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Hyperventilation Reduces Transcutaneous Oxygen Tension and Skin Blood Flow

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Transcutaneous oxygen tension (Ptc_{O_2}) is often used to monitor neonates and infants in special care units and the operating room. The transcutaneous index (TCI = Ptc_{O_2}/arterial oxygen tension [Pa_{O_2}]) is known to depend both on age and on cardiac index but is assumed to be independent of other physiologic variables. In this study we have shown that TCI also depends upon arterial carbon dioxide tension ($PaCO_2$).

Five young pigs were anesthetized and paralyzed and their lungs mechanically ventilated while they were monitored with Ptc_{O_2} electrodes and serial arterial blood gas analyses. For a 45° C Ptc_{O_2} sensor, the mean TCI during normocapnia was 0.78, whereas during hyperventilation ($PaCO_2$ = 20 mmHg) the mean TCI was reduced 65%, to 0.27. The corresponding TCI values for a 43° C sensor were 0.33 and 0.065, representing an 80% decrease in TCI during hyperventilation. Hypoventilation had little effect upon TCI as long as hypoxemia was avoided. Twelve awake adult volunteers with radial artery cannulas were monitored with Ptc_{O_2} sensors at several body sites and two sensor temperatures. For a 44° C sensor on the chest, the mean TCI decreased from 0.77 at normocapnia to 0.60 at a Pa_{CO_2} of 17 mmHg, a 22% change. For the same sensor on the foot, TCI decreased from 0.63 to 0.32, a 49% change. For a 42° C sensor under the same conditions, the corresponding TCI decreases were 51 and 64%. Six of the volunteers were also monitored with laser-Doppler skin blood flow probes located on the chest, hand, and foot. The skin blood flow measured on the chest decreased by an average of 8% during hyperventilation; blood flow on the hand (thenar eminence) decreased by 60%; and blood flow on the foot decreased by 51%. The TCI is significantly reduced in both swine and adult humans during hyperventilation. This effect is greater on the extremities and at lower sensor temperatures. Laser-Doppler velocimetry measurements indicate a corresponding reduction in cutaneous blood flow during hyperventilation. Transcutaneous oxygen should be used with caution as a predictor of Pa_{O_2} during spontaneous or controlled hyperventilation. (Key words: Carbon dioxide: hypocapnia. Oxygen: measurement technique; transcutaneous; perfusion.)

THE MEASUREMENT of transcutaneous oxygen tension (Ptc_{O_2}) was introduced in 1972, and the technique gained rapid acceptance as a noninvasive estimate of arterial oxygen tension (PaO_2) in the neonatal intensive care unit.[1–3] In patients suffering from respiratory distress syndrome, Ptc_{O_2} monitoring reduces the required number of arterial blood gas samples and provides continuous oxygenation data. The near equivalence of neonatal Ptc_{O_2} values and Pa_{O_2} values is a fortunate coincidence. Diffusion gradients and metabolism in the skin tend to decrease Ptc_{O_2}, whereas heat-induced hyperemia and rightward shift of the hemoglobin dissociation curve tend to increase Ptc_{O_2}. These competing effects usually cancel out in neonates, but not in other age groups. The transcutaneous index (TCI), defined as Ptc_{O_2} divided by Pa_{O_2}, decreases with increasing age to a normal value of 0.8 in healthy adults.[4]

Investigators in the late 1970s found that Ptc_{O_2} values were much less than Pa_{O_2} in patients in shock.[5,6] This led to the additional discovery that Ptc_{O_2} values tend to reflect changes in cardiac output when the latter is less than normal. Tremper *et al.* collected over 1,000 data sets in 106 adult intensive-care-unit patients whose cardiac index spanned a wide range.[7] Patients in the study were divided into three groups. For those having a cardiac index greater than 3.0, the mean value ± standard deviation of TCI was 0.79 ± 0.12. In patients whose cardiac index was between 1.5 and 2.2, the mean TCI was 0.48 ± 0.07. Finally, those in cardiogenic shock had cardiac index values less than 1.5, and their mean TCI was 0.12 ± 0.12. Animal studies combining hypoxemia with hemorrhagic shock also demonstrated flow dependence of Ptc_{O_2}.[8] These studies showed that Ptc_{O_2} is a measure of oxygen delivery to tissue, and as such it reflects decreases in either Pa_{O_2} or cardiac output.

