

Can transcutaneous oxygen and carbon dioxide tensions reflect arterial levels during exercise testing?

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This lab study shows that transcutaneous values can closely follow changes in arterial blood gas by using an electrode temperature of 45 °C to monitor exercise with gradual workload.

Introduction

The measurement of arterial oxygen tension ($pO_2(a)$) and carbon dioxide tension ($pCO_2(a)$) are important and widely used tests in clinical patient monitoring and in the pulmonary function laboratory for assessing patients before and after exercise testing.

Arterial blood is obtained by puncture or from an indwelling cannula, but the measurements of $pO_2(a)$ and $pCO_2(a)$ in blood sampled intermittently have the serious disadvantage that critical changes may be missed, especially if a patient is assessed with an arterial sample obtained immediately post-exercise where the blood sampled may not reflect pulmonary gas exchange during exercise [1].

Arterial punctures are therefore impractical for continuous monitoring, while indwelling arterial cannulas may be uncomfortable and are potentially hazardous [2].

It would be advantageous, therefore, to have available a system which could monitor gas exchange in exercise without the potential morbidity associated with arterial cannulation.

The development of a combined transcutaneous sensor for the non-invasive, continuous measurement of oxygen tension ($tcpO_2$) and carbon dioxide tensions ($tcpCO_2$) at the skin surface has stimulated interest in the information that could be derived from the simultaneous monitoring of $pO_2(a)$ and $pCO_2(a)$ in the adult patient during exercise testing.

Several previous studies have demonstrated that the transcutaneous oxygen electrode provides a reliable method of continuously monitoring the changes in

arterial oxygen tensions of adults undergoing exercise testing [3, 4, 5], but differences were found in other studies [6].

Some of these differences can be accounted for by the different temperature settings used with the transcutaneous electrode (42-44 °C) producing a range of response times, or whether an *in vivo* calibration procedure was performed using a single arterial stab or earlobe capillary measurement prior to the exercise test.

More controversy surrounds the use of the transcutaneous carbon dioxide monitor in the monitoring of arterial carbon dioxide tensions during exercise testing. It has been shown that $tcpCO_2$ accurately reflects arterial.

$pCO_2(a)$ in hemodynamically stable patients at rest using an electrode temperature of 39-40 °C. At this temperature, however, there was a slow response time for the monitoring of changes which, in some cases, was up to three minutes, which would preclude its use in exercise testing [7].

Higher temperatures of the electrodes produce an increase in the temperature of capillary blood which leads to a systematically higher skin surface carbon dioxide tension than would be the case if measured at 37 °C.

It is now possible to incorporate a temperature correction factor based on the Siggaard-Andersen equation [8] to transcutaneous monitors, which allows the $tcpCO_2$ electrode to be used at a higher temperature with the $tcpCO_2$ reading corrected to 37 °C.

The use of the higher temperature has the advantage that it improves the response time of the $tcpCO_2$ electrode, so that more rapid changes in arterial carbon dioxide tensions can be followed.

Transcutaneous monitoring in the adult patient: a personal experience

It has previously been shown, in our own laboratory,

that the oxygen and carbon dioxide tension measured by the transcutaneous method correlates with the direct measurement of $pO_2(a)$ and $pCO_2(a)$ in hemodynamically stable patients at rest using an electrode temperature of 45 °C [9].

This confirms the findings of previous studies [10]. In 42 subjects, who were referred from a respiratory clinic for arterial blood sampling, the $pCO_2(a)$ ranged from 3.07 to 7.47 kPa. These values were compared to those obtained from a transcutaneous monitor following an *in vitro* calibration procedure using standard gas mixtures.

The site of attachment chosen for the electrode was the flexor surface of the forearm, which has a high capillary density and a relatively thin epidermis and is therefore a convenient site for measuring transcutaneous gases.

There was a significant correlation between $tcpCO_2$ (37 °C) and $pCO_2(a)$ derived from these 42 simultaneous measurements ($r=0.98$; $p<0.001$). The mean $tcpCO_2$ and $pCO_2(a)$ were 5.0 +/- 0.15 kPa and 5.05 +/- 0.15 kPa, respectively.

An analysis of Bland and Altman showed a mean difference of 0.02 kPa with no significant difference between the two measurements.

In the same group of patients the $pO_2(a)$ ranged from 6.8 to 14.0 kPa. Although there was a significant correlation between the two measurements ($r=0.91$; $p<0.001$), the transcutaneous level of pO_2 was generally lower than the simultaneous measurement of $pO_2(a)$.

There were, however, large inter-individual differences between the two measurements. The analysis of Bland and Altman showed a mean difference of 0.89 kPa. The mean $tcpO_2$ and $pO_2(a)$ were 9.3 +/- 0.25 kPa and 10.4 +/- 0.28 kPa, respectively.

Analysis of variance showed a significant difference between these two methods of measurement ($F=7.9$; $p=0.006$). This difference is due to a number of factors as described by Hutchinson *et al* [11].

The oxygen pressure recorded at the transcutaneous electrode will tend to be lower than the true arterial pO_2 because of oxygen consumption by the skin itself. Conversely, the temperature of the skin underlying the electrode is several degrees above that of normal blood.

This displaces the oxyhemoglobin dissociation curve to the right, producing an increase in the $pO_2(a)$ at a given hemoglobin saturation. Thus, the final $tcpO_2$ reflects a balance between these two factors.

