Use of Transcutaneous Oxygen Sensors to Titrate PEEP

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The relationship of transcutaneous oxygen tension ($P_{tc}O_2$) to arterial oxygen tension (Pa_{O_2}), pulmonary shunt ($\dot{Q}sp/\dot{Q}t$), mixed venous oxygen tension ($P\bar{v}_{O_2}$), and O_2 delivery was determined in patients with respiratory failure in order to explore the possible usefulness of $P_{tc}O_2$ to titrate the level of positive end expiratory pressure (PEEP). Transcutaneous oxygen sensors were applied to the chest of surgical ICU adult patients who were in acute postoperative respiratory failure. The patients had mechanical ventilation with volume ventilators and an intermittent mandatory ventilation (IMV) rate, which allowed normal pH and arterial CO_2 tension ventilation (Pa_{CO_2}). Swan-Ganz and arterial catheters were inserted. The blood volume was measured by iodinated I-125-serum albumin and brought into the normal range, before the study began, with appropriate volume therapy. Serial cardiorespiratory data were taken before and after PEEP was increased from zero to 20 cm H_2O, in 5 cm increments. $P_{tc}O_2$ correlated well with Pa_{O_2} and $P\bar{v}_{O_2}$; it was inversely correlated with $\dot{Q}sp/\dot{Q}t$. $P_{tc}O_2$ correlated with O_2 delivery in only seven severely ill patients mean alveolar-arterial oxygen tension difference [$A\text{-}aD_{O_2}$] was 380 mmHg and the pulmonary shunt was 37%). For the eight other patients, variations in the greatly elevated cardiac output associated with hypoxemia led to poor correlations between $P_{tc}O_2$ and O_2 delivery. There was no significant depression of cardiac output in any of the studies. We conclude that the continuous noninvasive nature of $P_{tc}O_2$ monitoring greatly increased the safety and simplicity of PEEP optimization and respiratory management of adult patients with respiratory failure.

SINCE ITS INTRODUCTION in 1972, the transcutaneous oxygen sensor has been successful in predicting arterial oxygen tension (Pa_{O_2}) in neonates with respiratory distress.[1–5] Until recently, it was thought that these sensors were of little value in monitoring adult patients with respiratory failure, because the sensors could not predict Pa_{O_2} consistently.[6–8] Because these sensors measure oxygen that diffuses through the skin, they primarily reflect skin tissue P_{O_2} rather than Pa_{O_2}. Factors influencing the oxygen delivery to the tissue may affect the measured value. Therefore, the transcutaneous oxygen tension ($P_{tc}O_2$) might be affected by cardiac output as well as Pa_{O_2}. Experimental animal studies have shown that $P_{tc}O_2$ follows Pa_{O_2} during hypoxia and cardiac output during shock, and it reflects oxygen delivery during either insult.[9–11] In a recent study, preterminal adult patients were monitored continuously with a $P_{tc}O_2$ sensor, and intermittently with arterial oxygen and cardiac output measurements, before and during cardiopulmonary resuscitation (CPR).[12] This study confirmed that $P_{tc}O_2$ correlates with oxygen delivery during shock and hypoxia in adults.

Various workers[13–15] have advocated that an important goal of ventilation management is attainment of the level of PEEP that would minimize pulmonary shunt ($\dot{Q}sp/\dot{Q}t$) and maximize the Pa_{O_2}, the mixed venous oxygen tension ($P\bar{v}_{O_2}$), and the oxygen delivery. This may also coincide with the maximal pulmonary compliance.[14] In essence, a major therapeutic goal is to determine the PEEP that will increase the functional residual capacity maximally without reducing cardiac output and O_2 delivery.

In the present study, we assessed the usefulness of continuous $P_{tc}O_2$ monitoring while optimizing PEEP, by comparing $P_{tc}O_2$ to Pa_{O_2}, $\dot{Q}sp/\dot{Q}t$, $P\bar{v}_{O_2}$, and oxygen delivery.

Materials and Methods

The subjects for this study were critically ill, postoperative, ICU patients with varying degrees of pulmonary failure. All patients were connected to volume ventilators, and had Swan-Ganz and arterial catheters. The ventilators were set on an IMV rate which kept the patients' pH and P_{CO_2} levels within normal limits.

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Physiologic Measurement

Each data set included heart rate, systolic, diastolic, pulmonary arterial, wedge and central venous pressures, central blood temperature, cardiac output, FI_{O_2}, arterial and mixed venous blood gases, and $P_{tc}O_2$. Cardiac output was determined, in duplicate or triplicate, by the thermal dilution technique (Edwards Laboratories, Santa Ana, CA). The blood gas samples were obtained anaerobically, and promptly analyzed with a Corning® Model 165 pH/blood gas analyzer.