Because of its dependence on local skin perfusion as well as on Pa_{O_2}, the interpretation of Ptc_{O_2} is sometimes difficult. Despite this and other practical limitations of the technique, Ptc_{O_2} remains a commonly used monitor of oxygenation in the neonatal intensive care unit. It is also often used to monitor neonates and infants during anesthesia and surgery. Although the TCI is known to be a function of both age and cardiac output, it has not been reported to depend upon any other physiologic variables. During a recent animal study of transcutaneous oxygen during hypothermia, we discovered that the TCI may also be influenced by the arterial carbon dioxide tension ($PaCO_2$).§ In the present study, we investigate and characterize this effect in both animals and humans.

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Materials and Methods

Animal Study

Five healthy pigs aged 3–6 weeks and weighing 9–14 kg were studied after approval by the University Animal Research Committee had been obtained. Young swine were selected because their TCI is similar to that of young adult humans.¶ Anesthesia was induced in each animal with intramuscular ketamine 10 mg/kg, followed by tracheal intubation. An intravenous cannula was inserted and a continuous infusion of pentobarbital, 10–15 $mg \cdot kg^{-1} \cdot h^{-1}$, was given for anesthetic maintenance. The animals were paralyzed with pancuronium 0.1 $mg \cdot kg^{-1} \cdot h^{-1}$ to prevent spontaneous respiration. After an inguinal dissection to expose the femoral artery and vein, a 20-G femoral artery cannula was inserted for blood sampling and pressure measurement. A 7.5-Fr thermodilution pulmonary artery catheter was inserted *via* the femoral vein for monitoring filling pressures and cardiac output. Five Ptc_{O_2} electrodes (model 807, Novametrix) were applied to shaved skin on the chest. These electrodes were maintained at temperatures from 43 to 45°C. Additional monitoring included electrocardiogram and capnometry.

Data were recorded only during steady-state conditions at inspired oxygen fraction (FI_{O_2}) values of 1.0 and 0.3. At the beginning of each experiment and after each change in FI_{O_2}, the animals' lungs were ventilated at normocapnia ($PaCO_2 = 36 \pm 2$ mmHg) for 30 min prior to recording data. Each data set consisted of Ptc_{O_2} values from all five electrodes, hemodynamic data (blood pressure, pulmonary artery wedge pressure, and cardiac output), and an *in vitro* arterial blood gas analysis determined by standardized electrodes (Radiometer ABL-2 blood gas analyzer). At each FI_{O_2} value for each animal, the respiratory rate was varied stepwise to achieve six values of Pa_{CO_2} ranging from 18 to 55 mmHg. Each steady Pa_{CO_2} value was maintained for 15 min before data were recorded. The initial Pa_{CO_2} was repeated at the end of each series. Pulmonary artery wedge pressures were maintained at or within 3 mmHg above baseline values. The cardiac index was greater than 3.0 $l \cdot min^{-1} \cdot m^{-2}$ throughout the study. No inotropic or vasoactive drugs were used during the experiment.

