# Transcutaneous oxygen monitoring<sup>1</sup>

Herbert P. Wiedemann, M.D. Douglas K. Orens, R.R.T. Edward D. Sivak, M.D.

The technology to measure noninvasively the partial pressure of oxygen at the skin surface ("transcutaneous" PO<sub>2</sub>, or PtcO<sub>2</sub>) is now commercially available. In patients with normal cardiac output and cutaneous blood flow, the PtcO<sub>2</sub> accurately monitors changes in the arterial partial pressure of oxygen (PaO<sub>2</sub>). However, if the cardiac output is reduced, the PtcO<sub>2</sub> diverges from PaO<sub>2</sub> and reflects tissue oxygen delivery instead. Thus, the physiologic interpretation of the PtcO<sub>2</sub> varies according to the hemodynamic status of the patient. This limits the utility of transcutaneous oxygen monitoring in critically ill patients, but such monitoring can be useful in the noninvasive detection of adverse changes in arterial oxygenation or tissue perfusion.

Index terms: Blood gas analysis · Oximetry · Oxygen

Cleve Clin Q 52:483–488, Winter 1985

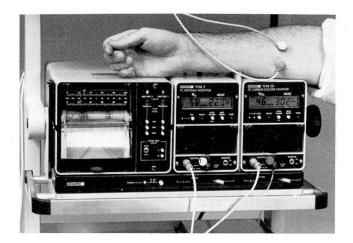
The arterial oxygen partial pressure (PaO<sub>2</sub>) traditionally is measured directly from a blood sample obtained through arterial puncture. In recent years, there has been considerable progress in the noninvasive monitoring of arterial oxygenation. <sup>1,2</sup> One commercially available technique permits the noninvasive measurement of the "transcutaneous" oxygen partial pressure (PtcO<sub>2</sub>) (Fig. 1).

Although transcutaneous oxygen monitoring has been proposed to be of value in several different clinical settings (Table), there remains some controversy regarding its usefulness, especially in the monitoring of critically ill adult patients with hemodynamic instability. <sup>14,15</sup> Initial expectations that the PtcO<sub>2</sub> would prove always to be a reliable indicator of the PaO<sub>2</sub> have not been fulfilled. Such an

Copyright © 1985, The Cleveland Clinic Foundation

<sup>&</sup>lt;sup>1</sup> Department of Pulmonary Disease, The Cleveland Clinic Foundation. Submitted for publication Sept 1985; accepted Sept 1985.

<sup>0009-8787/85/04/0483/06/\$2.50/0</sup> 



**Fig. 1.** The Radiometer transcutaneous monitor with  $PO_2$  and  $PCO_2$  sensors placed on the forearm. The strip chart recorder allows display of continuous measurements. For the purpose of this illustration, the sensors were not calibrated and therefore the measurements displayed do not reflect the true physiologic status.

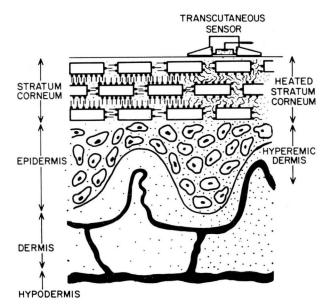
assumption is true when the circulation is normal, but under conditions of reduced cardiac output PtcO<sub>2</sub> diverges from PaO<sub>2</sub> and appears to reflect tissue oxygen delivery instead. The purpose of this article is to summarize the physiologic interpretation of the PtcO<sub>2</sub> measurement and to review the clinical experience that has been obtained with this technology.

## Historical development and theoretical considerations

The transport of oxygen across the skin barrier was first studied in 1851 by Gerlach, who noted that "cutaneous respiration depended on the quantity of blood streaming through the most superficial skin capillaries and its flow velocity." Exactly 100 years later, Baumberger and Goodfriend found that the PO<sub>2</sub> immediately surrounding a finger immersed in a 45° C electrolyte solution was close to the PaO<sub>2</sub>. This established the important concept that the PO<sub>2</sub> of heated

