# Transcutaneous, Noninvasive $P_{O_2}$ Monitoring in Adults During Exercise and Hypoxemia

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Abstract. A new, commercially available, transcutaneous (tc)  $P_{O_2}$  monitor was tested in adult females and in laboratory animals to assess its applicability in measuring arterial oxygen tension during physiological stress. Observed values on dogs correlated well with direct measurements of arterial  $P_{O_2}$  and with previous data obtained from measurements of arterial blood during exercise and hypoxemia. In our female subjects the unit responded rapidly to changes in inspired ambient oxygen and electrical stability was excellent during maximal exercise tests. Transcutaneous  $P_{O_{1}}$ decreased to an average of 87.8 Torr during maximum exercise breathing 20.9% O<sub>2</sub>, and to 32 Torr while breathing 12.6% O2 at maximum work. Two distinct patterns of response in tc  $P_{O_2}$  were observed during hypoxic and normoxic exercise. The technique appears to have substantial future application both in clinical and physiological investigation involving adult subjects.

Key words: Transcutaneous  $P_{O_2}$  – Exercise – Hypoxemia – Cutaneous blood flow.

## Introduction

The ability to continuously monitor arterial oxygen tension in humans is of particular advantage in a wide range of clinical and physiological investigations. The recent development of a transcutaneous oxygen monitor may provide a suitable noninvasive alternative to direct arterial sampling. Qualitative assessment of minute-to-minute changes in blood oxygen tension and skin blood flow can be obtained while eliminating the potential discomfort and risk of arterial punctures and catheterization. The principle of transcutaneous oxygen monitoring (tc  $P_{O_2}$ ) was developed by Huch, Huch, and Lübbers [4, 5] and involves the use of a heated Clark-type electrode which is attached to the skin by an adhesive ring. Although oxygen tension at the skin surface is normally close to zero, heat generated by the electrode  $(43-45^{\circ} \text{ C})$  produces local tissue hyperthermia and the vasodilation induced results in blood under the probe becoming arterialized. Oxygen diffuses through the skin and contact solution to the  $P_{O_2}$  electrode, where it is reduced, and the resultant current is proportional to the oxygen partial pressure of the dilated capillaries under the probe.

This technique was primarily developed to assist in fetal and neonatal monitoring of PaO2, and excellent correlation (r = 0.97 and 0.96) between arterial and tc  $P_{0}$ , have been reported in healthy neonates [3,8]. High correlations have also been reported for obstetrical-gynocological and surgical patients (r = 0.92) [3], but data on adults has not been extensive and the method has not been validated under laboratory conditions. We recently compared to  $P_{O_2}$  and arterial  $P_{O_2}$  in the anesthetized adult dog using a commercially available transcutaneous oxygen monitor developed by Radiometer Company [9] and a Radiometer ABL-1 bloodgas autoanalyzer. The correlations were sufficiently precise to warrant further evaluation of this method in humans during physiological stress. This report constitutes our finding from these tests.

## Methods

Six healthy female members of the American Anapurna Expedition<sup>1</sup> were monitored by transcutaneous  $P_{O_2}$  electrode while performing

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<sup>&</sup>lt;sup>1</sup> The nature and purpose of the study and the risks involved were explained verbally and given on a written form to each subject prior to their voluntary consent to participate. The protocol and procedures for this study have been approved by the Committee on Activities Involving Human Subjects, of the University of California, Santa Barbara

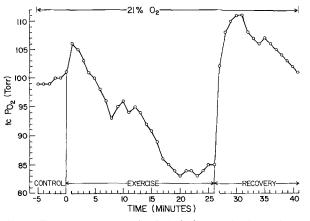


Fig. 1. Transcutaneous  $P_{O_2}$  (to  $P_{O_2}$ ) during maximal exercise and recovery

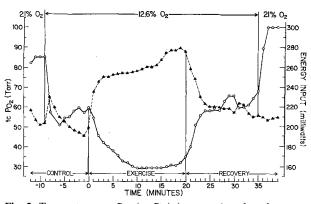


Fig. 2. Transcutaneous  $P_{O_2}$  (to  $P_{O_2}$ ) (O——O) and probe energy input (milliwatts) ( $\Delta$ ---- $\Delta$ ) during maximal exercise and recovery while breathing 12.6%  $O_2$ 

maximal treadmill exercise tests in ambient air and during exposure to a hypoxic gas mixture. Subjects ranged in age from 20-44 years and, with the exception of one individual, were recent sea level residents. Their physiological responses to exercise and hypoxia (other than te  $P_{O_2}$ ) will constitute a separate report.

The Clark skin probe was re-membraned prior to each day of testing and a sufficient time interval was allowed for electrode polarization and stabilization. A two-point calibration of the electrode was performed before and after each test using a glycerine/water solution and the probe was electronically zeroed. The probe was attached to the lower thorax in an area previously prepared by slightly abrading the skin. Cutaneous  $P_{O_2}$  monitoring was performed at an electrode temperature of 44° C. Approximately 10–15 min was required for electrode temperature and te  $P_{O_2}$  to stabilize.

After instrumentation, each subject rested for 15 min while control values were recorded. This was followed by a modified Balke maximal exercise test [1] (Fig. 1). This test has the subject starting her exercise at 0% grade and at a speed of 90 meters/min. The grade is increased progressively 1% each minute until exhaustion occurs, i.e. when the subject is unable to continue further work. Recovery measurements were recorded for an additional 15 min. On the following day, the maximal exercise tests were repeated while subjects inhaled 12.56% O<sub>2</sub> (balance N<sub>2</sub>) (Fig. 2). Control values were recorded before and after each test while subjects breathed room air.

