
TRANSCUTANEOUS P_{CO_2} AND P_{O_2} : A MULTICENTER STUDY OF ACCURACY

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ABSTRACT. A multicenter study used 756 samples from 251 patients in 12 institutions to compare arterial (PaO_2 , $PaCO_2$) with transcutaneous (PsO_2 , $PsCO_2$) oxygen and carbon dioxide tensions, measured usually at 44°C. Of these samples, 336 were obtained from 116 neonates, 27 from 25 children with cystic fibrosis, and 140 from 40 patients under general anesthesia. Ninety-one patients were between 4 weeks and 18 years of age, 32 were between 18 and 60 years, and 12 were over 60. The ratio of transcutaneous to arterial $P(s/a)CO_2$ was 1.01 ± 0.11 with $PaCO_2$ less than 30 mm Hg, increasing to 1.04 ± 0.08 at $PaCO_2$ greater than 40 mm Hg. Mean bias and its standard deviation ($PsCO_2 - PaCO_2$) were $+1.3 \pm 3.9$ mm Hg in the entire group, $+1.8 \pm 4.2$ mm Hg in neonates (NS). Bias was $+0.2 \pm 2.7$ mm Hg when $PaCO_2$ was less than 30 mm Hg ($N = 175$, NS), 1.0 ± 3.4 with $30 < PaCO_2 < 40$ ($n = 329$, $p < 0.001$), and $+2.04 \pm 4.00$ mm Hg with $40 < PaCO_2 < 70$ ($n = 229$, $p < 0.001$). These data suggest that, using transcutaneous PCO_2 monitors with inbuilt temperature correction of 4.5%/°C, the skin metabolic offset should be set to 6 mm Hg. The linear regression was $PsCO_2 = 1.052(PaCO_2) - 0.56$, $Sy \cdot x = 3.92$, $R = 0.929$ ($n = 756$); and $PsCO_2 = 1.09(PaCO_2) - 1.57$, $Sy \cdot x = 4.17$, $R = 0.928$ in neonates ($n = 336$). The use of vasopressors and vasodilators had no significant effect on bias or its standard deviation or on regression slope and intercept ($n = 78$). In cystic fibrosis patients, bias and standard deviation were 0.0 ± 1.7 mm Hg ($n = 27$). Under anesthesia, $PsCO_2 = 1.07PaCO_2 - 1.58$, with bias and standard deviation = 0.6 ± 3.5 ($n = 140$). For oxygen, at $PaO_2 \leq 80$ the ratio $P(s/a)O_2 = 1.05 \pm 0.16$ in neonates and 0.93 ± 0.21 in older patients, but when $PaO_2 > 80$, $P(s/a)O_2$ fell to 0.88 ± 0.18 in neonates and 0.74 ± 0.21 in older patients. The errors were significantly greater ($p < 0.001$) in older patients than in neonates above but not below 80 mm Hg, and within both groups errors were significantly greater above than below 80 mm Hg.

KEY WORDS. Monitoring. Measurement techniques: blood gases. Blood gas analysis.

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Beginning about 1980, many transcutaneous blood gas monitors combined O_2 and CO_2 electrodes in a single probe, and users now generally have combined systems. Transcutaneous oxygen tension (PsO_2) has been widely reported to be lower than arterial oxygen tension (PaO_2) in all patients after a few months of age, and its errors are so inconstant and unpredictable that it can be used at best only as a guide to changes in either PaO_2 or skin perfusion [1,2]. $PsCO_2$ has been found at least as accurate as end-tidal PCO_2 for noninvasive monitoring by Phan et al [3] and McEvedy et al [4].

The following multiinstitutional study was carried out in 1984 through 1986 to provide evidence of accuracy of these methods to facilitate approval by the Food and Drug Administration. During the last 4 years, however, advantages of pulse oximetry over PsO_2 and em-

phasis on monitoring end-tidal PCO_2 have resulted in substantial distrust and disuse of transcutaneous blood gas monitoring. The results are published to confirm the accuracy and hence usefulness of transcutaneous PCO_2 monitoring.