**Table.** Clinical settings in which transcutaneous oxygen monitoring may prove useful

- 1. Intensive care units<sup>3-5</sup>
- 2. Cardiopulmonary rescusitation<sup>6,7</sup>
- 3. Monitoring during anesthesia and surgery<sup>8,9</sup>
- 4. Exercise testing<sup>10–12</sup>
- 5. Sleep labs (apnea studies)
- 6. Evaluation of peripheral vascular disease<sup>13</sup>

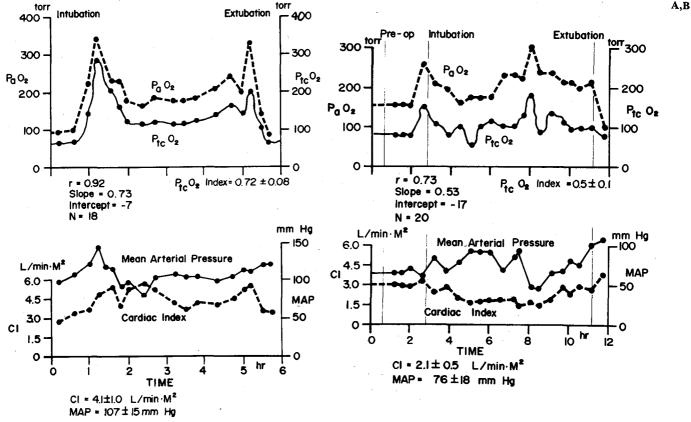


**Fig. 2.** Schematic cross-section of the transcutaneous sensor, the skin, and the dermal capillaries. Application of heat by the sensor alters the lipid structure of the stratum corneum and causes hyperemia of the dermis. These effects are important in aiding the diffusion of oxygen (represented by small dots) from the capillaries to the sensor membrane. (Reproduced with permission from Tremper et al.<sup>18</sup>)

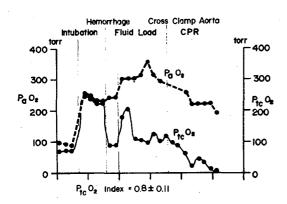
skin approached the PaO<sub>2</sub>. By 1972, a practical method of using a miniaturized and heated Clark electrode to measure the PO<sub>2</sub> at the skin surface was developed.<sup>17</sup> Almost immediately, the measurement of PtcO<sub>2</sub> gained widespread use in neonatal intensive care units, where the PtcO<sub>2</sub> was found to be nearly equal to PaO<sub>2</sub> in hemodynamically stable infants. This reduced the need for frequent arterial blood sampling.

The Clark electrode applies voltage between a platinum cathode and a silver anode in a KCl solution, thereby reducing oxygen. A current is produced that is proportional to the number of oxygen molecules reduced. Application of local heat is important for three reasons: (1) The oxygen-hemoglobin dissociation curve is shifted to the right; (2) The lipid structure of the stratum corneum is altered, allowing faster oxygen diffusion; and most importantly, (3) blood flow through the skin capillaries is greatly increased ("arterialization" of capillary blood). These effects increase the measured PtcO<sub>2</sub>, and allow this value to approximate PaO<sub>2</sub>. Figure 2 schematically illustrates the latter two effects of local heating.





C



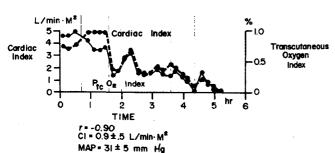


Fig. 3. The time course of PaO<sub>2</sub>, PtcO<sub>2</sub>, mean arterial pressure (MAP), and cardiac index (CI) in three separate patients is illustrated. Fig. 3A represents a hemodynamically stable patient monitored during surgery. The PtcO2 closely tracks changes in the PaO2, and the PtcO2 is about 72% of the corresponding PaO2 value. Fig. 3B shows data from a patient with moderate low-flow shock. At times PtcO2 diverges from PaO2 (although the correlation is significant), and the PtcO2 averages only 50% of the corresponding PaO2. Fig. 3C shows data from a patient who had intraoperative arrest due to an acute hemorrhage. PtcO2 is not correlated with PaO2, but both PtcO2 and the PtcO2 index track the cardiac index. (Reproduced with permission from Tremper et al.3)