The statistical significance of reported changes in the TCI was assessed by analysis of variance (ANOVA), followed by the Dunnett *t* test for intergroup comparisons. In comparing mean TCI values for several different groups (different Pa_{CO_2} values), ANOVA yields a *P* value or probability that the "true" mean TCI values for all of the groups are actually the same. To compare two specific groups within the set for statistical significance, we must follow the ANOVA calculation with an appropriate *post hoc* test for intergroup differences. In this case, we wish to compare the mean TCI of a "control" group (*i.e.*, the normocapnic pigs) to the TCI values of each of several hypocapnic groups. The most appropriate or "robust" test for such a comparison is the Dunnett *t* test, which yields a *P* value for the null hypothesis of each intergroup comparison.[9]

Human Volunteer Studies

Twelve healthy volunteers, aged 23–38 yr, participated in this study, which was approved by the University Human Subjects Review Committee. After a normal Allen's test of collateral circulation, each subject had a 20-G cannula inserted into the radial artery of the nondominant hand. Subjects were monitored by electrocardiogram, pulse oximeter, mass spectrometer respired gas analyzer (Ohmeda), four transcutaneous oxygen sensors (model 807, Novametrix), and two transcutaneous carbon dioxide tension (Ptc_{CO_2}) sensors (model 811, Novametrix). The oxygen electrodes were heated to temperatures between 42 and 44°C and were placed on the skin at various sites, including the chest, thigh, calf, dorsum of the hand, and dorsum of the foot. In 6 of the subjects, skin blood flow was continuously monitored on the chest and the hand (thenar eminence) or foot (anterior to medial malleolus) using a model BPM-403A Laserflo Blood Perfusion Monitor (TSI, Inc., St. Paul, MN). This device uses the principles of laser-Doppler velocimetry to measure perfusion near the skin surface. The Laserflo provides a digital display of either flow velocity or volume of blood flow. Two Laserflo devices were used, and the flow probes were not moved during the study once they were attached to the skin. The two probes were located on the chest and hand of 3 subjects and on the chest and foot of the other 3 subjects.

Data were recorded during normocapnia and steady-state voluntary hyperventilation with subjects breathing room air. After 20 min of baseline data recording, the subjects began breathing at a rate of 24 breaths per min, while maintaining a larger than normal tidal volume. Noninvasive data, recorded every 2 min, included Ptc_{O_2}, Ptc_{CO_2}, laser-Doppler skin blood flow, inspired and end-tidal oxygen and carbon dioxide tensions, and vital signs. An arterial sample was obtained for *in vitro* blood gas analysis (Nova STAT-3 Blood Gas Analyzer) every 4 min. Hyperventilation was terminated after 20 min, and the subjects were then instructed to breathe normally. Data were recorded for an additional 30 min after the end of hyperventilation. The statistical significance of differences in mean TCI between normocapnia and hypocapnia was determined by Student's paired, two-tailed *t* test.

¶ Barker SJ, Tremper KK, Hyatt J: Effects of hypothermia on noninvasive gas monitoring. Proceedings of Ninth World Congress of Anaesthesiology, Washington, DC, 1988, p A0-410.

Subjects were asked to discontinue hyperventilation if they experienced any unpleasant symptoms such as dizziness, impending loss of consciousness, impaired vision, or muscle contractures. Although Pa_{CO_2} values as low as 17 mmHg were recorded, the volunteers reported only mild symptoms such as distal paresthesia and fatigue, and all completed the experimental protocol. Three subjects hyperventilated for less than 20 min due to fatigue; each of these, however, hyperventilated for at least 15 min.

Results

Animal Study

A total of 84 data sets were recorded during 13 series in which Pa_{CO_2} was varied stepwise while all other parameters were fixed. Figure 1 shows TCI plotted *versus* Pa_{CO_2} for three different electrode temperatures at FI_{O_2} = 1.0. Each data point in this figure is an average of data obtained from all five animals at the same conditions, *i.e.*, the same FI_{O_2} and Pa_{CO_2} values. Error bars indicate standard deviations. In every series, the TCI decreased significantly with decreasing Pa_{CO_2} ($P < 0.002$ by ANOVA), often reaching values of less than 0.1 for Pa_{CO_2} less than 22 mmHg. The P values for the comparison of each hypocapnic TCI with the control normocapnic value (Dunnett t test) are shown in the figure. At each of the three temperatures, the decrease in TCI at the lowest Pa_{CO_2} value is significant to the level $P < 0.01$.