METHODS

Nomenclature

Partial pressures are given in millimeters of mercury (mm Hg). Subscripts are: a = arterial, s = skin (transcutaneous electrodes), ET = end-tidal. PsCO_2 is corrected for temperature and skin metabolism [5]. PsO_2 is corrected ($\times 1.1$) for electrode O_2 consumption (the "stirring effect") but not for temperature or skin metabolism.

Data reported herein were obtained in 12 clinical facilities (2 of which were at the University of California, San Francisco, the adult and the pediatric intensive care units) using a combined transcutaneous PO_2 and PCO_2 electrode (Radiometer TCM-11, Copenhagen, Denmark; later marketed as TMC-3 or TINA). With approval of the human research committees of each institution, studies were conducted in patients in critical care units or operating rooms. There were no subject exclusion criteria but all had indwelling arterial catheters and available blood gas analysis. Most patients were receiving mechanical ventilation. Of 251 patients, 116 were less than 1 month of age, 123 were between 1 month and 60 years of age, and 12 were over 60. The patients represented a wide spectrum of disease processes typically seen in intensive care units and operating rooms. There were 75 infants with respiratory distress syndrome and 17 with persistent pulmonary hypertension. Fifteen adults had had intracranial hemorrhage, and 25 patients had cystic fibrosis. Forty-five were in the postoperative period, 34 after cardiac and 11 after noncardiac operations. Forty patients were tested during surgery and anesthesia, which consisted of nitrous oxide and a vapor (halothane, enflurane, or isoflurane), or nitrous oxide and fentanyl. Twenty-three were receiving continuous infusions of vasoactive substances: 16 of dopamine; 4 of tolazoline; 2 of each of the following: isoproterenol, nitroprusside, aminophylline, and hydralazine; and 1 of epinephrine.

One TCM-11 was supplied to each test location. The combined PO_2 - PCO_2 electrode was covered with a 25- μm FEP Teflon membrane. The Clark type PO_2 electrode contained a 20- μm diameter platinum cathode polarized to -0.7v . The CO_2 electrode contained a central glass pH electrode 5 mm in diameter. The chlorided

silver body served as reference electrode for both PO_2 and PCO_2 . No spacer was used to separate the Teflon membrane from the pH glass and platinum cathode. The electrolyte was 50 mM KHCO_3 , 50 mM KCl, 40% propylene glycol and 40% glycerine, 1% hydroxyethyl-cellulose, and the remainder water.

The electrodes were heated to 44°C and were calibrated in the "gas" switch position using 10% CO_2 , 15% O_2 , balance N_2 , saturated with water in a 37°C calibration cuvette mounted on the side of the monitor amplifier. The PCO_2 reading was set to $\text{FCO}_2(\text{P}_b - 60)$, where 60 mm Hg is the estimated PH_2O at the surface of the heated electrode during calibration. PO_2 reading was set to $1.1(\text{FO}_2)(\text{P}_b - 60)$, where 1.1 is the gas/liquid or gas/skin ratio of the Clark electrode (the "stirring effect") [6], a function of membrane permeability, electrolyte composition and thickness, and cathode diameter.

Electrodes were mounted on soft, thin skin, such as lateral abdomen, anterior or lateral chest, volar forearm, inner upper arm, or inner thigh. The skin was cleaned with alcohol and an adhesive mounting ring applied. A contact liquid consisting of 50% propylene glycol in water was added in the well of this ring (2-3 drops), and the electrode was screwed into the ring, which held the electrode surface flush with the skin surface. For all subjects except newborns, the electrode temperature was increased to 46°C for the first 4 minutes using an inbuilt timed "preheat" control to enhance rapid skin vasodilation. After 4 minutes, temperature automatically decreased to 44°C . For some newborns, when the clinicians in charge preferred to preset temperature to 43°C to decrease the possibility of burning the skin, calibration was appropriately adjusted and no preheating was used.

