

Transcutaneous oxygen monitoring¹

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The technology to measure noninvasively the partial pressure of oxygen at the skin surface ("transcutaneous" PO_2 , or $PtcO_2$) is now commercially available. In patients with normal cardiac output and cutaneous blood flow, the $PtcO_2$ accurately monitors changes in the arterial partial pressure of oxygen (PaO_2). However, if the cardiac output is reduced, the $PtcO_2$ diverges from PaO_2 and reflects tissue oxygen delivery instead. Thus, the physiologic interpretation of the $PtcO_2$ 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

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The arterial oxygen partial pressure (PaO_2) 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.^{1,2} One commercially available technique permits the noninvasive measurement of the "transcutaneous" oxygen partial pressure ($PtcO_2$) (*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.^{14,15} Initial expectations that the $PtcO_2$ would prove always to be a reliable indicator of the PaO_2 have not been fulfilled. Such an

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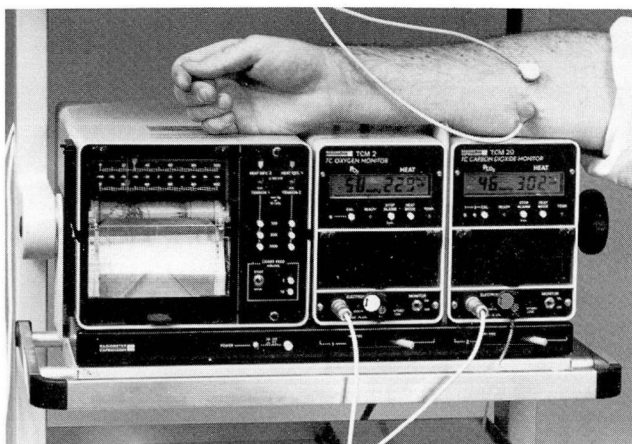


Fig. 1. The Radiometer transcutaneous monitor with PO_2 and PCO_2 sensors placed on the forearm. The strip chart recorder allows display of continuous measurements. For the purpose of this illustration, the sensors were not calibrated and therefore the measurements displayed do not reflect the true physiologic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

assumption is true when the circulation is normal, but under conditions of reduced cardiac output PtcO_2 diverges from PaO_2 and appears to reflect tissue oxygen delivery instead. The purpose of this article is to summarize the physiologic interpretation of the PtcO_2 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."¹⁶ Exactly 100 years later, Baumberger and Goodfriend found that the PO_2 immediately surrounding a finger immersed in a 45°C electrolyte solution was close to the PaO_2 . This established the important concept that the PO_2 of heated

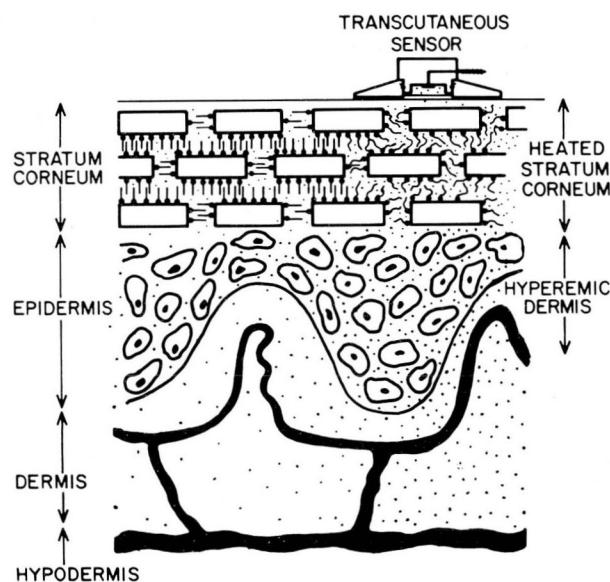


Fig. 2. Schematic cross-section of the transcutaneous sensor, the skin, and the dermal capillaries. Application of heat by the sensor alters the lipid structure of the stratum corneum and causes hyperemia of the dermis. These effects are important in aiding the diffusion of oxygen (represented by small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

skin approached the PaO_2 . By 1972, a practical method of using a miniaturized and heated Clark electrode to measure the PO_2 at the skin surface was developed.¹⁷ Almost immediately, the measurement of PtcO_2 gained widespread use in neonatal intensive care units, where the PtcO_2 was found to be nearly equal to PaO_2 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_2 , and allow this value to approximate PaO_2 . Figure 2 schematically illustrates the latter two effects of local heating.