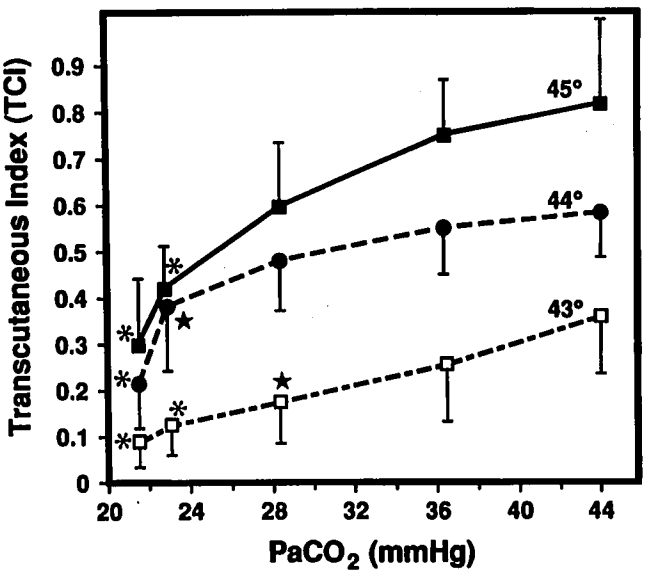

FIG. 1. Transcutaneous index (TCI) *versus* Pa_{CO_2} for FI_{O_2} = 1.0. Each point is an average of data from five pigs. Three electrode temperatures are shown: 43, 44, and 45° C. Error bars indicate standard deviations. During hyperventilation at FI_{O_2} = 1.0, TCI decreased by 65% of its highest value at electrode temperature = 45° C and by 80% of its highest value at electrode temperature = 43° C. Statistical significance compared to TCI at normocarbia: asterisk = $P < 0.01$; star = $P < 0.05$ (ANOVA, Dunnett t test).

TABLE 1. Mean Transcutaneous Index Values for Five Pigs *versus* Pa_{CO_2}, FI_{O_2}, and Electrode Temperature

FI_{O_2}	Pa_{CO_2} (mmHg)	Mean TCI (T_E [° C])		
		43	44	45
1.0	44	0.33	0.56	0.78
	20	0.065	0.20	0.27
	ΔTCI	80%*	63%†	65%†
0.3	44	0.42	0.58	0.82
	20	0.097	0.25	0.44
	ΔTCI*	77%†	58%‡	46%‡

ΔTCI = 100 × (TCI [44 mmHg] − TCI [20 mmHg]/TCI [44 mmHg].
TCI = transcutaneous index; T_E = electrode temperature.
* $P < 0.005$ (ANOVA with Dunnett–t *post hoc*).
† $P < 0.01$.
‡ $P < 0.05$.

Although Pa_{O_2} increased slightly during hyperventilation (due to increased Pa_{O_2}), the decreases in TCI are due mainly to large decreases in Ptc_{O_2}. The highest TCI value of 0.78 was achieved at the highest electrode temperature (45° C) and at the highest Pa_{CO_2} value (44 mmHg) shown in figure 1. At the lowest Pa_{CO_2} value, 20 mmHg, the mean TCI for the 45° C electrode temperature decreased to 0.27, a 65% decrease from its normocapnic value. Under the same conditions for an electrode temperature of 43°C, the mean TCI decreased to 0.065, an 80% decrease from its normocapnic value. The relative effect of hypocapnia upon TCI is most significant at lower electrode temperatures ($t = 4.8$, $P < 0.005$ for an electrode temperature of 43° C and an FI_{O_2} of 1.0).