After placing the sensor on the skin, the user set a panel switch from "gas" to "skin." This electrically corrected the PsCO_2 reading for the effects of heating (by multiplying by 0.73) and of skin metabolism (by subtracting 4 mm Hg) [5,7]. Without this correction, PsCO_2 exceeds PaCO_2 for two reasons: (1) heating of the skin increases the local tissue and blood PCO_2 by about 4.5% per degree Celsius, and (2) skin metabolism raises surface PCO_2 about 4 mm Hg above capillary PCO_2 . Under a heated skin electrode, temperature at the capillary level has been estimated indirectly [6] to be heated to about 80% of the electrode-body temperature difference, which at 44°C is about 1.4°C lower than the electrode temperature. The electrode reading is also increased by the cooling of its surface due to heat loss to skin. The net result is that these effects combine to an observed temperature correction factor of about 4.5% per degree Centigrade. At 44° , the factor was then cal-

Table 1. Transcutaneous versus Arterial PCO_2 in Multicenter Study

Group	No.		Linear regression				P(s/a)CO ₂ (mm Hg)		P(s-a)CO ₂ (mm Hg)	
	Subjects	Data	Slope	Intercept	S _{Y·X}	R	Mean	SD	Bias	SD
Total	251	756	1.052	-0.560	3.92	0.929	1.035	0.118	1.30	3.95
Subset	251	733	1.067	-1.307	3.48	0.944	1.028	0.099	1.13	3.53
<4 weeks	116	336	1.09	-1.57	4.17	0.928	1.043	0.113	1.79	4.25
5 wk-18 yr	91	260	1.001	0.603	3.099	0.946	1.019	0.089	0.654	3.093
19-60 yr	32	121	1.073	-2.061	3.621	0.926	1.004	0.113	0.217	3.656
>60 yr	12	39	1.15	-3.15	4.097	0.891	1.06	0.11	2.22	4.18
Vasoact drug	23	78	1.016	-0.086	3.085	0.963	1.01	0.10	0.47	3.07
Cystic fibrosis	25	27	1.05	-2.27	1.585	0.991	0.997	0.039	0.01	1.66
Anesthesia	40	140	1.07	-1.58	3.47	0.911	1.016	0.103	0.58	3.50

Note: The subset excludes 23 points where $P(s/a)CO_2 > 2SD$ from mean, illustrating that aberrant data did not significantly skew the results. "Vasoact drug" represents patients receiving either vasopressors or vasodilators.

culated as $e^{0.045(37-44)} = 0.73$. When temperature and skin metabolism corrections are not made, $PsCO_2$ values are about 15 to 30 mm Hg higher than $PaCO_2$, depending on the $PaCO_2$ level [8].

Comparison readings were made after at least 20 minutes of equilibration on skin. The $PsCO_2$ and PsO_2 readings were made 1 minute after withdrawal of arterial samples to permit equilibration of skin surface with small changes in arterial gas tensions. Arterial blood gases were measured and reported at 37°C. Generally 3 or 4 comparison samples were obtained from each patient.

Data were analyzed by linear regression and by Student's paired *t* test analysis. Bias and standard deviation are the mean and standard deviation of $P(s-a)CO_2$. The ratio $P(s/a)CO_2$ and its standard deviation were also computed to determine whether the errors of PsO_2 were proportional to the absolute level of $PaCO_2$.

RESULTS

The mean bias and its standard deviation in 756 sample pairs were $+1.30 \pm 3.95$ mm Hg. Exclusion of 23 data points exceeding two standard deviations has no significant effect on bias or standard deviation of the group, confirming that the data were not skewed by a small number of data far out of range. Table 1 presents the linear regression relationships, the bias and standard deviation, and the transcutaneous/arterial ratio with its standard deviation for all 756 comparisons and for various subgroups.