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

1. Intensive care units ³⁻⁵
2. Cardiopulmonary resuscitation ^{6,7}
3. Monitoring during anesthesia and surgery ^{8,9}
4. Exercise testing ¹⁰⁻¹²
5. Sleep labs (apnea studies)
6. Evaluation of peripheral vascular disease ¹³

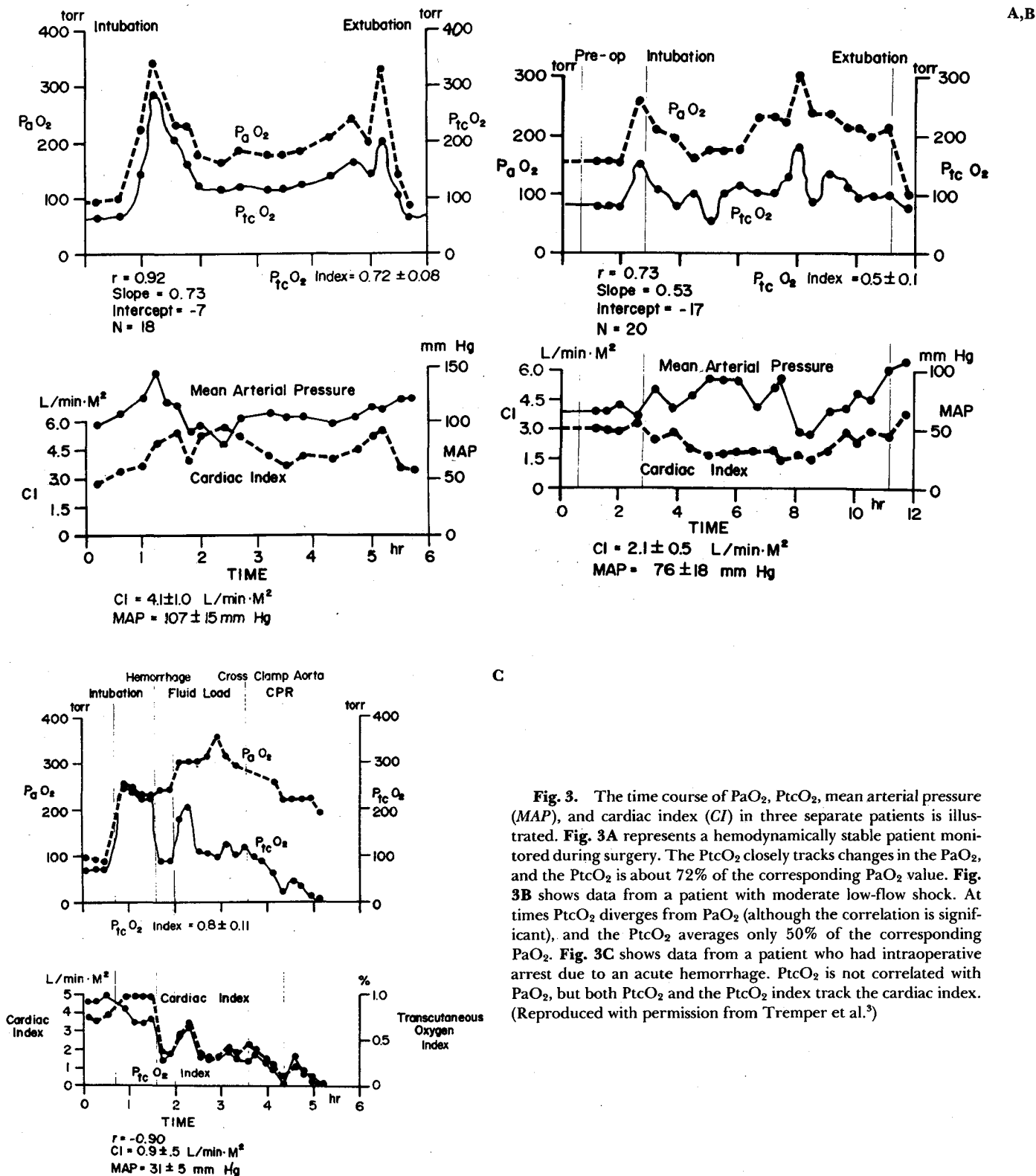


Fig. 3. The time course of P_{aO_2} , P_{tcO_2} , mean arterial pressure (MAP), and cardiac index (CI) in three separate patients is illustrated. **Fig. 3A** represents a hemodynamically stable patient monitored during surgery. The P_{tcO_2} closely tracks changes in the P_{aO_2} , and the P_{tcO_2} is about 72% of the corresponding P_{aO_2} value. **Fig. 3B** shows data from a patient with moderate low-flow shock. At times P_{tcO_2} diverges from P_{aO_2} (although the correlation is significant), and the P_{tcO_2} averages only 50% of the corresponding P_{aO_2} . **Fig. 3C** shows data from a patient who had intraoperative arrest due to an acute hemorrhage. P_{tcO_2} is not correlated with P_{aO_2} , but both P_{tcO_2} and the P_{tcO_2} 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 PtcO₂ 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.^{7,19} 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 PtcO₂) in detecting physiologic changes following initial equilibrium.⁷