Similar results for TCI during hypocapnia were obtained at FI_{O_2} = 0.3, as shown in table 1. In this table, the highest TCI value for each electrode temperature occurred at a mean Pa_{CO_2} of 44 mmHg, and the lowest TCI value occurred at a mean Pa_{CO_2} of 20 mmHg. The ΔTCI values given in the table are the percentage decreases in TCI caused by hypocapnia for each value of electrode temperature and FI_{O_2}. P values (Dunnett t test) are indicated for each ΔTCI. Hypercapnia with Pa_{CO_2} values as great as 65 mmHg had little effect upon TCI as long as concurrent hypoxemia was avoided.

Human Volunteer Study

Table 2 illustrates TCI results from human volunteers for two Pa_{CO_2} levels, two sensor temperatures (42 and 44° C), and three transcutaneous sensor locations (chest, hand, and foot). The mean Pa_{CO_2} decreased from 36 ± 2.8 mmHg during normal ventilation to 17 ± 2.5 mmHg during voluntary hyperventilation. This decrease in Pa_{CO_2} was accompanied by a significant decrease in TCI in every subject. As in the animal experiments, the de-

TABLE 2. Transcutaneous Index Values for 12 Volunteers *versus* Pa_{CO_2}, Electrode Temperature, and Sensor Location

T_E (° C)	Pa_{CO_2} (mmHg)	TCI		
		Chest	Hand	Foot
42	36 2.8	0.51 ± 0.14	0.27 ± 0.10	0.39 ± 0.16
	17 2.5	0.25 ± 0.076	0.072 ± 0.034	0.14 ± 0.073
Δ TCI		51%	73%	64%
44	36 2.8	0.77 ± 0.10	0.48 ± 0.11	0.63 ± 0.083
	17 2.5	0.60 ± 0.069	0.24 ± 0.084	0.32 ± 0.075
Δ TCI		22%	50%	49%

Values are means ± SD.

ΔTCI = 100 × (TCI [36 mmHg] − TCI [17 mmHg])/TCI [36 mmHg]. All ΔTCI values are significant to level $P < 0.001$ (Student's *t* test).

TCI = transcutaneous index; T_E = electrode temperature.

creases in TCI were mainly the result of large decreases in Ptc_{O_2}, accompanied by modest increases in Pa_{O_2}. Ptc_{O_2} values on the extremities decreased to less than 10 mmHg during hyperventilation in most subjects. The percentage decreases in TCI during hypocapnia (Δ TCI) are also shown in table 2. All Δ TCI values are statistically significant to a level of $P < 0.001$ (paired, two-tailed Student *t* test). The greatest ΔTCI values occurred on the hand and foot, and greater TCI decreases occurred at lower sensor temperatures ($P < 0.01$), as in the animal study. TCI values for sensors located on the thigh and calf were intermediate between values obtained on the chest and those obtained on the hands and feet.

Figure 2 is a plot of TCI *versus* time for three sensor locations on a typical subject, for a sensor temperature of 42° C. Also shown in this figure is a plot of the laser-Doppler blood flow on the foot (anterior to the medial malleolus) during the same time period. Since the Laserflo device displays blood flow in arbitrary units and its values are highly variable between subjects, we have normalized the blood flow by dividing it by its baseline, prehyperventilation value. At 12 min on the time axis, voluntary hyperventilation was begun, and at 42 min the subject was instructed to breathe normally. This figure illustrates several characteristics common to all subjects. At the commencement of hyperventilation, both TCI and skin blood flow began to decrease immediately. Blood flow on the foot reached a minimum value of less than half of its baseline. The TCI decreased from baseline values at all three sensor locations, but the relative decreases were largest on the hand and foot. The same was true of laser blood flow, which exhibited much smaller changes on the chest than on the foot. After the period of hyperventilation, laser blood flow usually rebounded to values significantly greater than baseline. This posthyperventilation hyperemia was seen in four of the six subjects who were instrumented with the laser flow probes. TCI often exhibited the same phenomenon (fig. 2), although the overshoot above baseline occasionally was delayed. Laser-Doppler blood flow on the thenar eminence of the hand shows the same behavior seen in figure 2 for the foot.