#### Results

No discomfort from the transcutaneous  $P_{O_2}$  electrode was reported and direct visualization of the area under each probe failed to disclose evidence of blistering or irritation.

Figures 1 and 2 are representative records on a single female subject during maximal exercise and exercise in conjunction with hypoxemia. Mean resting tr  $P_{O_2}$  averaged 93.3 (± 5.4 SD) Torr for all subjects prior to the initial exercise run, and 84.7 (± 1.5 SD) prior to exposure to low ambient oxygen (day 2). Calibration settings were essentially the same on both days and the possibility that the initial exercise test may have residually influenced cutaneous blood flow and resistance during the subsequent evaluation cannot be discounted. However, input energy to the probe was the same at rest both days and laboratory ambient temperature was constant.

Transcutaneous  $P_{O_2}$  averaged 53 (± 4.7 SD) Torr at rest during inhalation of 12.6% O<sub>2</sub> (3,800 m equivalent). During hypoxic exercise tc  $P_{O_2}$  decreased to an average of 32 (± 2.3 SD) Torr during the last 10 min of exercise. Although arterial oxygen tensions of 32 Torr would result in arterial O<sub>2</sub> saturations as low as 60%, Hughes et al. [7] have reported O<sub>2</sub> saturations which ranged from 60-84% in male subjects performing progressive exercise during hypoxia at FI<sub>O2</sub> of 11%.

Initial response of the electrode to changes in inspired O<sub>2</sub> occurred within 10 s, and 95% of the maximum response was generally observed within 1–2 min (Fig. 2). Electrical stability of the probe was exceptional both at rest and during exercise. An average tc  $P_{O_2}$  drift of +4 Torr was noted post-exercise, but no drift was detected in half the recalibrations.

In Figure 2 input energy in milliwatts is plotted against tc  $P_{O_2}$  as a quantitative index of cutaneous blood flow. Since the probe is maintained at a constant temperature of 44° C, an increase in perfusion to the skin will necessitate an increase in energy output and vice versa [8]. A reciprocal relationship between tc  $P_{O_2}$ and relative cutaneous blood flow occurred in all subjects during exercise. In most instances the peak cutaneous flow (peak energy requirement) was observed just prior to exhaustion and occurred concomitantly with an elevation in tc  $P_{O_2}$  (Fig. 2). This suggests that relative skin blood flow in these subjects peaked simultaneously with, or just prior to, the attainment of  $\dot{V}_{O,max}$ .

## Discussion

It is generally agreed that to  $P_{O_2}$  is not the same as arterial  $P_{O_2}$ , but is a very close approximation [3,8]. Heating of the skin causes cutaneous oxygen con-

sumption to increase and should result in underestimation of arterial oxygen tension. However, this effect may be cancelled since increased skin and blood temperature elevates to  $P_{O_2}$  above  $Pa_{O_2}$  and causes a rightward shift of the oxygen-hemoglobin dissociation curve [8]. Since to  $P_{O_2}$  has been generally observed to slightly overestimate arterial oxygen tension [3, 6, 8], it has been suggested that a single comparison between arterial and to  $P_{O_2}$  at the onset of monitoring may be appropriate to provide a correction factor for subsequent recordings [3]. This may be valid for steady state monitoring but will require further evaluation under conditions imposed by stress.

Anticipatory hyperventilation in our six female subjects caused to  $P_{O_2}$  to increase by an average of 5 Torr at the onset of normoxic exercise. As work commenced transcutaneous oxygen tension gradually fell as oxygen uptake progressively increased. Oxygen tension (to  $P_{O_2}$ ) at max  $V_{O_2}$  was 87.8 (± 3.1 SD) Torr and this corresponded closely to the values reported by Holmér et al. [2] (86 Torr) and Vogel et al. [10] (88.6 Torr) obtained by direct arterial sampling in young men at maximal work. As a result of continuous monitoring, we confirmed the observations of these two groups of investigators as to the relative constancy of  $Pa_{O_2}$  at work loads of 40-65% of  $V_{O_2max}$ . At the onset of recovery te  $P_{O_2}$  overshot by 10-30 Torr as a consequence of persistent hyperventilation.

With our subjects sitting quietly and breathing 12.6 % O<sub>2</sub>, tc  $P_{O_2}$  averaged 53 (± 4.7 SD) Torr, which was expected for this level of hypoxia. Maximal aerobic capacity was reduced for all subjects by approximately 25 %, as predicted, and tc  $P_{O_2}$  decreased more rapidly and stabilized more quickly than during normoxic exercise. Although we were surprised by the low oxygen tensions, the consistency of tc  $P_{O_2}$  observed in our subjects provided a certain degree of assurance in the credibility of these values. Similar stability in  $Pa_{O_2}$  during exercise at 40, 65, and 100 % max  $V_{O_2}$  has been reported by Vogel et al. [10] in male sea level residents exercising on their 2nd and 10th day at altitude (3,800 m), although  $Pa_{O_2}$  was 8 Torr higher than we observed at maximum work. While we were unable to

obtain direct arterial samples to confirm these low transcutaneous oxygen tensions during hypoxic exercise, we intend to investigate this in the future.

Transcutaneous monitoring of arterial  $P_{O_2}$  appears to be a reliable alternative to direct arterial sampling at least in adult females, and has the added advantage of providing continuous monitoring of relative changes in blood oxygen tension and local cutaneous blood flow. On this basis, the technique warrants further consideration and evaluation in both clinical and physiological investigations.

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