There is some concern that anesthetic gases (particularly halothane and nitrous oxide) may interfere with PtcO₂ determinations, thus limiting the usefulness of such monitoring during surgery.²⁰ 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.²¹ The sensor membrane (e.g., polypropylene versus Teflon) may prove important in minimizing anesthetic gas interference. 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 interpretation of the PtcO₂ is now available. The largest amount of reported experience is that of Shoemaker, Tremper, and colleagues.^{3–6,8,15,22,23} These investigators recently published data regarding the relationship of PtcO₂ to PaO₂ in critically ill adult patients in the intensive care unit.³ 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 PtcO₂ was compared with the PaO₂; the ratio of these values (PtcO₂/PaO₂) is the PtcO₂ "index". In patients with normal flow, the PtcO₂ index was 0.79 ± 0.12 (SD) with $r = 0.89$. In moderate shock, the PtcO₂ index was 0.48 ± 0.07 with $r = 0.78$. In severe shock, the PtcO₂ was only 0.12 ± 0.12 with $r = 0.06$ (no correlation). However, in these Group 3 patients, the PtcO₂ 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 PaO₂ depends upon the cardiac output. At relatively normal cardiac outputs, the PtcO₂ is a reliable trend monitor of the PaO₂, although the PtcO₂ will average only about 80% of the PaO₂ (a PtcO₂ value of 80 mmHg corresponds to a PaO₂ of 100 mmHg). However, at moderate levels of hypoperfusion, even when not associated with frank hypotension, the PtcO₂ averages only about 50% of the PaO₂. 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 PtcO₂ actually reflect changes in the cardiac output (or tissue oxygen delivery), rather than in the PaO₂.

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 PtcO₂ is a reliable monitor of changes in the PaO₂ in hemodynamically stable patients, although the actual PtcO₂ 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 PtcO₂ value appears well established. In patients with normal cardiac output and cutaneous blood flow, the PtcO₂ reflects PaO₂. However, in low flow