The transcutaneous electrode was placed on the shoulder or anterior chest. The patient's skin was cleaned with alcohol and dried to ensure good adhesion of the adhesive O-ring. The electrode was attached with a contact gel interface, as recommended by the manufacturers. Three types of electrodes were used: TCOM Transcutaneous Blood Gas Monitor (Novametrix Medical Systems, Inc., Wallingford, CT), Sensomat III (Biochem International, Milwaukee, WI), and TCMI TCI Oxygen Monitor (Radiometer Corporation, Copenhagen, Denmark). The electrode temperature was set at 44–45 C. A two-point gas calibration was used.

Prior to each study, the patient's blood volume was measured with iodinated I-125-serum albumin. If the indexed volume was less than normal (2.37 L/m² in females and 2.74 L/m² in males), the patient was given a volume load of 5% plasma protein fraction, if the hematocrit was greater than 32%, or whole blood if the hematocrit was 32% of less.[15]

Protocol

In the control period, two sets of baseline data were taken on zero PEEP. The PEEP was increased in increments of 5 cm H_2O to a maximum of 20 cm H_2O, with two sets of data taken at each level. In severe adult respiratory distress syndrome (ARDS) patients, higher PEEP pressures were used. Twenty minutes of stabilization was allowed after each change in PEEP. If the cardiac output was depressed by more than 15%, the mixed venous oxygen was checked. If the mixed venous oxygen was not depressed, the conditions were held constant for another 20 minutes, and the measurements repeated. If the values were similar, PEEP was increased. If the mixed venous oxygen was depressed and if the wedge pressure was less than 14 mmHg, a volume load of 500 ml of 5% plasma protein fraction was given and the data set was then repeated. If the wedge pressure was greater than 15 mmHg, the PEEP was reduced and the study was ended. If at any time during the study, the $P_{tc}O_2$ dropped by 15%, a data set was taken and the electrode calibration was checked. All patients were left on optimal PEEP, as determined by the maximal $P\bar{v}_{O_2}$, O_2 delivery and the minimum $\dot{Q}sp/\dot{Q}t$. A similar protocol has been used to titrate the preferred or "optimal" values of PEEP prior to the availability of transcutaneous sensors.[15].

TABLE 1. *Correlation Coefficients Between $P_{tc}O_2$ and Pa_{O_2}, $\dot{Q}sp/\dot{Q}t$, $P\bar{v}_{O_2}$, and O_2 Delivery*

	Pa_{O_2}	Qsp/Qt*	$P\bar{v}_{O_2}$	O_2 Delivery
1	.99	−.70	.96	.98
2	.88	−.97	.81	.20
3	.93	−.92	.96	.58
4	.99	−.72	.85	.50
5	.85	−.70	.87	.81
6	.96	−.90	.88	.96
7	.95	−.67	.98	.91
8	.91	−.93	.70	.70
9	.99	−.99	.97	.77
10	.96	−.97	.94	.10
11	.91	−.90	.90	.79
12	.97	−.96	.97	.98
13	.87	−.90	.96	.33
14	.99	−.99	.92	.20
15	.99	−.75	.95	.43
r_w	0.93	−0.89	0.88	0.60

* Values at PEEP of zero.

Results

Fifteen PEEP studies were performed on 11 patients. Eight of the 11 patients eventually died. The mean alveolar-arterial oxygen tension difference ($A\text{-}aD_{O_2}$) and $\dot{Q}sp/\dot{Q}t$ on zero PEEP were 387 ± 149 (SD) mmHg and 34 ± 8 (SD)%, respectively. $P_{tc}O_2$ was compared with Pa_{O_2}, $\dot{Q}sp/\dot{Q}t$ $P\bar{v}_{O_2}$, and O_2 delivery. The correlation coefficients are shown in Table 1. $P_{tc}O_2$ correlated well with Pa_{O_2}, $\dot{Q}st/\dot{Q}t$ and $P\bar{v}_{O_2}$ in all 15 PEEP studies; weighted mean correlation coefficients being 0.93, −0.89 and 0.88, respectively. $P_{tc}O_2$ correlated well with O_2 delivery in only seven of the severely ill patients. In only two studies did PEEP produce a greater than 15% depression of cardiac output; in neither case was the $P\bar{v}_{O_2}$ affected or the cardiac output depressed below normal limits. Figure 1 illustrates a representative PEEP study. Note in Figure 1 as PEEP was increased in increments of 5 cm of water pressure, the cardiac output was not depressed (lower graph), the $P_{tc}O_2$ and O_2 delivery rose (upper graph); the Pa_{O_2} and $P\bar{v}_{O_2}$ rose, and the $\dot{Q}sp/\dot{Q}t$ dropped (middle graph).