### **Practical considerations**

A number of practical considerations affect the use of currently available transcutaneous oxygen monitors. The heat (43-45° C) necessary to increase cutaneous blood flow may induce local erythema or mild burns. Thus, electrodes should be moved approximately every 3-4 hours, especially in infants. After initial placement of the electrode, it may take 5–10 minutes for the PtcO<sub>2</sub> to reach equilibration. This obviously represents a major limitation to the use of transcutaneous monitoring during cardiopulmonary resuscitation or in other situations in which monitoring needs to be instituted quickly following an acute clinical decompensation. Conjunctival oxygen monitoring devices, recently approved by the FDA, may have a role in such settings since the initial equilibrium time is much shorter.<sup>7,19</sup> This is true in part because the absence of the stratum corneum layer in the conjunctivae makes local heating and hyperemia unnecessary for accurate monitoring. Conjunctival oxygen monitoring also has the advantage of a shorter response time (0-60 seconds versus 60-180 seconds for PtcO<sub>2</sub>) in detecting physiologic changes following initial equilibrium.7

There is some concern that anesthetic gases (particularly halothane and nitrous oxide) may interfere with PtcO<sub>2</sub> determinations, thus limiting the usefulness of such monitoring during surgery.<sup>20</sup> However, many of the studies on this issue have involved in vitro experiments in which anesthetic gas concentrations have been much higher than usual tissue concentrations. A recent report suggests that halothane interference may not be significant in patients actually undergoing anesthesia.<sup>21</sup> The sensor membrane (e.g., polypropylene versus Teflon) may prove important in minimizing anesthetic gas inerference. Questions remain on this subject, and further studies are necessary.

## Clinical experience with transcutaneous monitoring in the intensive care unit

A considerable amount of information about the clinical usefulness and interpetation of the PtcO<sub>2</sub> is now available. The largest amount of reported experience is that of Shoemaker, Tremper, and colleagues.<sup>3-6,8,15,22,23</sup> These investigators recently published data regarding the relationship of PtcO<sub>2</sub> to PaO<sub>2</sub> in critically ill adult patients in the intensive care unit.<sup>3</sup> They col-

lected 1073 sets of data from 106 patients. Patients were divided into three groups based upon the cardiac index (CI): Group 1 patients with relatively normal cardiac output (CI > 2.2); Group 2 patients with moderate shock (2.2 > CI)> 1.5); and Group 3 patients with severe shock (CI < 1.5). The  $PtcO_2$  was compared with the PaO<sub>2</sub>; the ratio of these values (PtcO<sub>2</sub>/PaO<sub>2</sub>) is the PtcO<sub>2</sub> "index". In patients with normal flow, the PtcO<sub>2</sub> index was  $0.79 \pm 0.12$  (SD) with r =0.89. In moderate shock, the PtcO2 index was  $0.48 \pm 0.07$  with r = 0.78. In severe shock, the  $PtcO_2$  was only  $0.12 \pm 0.12$  with r = 0.06 (no correlation). However, in these Group 3 patients, the PtcO<sub>2</sub> index had a significant correlation with cardiac index. Data from representative Group 1, 2, and 3 patients are illustrated in *Figure 3*.

The important conclusion from this study is that the relationship between the PtcO2 and the PaO<sub>2</sub> depends upon the cardiac output. At relatively normal cardiac outputs, the PtcO<sub>2</sub> is a reliable trend monitor of the PaO<sub>2</sub>, although the PtcO<sub>2</sub> will average only about 80% of the PaO<sub>2</sub> (a PtcO<sub>2</sub> value of 80 mmHg corresponds to a PaO<sub>2</sub> of 100 mmHg). However, at moderate levels of hypoperfusion, even when not associated with frank hypotension, the PtcO<sub>2</sub> averages only about 50% of the PaO<sub>2</sub>. This represents an important limitation of transcutaneous monitoring since such patients may not be easily distinguished from Group 1 patients without invasive monitoring of the cardiac output. And finally, in cardiogenic shock, changes in PtcO<sub>2</sub> actually reflect changes in the cardiac output (or tissue oxygen delivery), rather than in the PaO<sub>2</sub>.