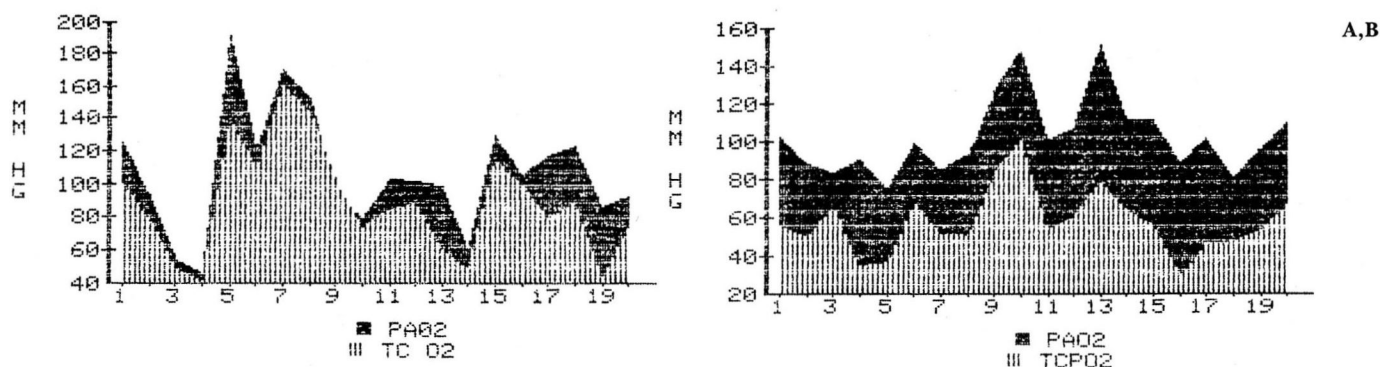


Fig. 4. This illustrates the comparison of 20 simultaneous PtcO₂ and PaO₂ values obtained during a 12-hour period in two patients monitored in the medical intensive care unit. **Fig. 4A** represents data from a patient with pancreatitis, diffuse pulmonary infiltrates, and metabolic acidosis. **Fig. 4B** 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 PtcO₂ tracked changes in PaO₂ extremely well in both patients, the PtcO₂ 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 PtcO₂ diverges from PaO₂ and reflects tissue oxygen delivery instead. This represents both a problem and an opportunity. The problem is that monitoring of the PtcO₂ alone may be inadequate in many clinical situations, since decreasing PtcO₂ may reflect either pulmonary decompensation (decreasing PaO₂) or hemodynamic failure (decreasing cardiac output). A separate, independent measurement of respiratory or cardiac function may be necessary to interpret the change in PtcO₂. On the other hand, PtcO₂ 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 PtcO₂ 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 PO₂ and pH, nuclear magnetic resonance, positron emission tomography, fluorometry and spectrophotometry, and purine nucleotide levels)^{24,25} may compete with transcutaneous monitoring. The role that transcutaneous oxygen monitoring eventually may play in the

assessment of critically ill patients remains to be determined.

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References

1. Wiedemann HP, Matthay MA, Matthay RA. Cardiovascular-pulmonary monitoring in the intensive care unit (Part 2). *Chest* 1984; **85**:656-668.
2. Burki NK, Albert RK. Noninvasive monitoring of arterial blood gases: A report of the ACCP section on respiratory pathophysiology. *Chest* 1983; **83**:666-670.
3. Tremper KK, Shoemaker WC. Transcutaneous oxygen monitoring of critically ill adults, with and without low flow shock. *Crit Care Med* 1981; **9**:706-709.
4. Tremper KK, Shoemaker WC, Shippy CR, Nolan LS. Transcutaneous PCO₂ monitoring on adult patients in the ICU and the operating room. *Crit Care Med* 1981; **9**:752-755.
5. Tremper KK, Waxman K, Shoemaker WC. Use of transcutaneous oxygen sensors to titrate PEEP. *Ann Surg* 1981; **193**:206-209.
6. 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.
7. Abraham E, Smith M, Silver L. Conjunctival and transcutaneous oxygen monitoring during cardiac arrest and cardiopulmonary resuscitation. *Crit Care Med* 1984; **12**:419-421.
8. Shoemaker WC, Lawner PM. Method for continuous conjunctival oxygen monitoring during carotid artery surgery. *Crit Care Med* 1983; **11**:946-947.

9. Venus B, Patel KC, Pratap KS, Konchigeri H, Vidyasagar D. Transcutaneous PO₂ monitoring during pediatric surgery. *Crit Care Med* 1981; **9**:714-716.
10. Hughes JA, Gray BJ, Hutchison DC. Changes in transcutaneous oxygen tension during exercise in pulmonary emphysema. *Thorax* 1984; **39**:424-431.
11. McDowell JW, Thiede WH. Usefulness of the transcutaneous PO₂ monitor during exercise testing in adults. *Chest* 1980; **78**:853-855.
12. Schonfeld T, Sargent CW, Bautista D, et al. Transcutaneous oxygen monitoring during exercise stress testing. *Am Rev Respir Dis* 1980; **121**:457-462.
13. Hauser CJ, Shoemaker WC. Regional transcutaneous oximetry in noninvasive vascular diagnosis. *Prac Card* 1984; **10**:153-165.
14. Goldenheim PD, Kazemi H. Cardiopulmonary monitoring of critically ill patients. *N Engl J Med* 1984; **311**:717-720, 776-780.
15. Tremper KK, Shoemaker WC, Wender D. Monitoring oxygen transcutaneously in critically ill patients (letter). *N Engl J Med* 1985; **312**:241.
16. Lübbers DW. History of transcutaneous PO₂ measurement. *Crit Care Med* 1981; **9**:693.
17. Clark LC. Measurement of oxygen tension: a historical perspective. *Crit Care Med* 1981; **9**:690-692.
18. Tremper KK, Waxman K, Shoemaker WC. Effects of hypoxia and shock on transcutaneous PO₂ 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.
20. Eberhard P, Mindt W. Interference of anesthetic gases at skin surface sensors for oxygen and carbon dioxide. *Crit Care Med* 1981; **9**:717-720.
21. Samra SK. Halothane interference with transcutaneous oxygen monitoring: In vivo and in vitro. *Crit Care Med* 1983; **11**:612-615.
22. 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.
24. 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.
25. Dantzker DR, Gutierrez G. The assessment of tissue oxygenation. *Resp Care* 1985; **30**:456-462.

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