Discussion

The transcutaneous oxygen sensor measures oxygen that diffuses through the skin (Fig. 2) by a modified

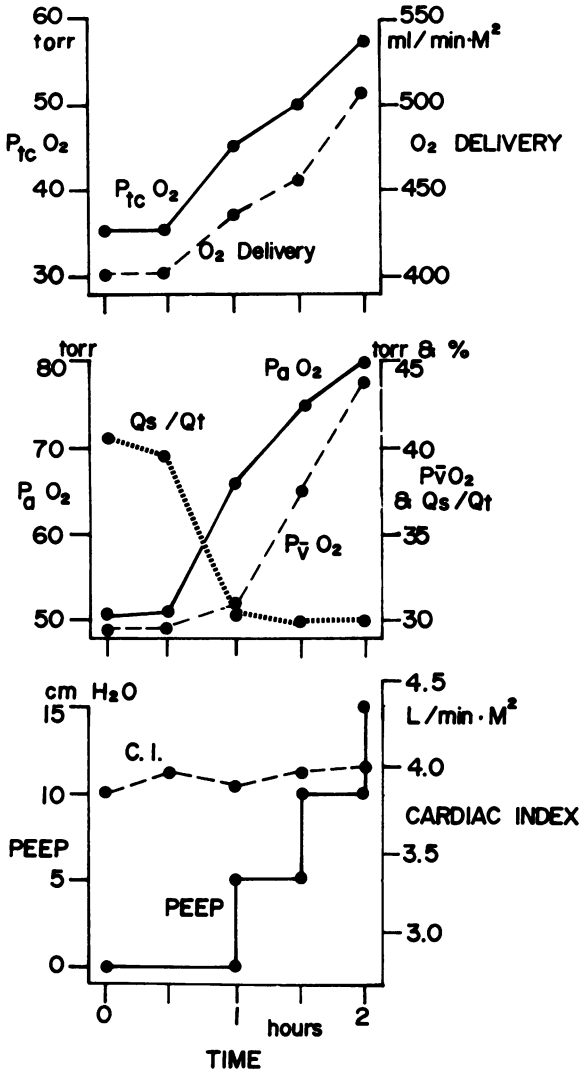

FIG. 1. PEEP study parameters during the course of a representative study. Note: $P_{tc}O_2$, O_2 delivery, Pa_{O_2}, and $P\bar{v}_{O_2}$ all rose with increasing PEEP. Pulmonary shunt fell as PEEP was increased while cardiac index was stable.

Clark polarographic electrode.[1,2] The skin must be heated to obtain a measurable oxygen concentration on the surface. Heating causes three effects: a) it changes the lipid structure of the stratum corneum allowing oxygen to diffuse faster; b) the tissue and blood beneath the electrode are also heated, slightly decreasing the oxygen solubility and shifting the oxygen–hemoglobin dissociation curve to the right, and c) heat dilates the local capillaries, and arterializes the capillary blood.[10,16,17] The last effect may not occur when there is hemodynamic instability.[9–12] Heating the electrode increases the $P_{tc}O_2$ and compensates, to some degree, for the two transport gradients, *i.e.*, the O_2 diffusion gradient from the capillary to the consuming electrode and the gradient caused by the oxygen metabolism of the dermal and epidermal cells (Fig. 2).[12]

It has been proposed that PEEP is optimized in the treatment of ARDS by maximizing Pa_{O_2}, $P\bar{v}_{O_2}$ and O_2 delivery, while minimizing $\dot{Q}sp/\dot{Q}t$.[13–15] This involves the placement of a Swan-Ganz catheter, and taking multiple measurements of cardiac output, with simultaneous arterial and mixed venous blood gas analyses between each adjustment in PEEP. To help simplify this expensive, invasive, laborious procedure, the static compliance was offered as a non-invasive way of finding the "optimal" PEEP.[14] Unfortunately, the point of maximal static compliance does not always coincide with the optimal PEEP, as described by the oxygen transport variables.[18–19]

No significant depression of cardiac output was observed in any of these studies. Reduced cardiac output after PEEP may occur with relative hypovolemia. The fact that all patients in this study were normalized or volume loaded before each study probably accounts for the absence of diminished cardiac output. From the work on adults during shock and CPR, the $P_{tc}O_2$ value was found to correlate well with Pa_{O_2} until the cardiac index fell below 1.9 L/min·M², at which point $P_{tc}O_2$ became linear with the cardiac index. This was also observed in shock studies in experimental animals.[9–11] This result implies that $P_{tc}O_2$ would rise with increasing Pa_{O_2} as PEEP was increased. This is verified by the