Our experience with the transcutaneous oxygen monitor (Radiometer, see Fig. 1) in the medical intensive care unit parallels that obtained by other investigators. We have found that the  $PtcO_2$  is a reliable monitor of changes in the  $PaO_2$  in hemodynamically stable patients, although the actual  $PtcO_2$  index varies from patient to patient (Fig. 4).

#### Summary

Commercially available devices for the continuous and noninvasive monitoring of cutaneous (or conjunctival) oxygen pressures are now available. The physiologic interpretation of the PtcO<sub>2</sub> value appears well established. In patients with normal cardiac output and cutaneous blood flow, the PtcO<sub>2</sub> reflects PaO<sub>2</sub>. However, in low flow

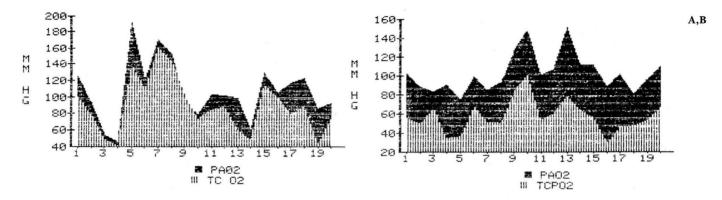


Fig. 4. This illustrates the comparison of 20 simultaneous PtcO<sub>2</sub> and PaO<sub>2</sub> values obtained during a 12-hour period in two patients monitored in the medical intensive care unit. Fig. 4A represents data from a patient with pancreatitis, diffuse pulmonary infiltrates, and metabolic acidosis. Fig. 4B shows data from a patient with chronic obstructive pulmonary disease and respiratory failure. Both patients were receiving mechanical ventilation; ventilatory parameters and inspired oxygen concentration were altered as clinical requirements dictated. Both patients were hemodynamically stable, with a mean arterial pressure greater than 90 mmHg during the monitoring period. Although PtcO<sub>2</sub> tracked changes in PaO<sub>2</sub> extremely well in both patients, the PtcO<sub>2</sub> index averaged .46 in the patient monitored in 4A and .83 in 4B. The cause of this difference between patients was not apparent.

states, the PtcO<sub>2</sub> diverges from PaO<sub>2</sub> and reflects tissue oxygen delivery instead. This represents both a problem and an opportunity. The problem is that monitoring of the PtcO<sub>2</sub> alone may be inadequate in many clinical situations, since decreasing PtcO<sub>2</sub> may reflect either pulmonary decompensation (decreasing PaO<sub>2</sub>) or hemodynamic failure (decreasing cardiac output). A separate, independent measurement of respiratory or cardiac function may be necessary to interpret the change in PtcO<sub>2</sub>. On the other hand, PtcO<sub>2</sub> can detect decreases in tissue oxygen delivery that are difficult to monitor directly (especially noninvasively) by any other technique currently available.

This technology can be of use in the contemporary critical care unit, assuming that those who use it are aware of the physiologic interpretation of the PtcO<sub>2</sub> value. The most exciting aspect of this technique is its potential in the assessment of oxygenation at the tissue level. 22-24 However, it may represent only an intermediate step in the evolution toward methods that are even more precise. Several emerging technologies (including microelectrode measurement of interstitial or intracellular PO<sub>2</sub> and pH, nuclear magnetic resonance, positron emission tomography, fluorometry and spectrophotometry, and purine nucleotide levels) may compete with transcutaneous monitoring. 24,25 The role that transcutaneous oxygen monitoring eventually may play in the

assessment of critically ill patients remains to be determined.

## Acknowledgment

We wish to thank the respiratory therapists for their help in evaluating the transcutaneous monitor in the medical intensive care unit.