The ratio of transcutaneous to arterial PCO_2 (a better index than bias in that errors tend to be proportional to the $PaCO_2$) expressed as $P(s/a)CO_2$, averaged 1.01 at low $PaCO_2$ and rose to 1.04 at high $PaCO_2$, while its

Table 2. Errors of $PsCO_2$ Related to Level of $PaCO_2$ by Decades in the Subset of 733 Comparisons

PaCO ₂			P(s/a)CO ₂ (mm Hg)		P(s-a)CO ₂ mm Hg	
Range	Mean	No.	Mean	SD	Bias	SD
All						
<30	25.5	175	1.008	0.110	0.22	2.69
30-40	35.5	329	1.028*	0.099	0.98*	3.44
40-50	44.1	173	1.047*	0.087	2.03*	3.77
50-60	54.7	36	1.042*	0.092	2.12*	5.02
60-70	64.1	20	1.030	0.064	1.84	4.04
Neonates						
<30	25.4	63	1.01	0.11	0.48	2.86
30-40	35.6	175	1.04*	0.12	1.56*	4.41
40-50	43.7	101	1.05*	0.08	2.50*	3.60
>50	57.4	36	1.05*	0.09	3.20*	5.29

* $P < 0.05$ indicates that $P(s/a)CO_2$ is significantly different from 1.00 or $P(s-a)CO_2$ is significantly different from 0.

standard deviation of about 0.1 was independent of the $PaCO_2$ level (Table 2).

These data are plotted in Figure 1 with asterisks for the 23 points in which $P(s/a)CO_2$ deviated more than 2 standard deviations from the mean. A separate analysis is shown for the subset excluding those points in Table 1. The bias, standard deviation, and correlation coefficient were not larger in cystic fibrosis patients than in other groups of patients in this study (Fig 2 and Table 1).

The 649 comparisons of PsO_2 with PaO_2 where PaO_2 is less than 220 mm Hg are displayed in Figure 3. The relationship between PsO_2 and PaO_2 was arbitrarily divided into 2 groups, above and below a PaO_2 of 80 mm Hg, and the population was subdivided into neonates

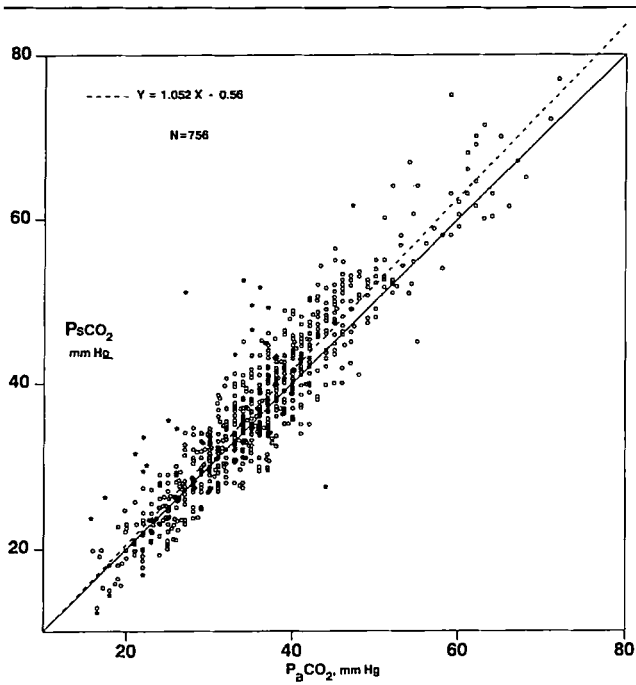


Fig 1. The relationship of P_sCO_2 to P_aCO_2 , $n = 756$. For 23 points denoted by (*), $P(s/a)CO_2$ deviates by more than 2 SD to indicate that aberrant data were not skewed. The solid line is identity.