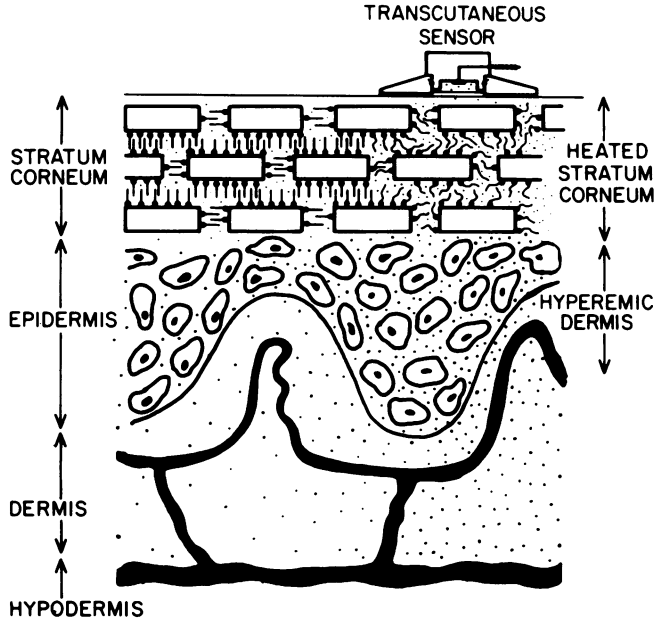

FIG. 2. Schematic cross-section of the electrode and skin: stratum corneum, epidermis, dermis with dermal capillaries, and hypodermis. The irregular structure of the stratum corneum beneath the electrode represents the melted lipid component. The small dots represent oxygen. From Tremper et al.[12]

high degree of correlation between $P_{tc}O_2$ and Pa_{O_2} in Table 1, and illustrated in Figure 1. Because there was no significant depression in cardiac output or change in total body oxygen consumption in any of the studies, $P_{tc}O_2$ also correlated well with $P\bar{v}_{O_2}$ and $\dot{Q}sp/\dot{Q}t$ (Table 1). If there had been a significant depression of flow with increased PEEP, we would expect a drop in $P\bar{v}_{O_2}$ and O_2 delivery. The $P_{tc}O_2$ would also drop, but only if the depression in flow had been severe, cardiac index (CI) 1.9 L/min·M².[12] This magnitude of decreased CI was not obtained in any of the studies. There was a decrease in CI by greater than 15% in only two studies, and in neither study was the CI below the normal range nor were there decreased $P\bar{v}_{O_2}$ values.

$P_{tc}O_2$ correlated well with O_2 delivery in only seven severely ill patients. In the other eight studies, the correlation coefficients were less than 0.7. We attribute the poor correlation to the random variation of CI which directly affected the O_2 delivery, but since the CI was always greater than 1.9 L/min·M², it had no effect on the $P_{tc}O_2$. This does somewhat limit the usefulness of $P_{tc}O_2$ in PEEP optimization, but the sensor is a significant aid in simplifying a PEEP optimization study.

In clinical practice, a safe fast PEEP optimization study can be achieved without the use of multiple invasive blood gas samples. As PEEP is increased, the $P_{tc}O_2$ will follow the increasing Pa_{O_2} and identify when equilibration at the new PEEP level is reached. Any acute pulmonary or ventilatory problem (pneumothorax or ventilator malfunction) will be identified with a 30-second response time. Intermittent cardiac output measurements should be taken, to ensure that flow is not being depressed. Reduction of FI_{O_2} can also be simplified without the necessity of multiple invasive arterial gas samples.

It should be noted that with adult patients, the $P_{tc}O_2$ value is approximately 70% of the Pa_{O_2} value, but there is significant patient to patient variation. There are also depressed $P_{tc}O_2$ values on the extremities of patients with peripheral vascular disease.[20] But as demonstrated in Table 1, the relationship between Pa_{O_2} and $P_{tc}O_2$ is almost linear if the CI is greater than 2 L/min·M².

We conclude that $P_{tc}O_2$ is a significant advance in adult respiratory management. This continuous noninvasive oxygen monitoring technique can greatly simplify PEEP optimization and reduction of FI_{O_2}. In addition, $P_{tc}O_2$ monitoring improves the quality of respiratory management by giving an early warning of pulmonary decompensation.

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