#### References

- Wiedemann HP, Matthay MA, Matthay RA. Cardiovascularpulmonary monitoring in the intensive care unit (Part 2). Chest 1984; 85:656–668.
- Burki NK, Albert RK. Noninvasive monitoring of arterial blood gases: A report of the ACCP section on respiratory pathophysiology. Chest 1983; 83:666–670.
- Tremper KK, Shoemaker WC. Transcutneous oxygen monitoring of critically ill adults, with and without low flow shock. Crit Care Med 1981; 9:706–709.
- Tremper KK, Shoemaker WC, Shippy CR, Nolan LS. Transcutaneous PCO<sub>2</sub> monitoring on adult patients in the ICU and the operating room. Crit Care Med 1981; 9:752– 755.
- Tremper KK, Waxman K, Shoemaker WC. Use of transcutaneous oxygen sensors to titrate PEEP. Ann Surg 1981; 193:206–209.
- Tremper KK, Waxman K, Bowman R, Shoemaker WC. Continuous transcutaneous oxygen monitoring during respiratory failure, cardiac decompensation, cardiac arrest, and CPR. Crit Care Med 1980; 8:377–381.
- Abraham E, Smith M, Silver L. Conjunctival and transcutaneous oxygen monitoring during cardiac arrest and cardiopulmonary resuscitation. Crit Care Med 1984; 12:419–421.
- Shoemaker WC, Lawner PM. Method for continuous conjunctival oxygen monitoring during carotid artery surgery. Crit Care Med 1983; 11:946–947.

- Venus B, Patel KC, Pratap KS, Konchigeri H, Vidyasagar D. Transcutaneous PO<sub>2</sub> monitoring during pediatric surgery. Crit Care Med 1981; 9:714-716.
- Hughes JA, Gray BJ, Hutchison DC. Changes in transcutaneous oxygen tension during exercise in pulmonary emphysema. Thorax 1984; 39:424-431.
- McDowell JW, Thiede WH. Usefulness of the transcutaneous PO<sub>2</sub> monitor during exercise testing in adults. Chest 1980; 78:853–855.
- Schonfeld T, Sargent CW, Bautista D, et al. Transcutaneous oxygen monitoring during exercise stress testing. Am Rev Respir Dis 1980; 121:457-462.
- Hauser CJ, Shoemaker WC. Regional transcutaneous oximetry in noninvasive vascular diagnosis. Prac Card 1984; 10:153-165.
- Goldenheim PD, Kazemi H. Cardiopulmonary monitoring of critically ill patients. N Engl J Med 1984; 311:717-720, 776-780.
- Tremper KK, Shoemaker WC, Wender D. Monitoring oxygen transcutaneously in critically ill patients (letter). N Engl J Med 1985; 312:241.
- Lübbers DW. History of transcutaneous PO<sub>2</sub> measurement. Crit Care Med 1981; 9:693.
- Clark LC. Measurement of oxygen tension: a historical perspective. Crit Care Med 1981; 9:690-692.
- Tremper KK, Waxman K, Shoemaker WC. Effects of hypoxia and shock on transcutaneous PO<sub>2</sub> values in dogs. Crit Care Med 1979; 7:526-531.
- 19. Fatt I, Deutsch TA. The relation of conjunctival PO2 to capillary bed PO2. Crit Care Med 1983; 11:445-448.

- Eberhard P, Mindt W. Interference of anesthetic gases at skin surface sensors for oxygen and carbon dioxide. Crit Care Med 1981; 9:717-720.
- Samra SK. Halothane interference with transcutaneous oxygen monitoring: In vivo and in vitro. Crit Care Med 1983; 11:612-615.
- Fink S, Ray CW, McCartney S, Ehrlich H, Shoemaker WC. Oxygen transport and utilization in hyperoxia and hypoxia: Relation of conjunctival and transcutaneous oxygen tensions to hemodynamic and oxygen transport variables. Crit Care Med 1984; 12:943–948.
- 23. Shoemaker WC, Fink S, Ray CW, McCartney S. Effect of hemorrhagic shock on conjunctival and transcutaneous oxygen tensions in relation to hemodynamic and oxygen transport changes. Crit Care Med 1984; 12:949–952.
- Sugimoto H, Ohashi N, Sawada Y, Yoshioka T, Sugimoto T. Effects of positive end-expiratory pressure on tissue gas tensions and oxygen transport. Crit Care Med 1984; 12:661-663
- Dantzker DR, Gutierrez G. The assessment of tissue oxygenation. Resp Care 1985; 30:456-462.

Department of Pulmonary Disease The Cleveland Clinic Foundation 9500 Euclid Avenue Cleveland, Ohio 44106