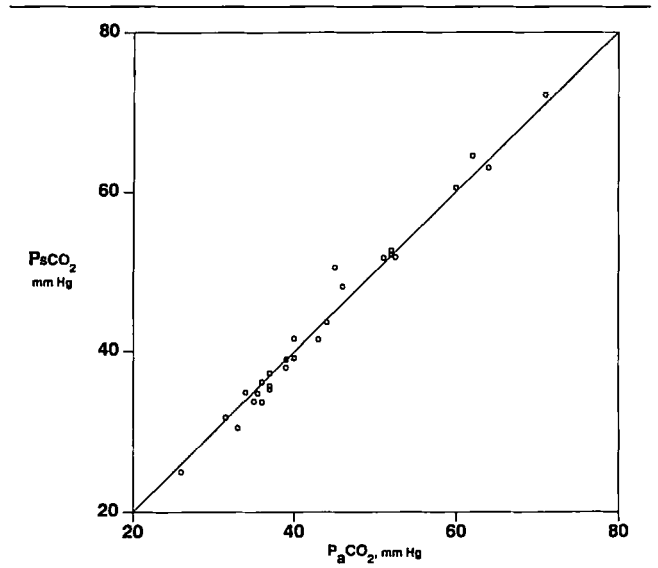


Fig 2. Twenty-seven samples of P_aCO_2 and P_sCO_2 from patients with cystic fibrosis, with line of identity.

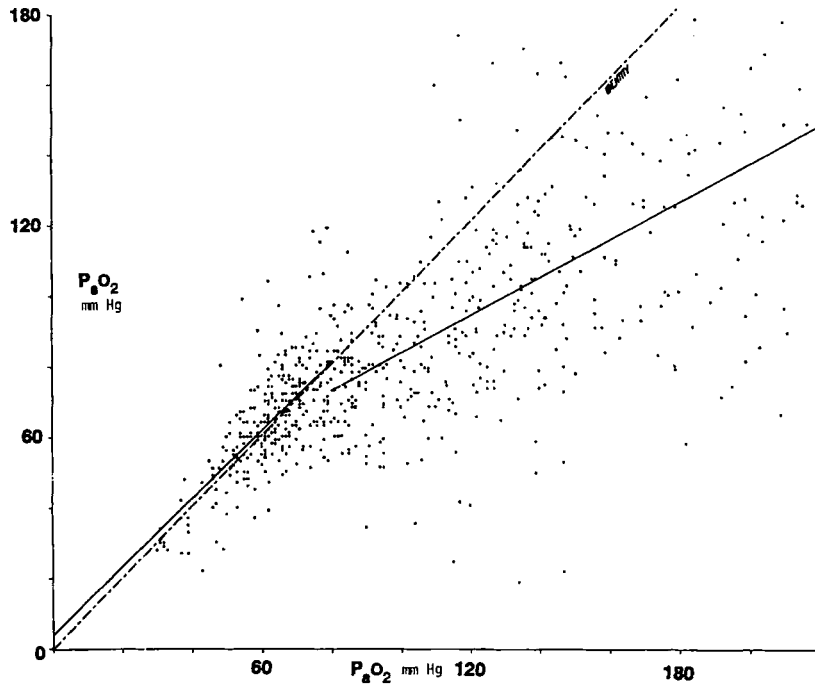


Fig 3. The relationship of P_sO_2 to P_aO_2 , $n = 723$. Regression lines are shown for P_aO_2 below and above 80 mm Hg.

Table 3. Transcutaneous Oxygen Electrode Results from 232 Patients

PaO ₂ (mm Hg)	No.	P(s/a)O ₂		P(a-s)O ₂		Slope	Intercept (mm Hg)	S _{y,x} (mm Hg)	R
		Mean	SD	Bias	SD				
<i>Neonates</i>									
≤80	213	1.045	0.155	3.0	10.2	1.01	2.5	10.3	0.714
>80	97	0.88	0.177	16.4	24.7	0.71	15.7	17.6	0.928
<i>Older</i>									
≤80	76	0.927	0.205	4.9	11.9	0.80	7.4	11.7	0.666
>80	327	0.737	0.209	42.9	40.5	0.65	10.5	34.5	0.758

Note: Neonates were less than 10 days of age. When PaO₂ > 80 mm Hg, P(s/a)O₂ is significantly lower in older patients than in neonates, and in both neonates and older patients is lower than with PaO₂ < 80 mm Hg ($P < 0.01$). When PaO₂ < 80, errors are not significantly greater in older patients than in neonates.

less than 10 days of age and all older subjects. The statistical results are presented in Table 3. Errors were significantly less ($p < 0.001$) in neonates than others and were less in all subjects when PaO₂ was 80 mm Hg or less.