Plots of TCI *versus* time for the 44° C sensor are similar in appearance to that of figure 2. However, the relative decreases in TCI during hyperventilation were smaller

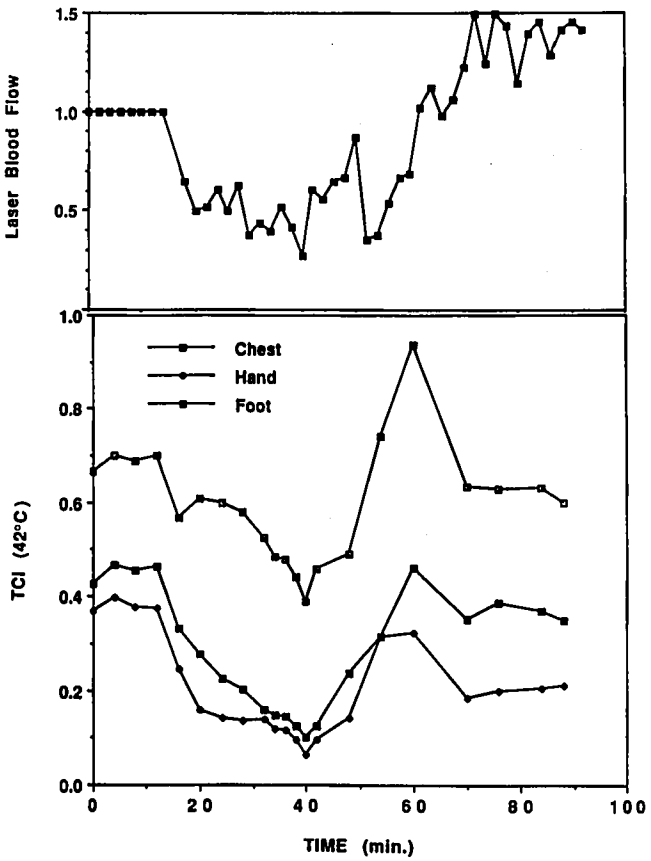

FIG. 2. *Top:* Normalized laser-Doppler (Laserflo) blood flow on the foot *versus* time in a hyperventilating subject. Hyperventilation begins at t = 12 min and ends at t = 40 min. *Bottom:* Transcutaneous index (TCI) for a 42° C electrode on the chest, hand, and foot. Both blood flow and TCI (chest) overshoot above baseline values after the end of hyperventilation.

(table 2), and the TCI values for the 44° C sensor reached a more well-defined plateau during hyperventilation because of its faster time response. Furthermore, the post-hyperventilation hyperemia effect was more pronounced for the 44° C sensor; that is, the TCI consistently overshot its prehyperventilation baseline value.

As stated above, we normalized the laser-Doppler blood flow values for each subject to the prehyperventilation baseline. Thus, the baseline blood flow was defined to be 1.0 for each subject at each site. The six-subject average normalized skin blood flow during hyperventilation is then 0.92 ± 0.35 on the chest, 0.40 ± 0.13 on the hand, and 0.49 ± 0.04 on the foot. That is, hyperventilation caused blood flow to decrease by 8% on the chest, 60% on the hand, and 51% on the foot. The decreases in flow on the hand and foot were statistically significant to a level of $P < 0.005$ (paired, two-tailed, Student t test). The change in blood flow on the chest was not statistically significant.

