lactate and decrease in standard bicarbonate values. These values correspond to the results of other investigations in which it was found that the mean anaerobic threshold in normal sedentary men ranged between 50 % to 70 % of maximal $VO_2$ (Hansen *et al.* 1984).

The heart rate deflection point has been proposed as a useful index to evaluate physical fitness. It has been suggested that the application of the heart rate deflection point to children and adolescents in running may prove to be useful in determining of aerobic power during growth (Ballarin *et al.* 1989, Baraldi *et al.* 1989). However, in a close correlation with the findings of previous investigations, we did not observe a deflection point in heart rate-work rate relationships in all subjects in our study group (Jones and Doust 1997, Bourgois and Vrijens 1998, Vachon *et al.* 1999). While there was a definitive and statistically significant increase in the pulmonary gas exchange response to an increasing work rate, the heart rate remained linear in some subjects (Fig. 2).

During incremental exercise, a heart rate deflection point was observed in two out of eight subjects in the control study and in four out of eight subjects in the hypoxia study. When a deviation of heart rate from linearity was evident, it occurred at a systematically higher intensity than the anaerobic threshold (Fig. 3). The break point in heart rate-work rate relationships occurred at approximately 80 % of peak $VO_2$. Furthermore, the deflection point in the heart rate-work rate relationships occurred at approximately 95 % of the maximum heart rate in both studies (Jones and Doust 1997).

During exercise, noradrenaline and adrenaline levels have been shown to be closely associated with the onset of systematic increase in blood lactate concentration (Mazzeo and Marshall 1989, Weltman *et*

*al.* 1994). In addition, increases in arterial catecholamine levels have been reported during exercise with hypoxic breathing (Rowell *et al.* 1986) which is also expected to affect the heart rate-work rate relationship. Hypoxia would be expected to result in impairment of aerobic ATP production by skeletal muscles (Idstrom *et al.* 1985). In hypoxia study, it is also logical to expect enhanced cardiovascular responses due to increased lactate (Gregory *et al.* 1987) resulting in an early rise in sympathetic activity (Warner and Mitchell 1991). Hypoxia which is known to have important effects on lactate metabolism did not systematically alter the heart rate-work rate relationships. Heart rate-work rate relationships during an incremental exercise test were not affected by 12 % $O_2$ breathing in six out of eight subjects. Hypoxia resulted in a shift between heart rate-work relationship from linear to a right side breakpoint in two subjects (Bodner and Rhodes 2000). The deflection point or the linearity in the heart rate-work rate relationships has not clear explanations yet. Increased lactate levels (Conconi *et al.* 1996), exercise-induced hyperkalemia (Lucia *et al.* 2002) and the left ventricular ejection fraction, which is an important parameter of myocardial functions (Hofmann *et al.* 1994) have been suggested as the reason for the heart rate deflection during exercise tests.

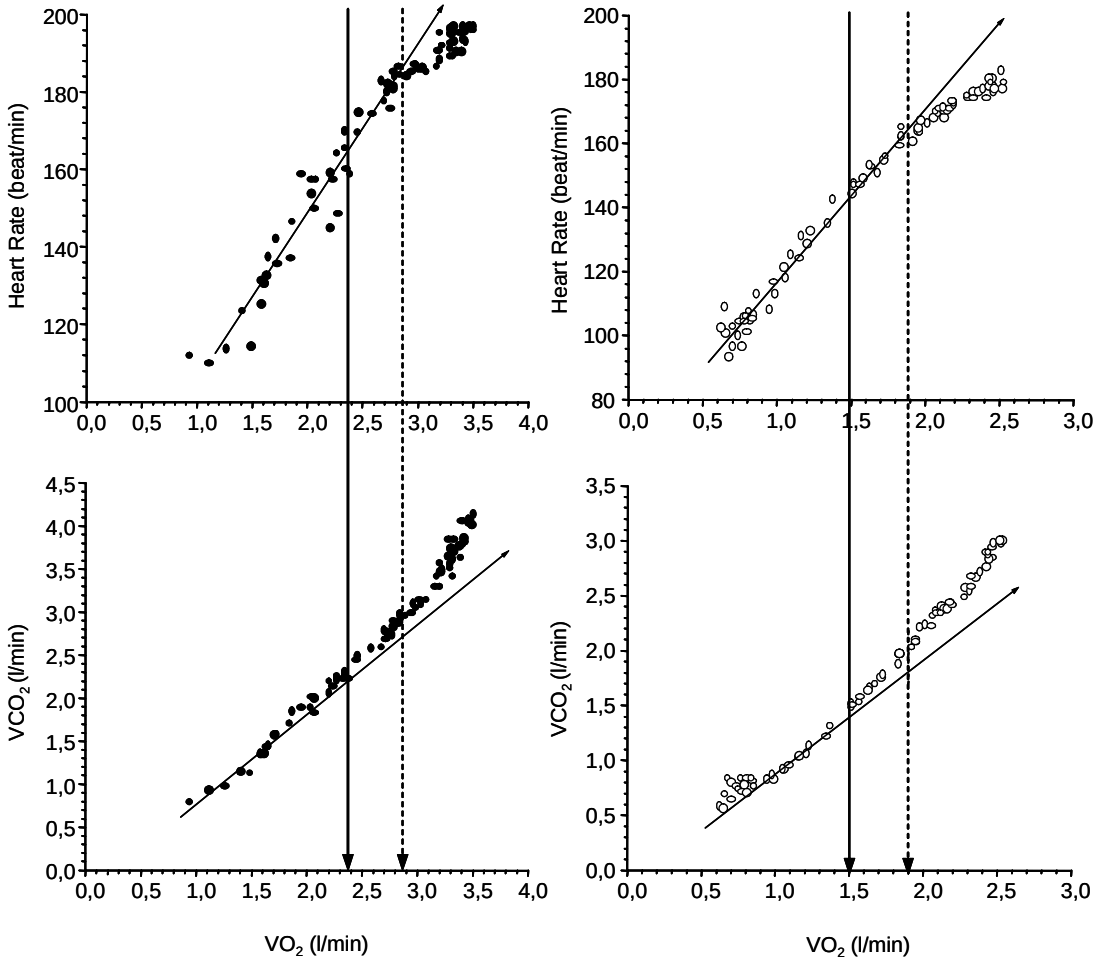


**Fig. 3.** $CO_2$ output-to-$O_2$ uptake ($VCO_2/VO_2$) relationships and heart rate in response to the incremental exercise tests for the control (•) and for the hypoxia (o). The vertical solid lines represent the estimated anaerobic threshold. The vertical dashed lines represent the deflection in heart rate response.

In addition, it has been suggested that the duration of exercise stages may also be responsible for the loss of linearity in heart rate-work rate relationships (Vachon *et al.* 1999). However, Conconi *et al.* (1996) showed that heart rate adapts to each new work rate within a 30-s interval. The degree of heart rate deflection is highly dependent upon the type of protocol used in the study (Bodner and Rhodes 2000). The subject's physical

condition may also be responsible for the deflection point in the heart rate-work rate relationships. This is because we used untrained male subjects, while some investigators used well trained athletes (Conconi *et al.* 1982, Droghetti *et al.* 1985).

The validity of the heart rate deflection point for assessing the anaerobic threshold is uncertain. In our study group, this may be due to a high degree of linear relationship between heart rate and work rate and to the fact that the deflection point was not associated with the anaerobic threshold both in the control and hypoxia studies. Therefore, caution should be used when determining the anaerobic threshold from single measurements of heart rate-work rate response with the intention to apply it in clinical and sports medicine.

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