While a good correlation between $tcpO_2$ and $pO_2(a)$ is evident from these results in adults, the scatter of results shows that the $tcpO_2$ cannot predict the $pO_2(a)$ with consistent accuracy when *in vitro* methods are used to calibrate the transcutaneous monitor.

The performance of an *in vivo* calibration using a sample of arterial or arterialized earlobe capillary blood has been shown to improve the accuracy of the $tcpO_2$ monitor by compensating for the difference between $pO_2(a)$ in individual patients [12].

The application of transcutaneous monitoring of pO_2 during cardiopulmonary exercise testing

The temperature of 45 °C used produces response times of the $tcpO_2$ and $tcpCO_2$ electrode to a change in breathing pattern or inspired gas of 30 and 55 seconds, respectively.

These response times are of an order which has allowed the investigation of the use of the combined electrode to non-invasively monitor arterial blood gases during exercise testing. We have therefore validated the transcutaneous monitor in 24 patients referred for progressive cardiopulmonary exercise testing against direct arterial sampling from an indwelling arterial cannula [13].

The group comprised patients with a range of cardiopulmonary disorders of varying degrees of severity, and also included patients with unexplained breathlessness. The transcutaneous electrode was firstly calibrated using standard gas mixtures.

The electrode was then attached to the skin and when a steady $tcpO_2$ and $tcpCO_2$ had been achieved (after about ten minutes), an arterial blood sample was obtained from a previously inserted arterial cannula, and pO_2 and pCO_2 measured. The gain controls of the device were then altered so that the output of the $tcpO_2$ and $tcpCO_2$ corresponded to the measured $pO_2(a)$ and $pCO_2(a)$ (*in vivo* calibration procedure).

A progressive exercise test was then performed using an electrically braked bicycle ergometer. The $tcpO_2$ and $tcpCO_2$ were monitored for two minutes while the patient was seated at rest.

An arterial blood sample was obtained at the midpoint of this period, and, taking into account the response time of the transcutaneous electrode, the transcutaneous monitor readings were recorded at the end of this period.

The patient was then asked to exercise for as long as possible until symptomatic limitation. During the first two minutes of exercise no additional load was applied, and an arterial blood sample was obtained at the midpoint of this period.

Transcutaneous values were recorded at the end of this period. Thereafter, the work load was increased by 10-25 watts, depending on the individual patient, every two minutes until symptomatic limitation.

Blood samples were taken at the midpoint and transcutaneous values taken at the end of each work load period. In these 24 patients 140 simultaneous measurements of arterial blood gases and transcutaneous values were obtained following the *in vivo* calibration. The range of arterial pO_2 measured was 6.1 to 14.2 kPa.

The analysis of Bland and Altman on the two pO_2 measurements showed a mean difference of 0.08 kPa. The mean $tcpO_2$ and $pO_2(a)$ were 11.5 +/- 0.35 kPa and 11.5 +/- 0.33 kPa, respectively. Analysis of variance shows that there is no significant difference between these measurements ($F=0.05$; $p=0.94$). The *in vivo* calibration procedure produces an even scatter of results

about the mean value with good limits of agreement suggesting that there is no difference between the measurement of $tcpO_2$ and $pO_2(a)$ on exercise testing.

The *in vivo* calibration procedure produced no adverse effects on the good relationship between $tcpCO_2$ and $pCO_2(a)$ already described. The $pCO_2(a)$ ranged from 3.6 to 7.6 kPa.

The analysis of Bland and Altman showed a mean difference of 0.02 kPa. The mean $tcpCO_2$ and $pCO_2(a)$ were 5.6 ± 0.2 and 5.6 ± 0.2 kPa, respectively, with no significant difference between the two measurements ($F=0.06$; $p=0.89$).

In all the exercise tests the trend of gas exchange as measured by the transcutaneous monitor was true to the trend as measured from direct arterial sampling. It might be suggested that the necessity of obtaining an arterial blood sample of arterialized earlobe capillary sample to perform the initial *in vivo* calibration would invalidate the non-invasive principle of the transcutaneous monitor.

However, a single arterial puncture or earlobe capillary sample carefully performed is a procedure with a very low complication rate, and once this has been completed, the usefulness of the transcutaneous monitor can be demonstrated.

Conclusion

The performance of an *in vivo* calibration procedure produces values of transcutaneous pO_2 and pCO_2 which are not significantly different from arterial blood gases during an exercise test.

It has been suggested that the slow response characteristics of transcutaneous monitors would render the use of a transcutaneous monitor unsuitable for exercise testing.

We have shown in our laboratory, however, that using the highest recommended temperature of 45 °C, producing a response time of 55 seconds for the CO_2

electrode, and combining this with an exercise protocol of gradual work load increments of two-minute periods to prevent abrupt and large changes in blood gas tensions allows changes in arterial blood gases to be closely followed by transcutaneous values.

The Respiratory Physiology Laboratory at Glasgow Royal Infirmary now routinely uses transcutaneous monitoring, after an *in vivo* calibration procedure using a single arterial stab or arterialized earlobe capillary sampling, to monitor changes in arterial blood gas values non-invasively during exercise testing.

When this is combined with mixed expired gas analysis this allows the calculation of sensitive indices of gas exchange including the alveolar-arterial oxygen gradient and the dead space/tidal volume ratio to be monitored throughout the exercise test.

The transcutaneous monitor has been tolerated without any associated morbidity in a wide range of patients who have been exercised in the laboratory [13, 14, 15, 16].

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