Discussion

Previous animal and clinical studies have shown that TCI, the ratio of transcutaneous to arterial oxygen tension, is a function of both age and cardiac output.[3–8] During stable hemodynamic conditions in a given patient, the TCI is assumed to have a constant value. The present study showed that this is not so. In our animal experiments the TCI decreased consistently and significantly during hyperventilation to any Pa_{CO_2} value less than 36 mmHg. These TCI decreases occurred in the absence of statistically significant changes in cardiac output or blood pressure. The largest relative decreases in TCI occurred at the lowest electrode temperatures, where TCI during hyperventilation reached about one fifth of its normocapnic value. Even at the highest electrode temperature, 45° C, TCI during hyperventilation decreased to less than one half of its normocapnic value (fig. 1).

In the study in human volunteers, the TCI also decreased significantly during hyperventilation, but the decreases for sensors on the chest were less than those for the corresponding sensors in the animal experiments. For the 44° C electrode located on the chest, the mean TCI decreased from 0.77 at normocapnia to 0.60 during hyperventilation, a change of only 22% (table 2). For the 42° C electrode in the same location, the mean TCI decreased by 51% during hyperventilation. For sensors located on the hand and foot, the decreases in TCI during hyperventilation were much greater than those on the chest (table 2 and fig. 2). As in the animal experiments, the relative decreases in TCI were greater at the lower electrode temperature. For example, the mean TCI on the hand for the 42° C electrode fell to about one fourth of its normocapnic value during hyperventilation.

Aside from species differences, there are several possible reasons for the smaller decreases in the chest TCI in the study in volunteers. The human volunteers were awake, whereas the animals were deeply anesthetized and paralyzed. The volunteers were breathing spontaneously, whereas the animals' lungs were mechanically ventilated. Cardiac output was maintained nearly constant during the animal study, and it was not monitored in the volunteer study. The volunteers were limited in the duration of hyperventilation by fatigue, whereas the animals were not. One previous study in volunteers found that Ptc_{O_2} on the arm actually increased during hyperventilation.[10] However, the hyperventilation in that study was continued for only 1 min, and Pa_{O_2} was not measured. The early increase in Ptc_{O_2} during hyperventilation merely reflected the almost immediate increase in Pa_{O_2} and was also seen in the present study. It was quickly followed by a much greater decrease in Ptc_{O_2}, which continued until the termination of hyperventilation. The TCI began its decrease within 1 min of the onset of hyperventilation, as shown in figure 2.

Laser-Doppler skin blood flow measurements on six subjects show that the decreases in TCI during hyperventilation are accompanied by significant decreases in skin blood flow on the extremities. On the chest, where the decreases in TCI during hyperventilation are smaller, the change in skin blood flow is not statistically significant. However, skin blood flow on the hand and foot decrease to an average of less than half of their baseline values during hyperventilation. After the termination of voluntary hyperventilation, the skin blood flow on the hand and foot usually rebound to values significantly greater than their prehyperventilation baseline. For the 44° C electrode, this apparent hyperemia is accompanied by a simultaneous overshoot of the TCI above baseline. It is well known that Ptc_{O_2} is affected by blood flow, particularly at lower electrode temperatures.[5–8] It is reasonable to conclude that the decreases in TCI seen during hyperventilation in this study are a consequence of the accompanying skin blood-flow changes.

The results of this study have two clinical implications. First, hyperventilation decreases skin perfusion in the distal extremities. Second, the relationship between transcutaneous and arterial oxygen tension does not remain constant during either controlled or spontaneous hyperventilation. A decrease in Ptc_{O_2} commencing with the onset of hyperventilation does not necessarily imply a decrease in Pa_{O_2}. The commonly accepted age-dependent values of TCI cannot be assumed under these conditions. Specifically, in a hyperventilating neonate, the Pa_{O_2} may be significantly greater than the Ptc_{O_2}, even under stable hemodynamic conditions. In neonates at risk from hyperoxemia, such an underestimate of Pa_{O_2} could have se-

rious consequences. In conclusion, this study has uncovered an interesting phenomenon of skin perfusion physiology, and it has pointed out yet another limitation to the clinical use of Ptc_{O_2}.

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