DISCUSSION

The data presented here suggest that PsCO₂ may be used to accurately estimate arterial PCO₂ in all age groups, in contrast to the problems with PsO₂. PsCO₂ monitoring deserves far wider use, particularly when end-tidal gas analysis is impractical, as in postoperative recovery, ventilator weaning of intensive care patients, epidural narcotic pain relief, sleep studies, and office endoscopy.

The need for correction of skin PCO₂ to estimate arterial PCO₂ was evident when PsCO₂ measurements were first attempted. The earliest approach was to reduce the gain of the amplifier before the signal was exponentiated. This could only match arterial PCO₂ at a single level, and resulted in too low a span, so that low PCO₂ read too high and high PCO₂ read too low. Some commercial systems (e.g., Novamatrix) used this method for several years despite this problem.

An appropriate correction was shown to require both a gain and an offset factor, the latter being rationalized as due to skin metabolism, adding a CO₂ diffusion gradient (4–6 mm Hg) that was independent of the absolute level of PCO₂ [5,7]. Suitable adjustment of these two factors permits electrodes to track PaCO₂ accurately over a wide range. This is, in effect, using a linear regression equation type of correction with slope and offset factors.

In one study, PsCO₂ corrected for temperature was compared with PaCO₂ corrected to body temperature, which is uniformly lower during general anesthesia [3]. This procedure is not logical, since the PsCO₂ correction factor is designed to correct to 37°C, the blood gas

measurement temperature, and the skin temperature is determined almost entirely by electrode, not body, temperature. The effect of body temperature on PETCO₂ (due to varying water vapor temperature) is negligible.

Hazinski and Severinghaus [7] reviewed data from 12 publications containing over 1,800 comparisons of PsCO₂ with PaCO₂ and applied these dual temperature and metabolism corrections to those data when uncorrected values were given. They found that the corrected values agreed with PaCO₂ to about ± 4 mm Hg. Almost all published data were described by regression equations, whereas mean and standard deviation of bias were not available for most data. Several papers noted that hypotension and shock generally have far less effect on PsCO₂ than on PsO₂, but that shock can seriously impair the accuracy of PsCO₂.

Mindt et al [9] also reviewed most of the published literature with emphasis on the methodologic errors in various calibration procedures. They suggested that it is not reasonable to apply a uniform regression correction to data from various authors to determine the corrected value of PsCO₂. A major problem with the use of regression equations is that in individual patients PCO₂ is not deliberately varied over a wide range to collect data for regression analysis. When the spontaneously varying values are used, coming from many different subjects, other errors are included, and the range covered is still too small and clustered around normal values.

A review by Cassady [10] in 1983 of 8 published reports involving PsCO₂ monitoring illustrated the wide variation of temperature correction algorithms which resulted from regression analysis.

Epstein et al [11] compared Radiometer PsCO₂ electrodes with PaCO₂. When they multiplied electrode gas calibration values by 0.73 to correct for temperature, they found a residual mean offset of 6.4 mm Hg presumed to be due to skin metabolism.

The regression data of the present study might be thought to suggest a temperature factor of 5.4% per

degree Celsius and a skin metabolic offset of 2.5 mm Hg; however, the problems with use of linear regression described above preclude recommending this method. After the present study was completed, in order to measure directly the optimal correction factors for $P_s\text{CO}_2$, Naifeh and Severinghaus [12] exposed 6 volunteers to hypercapnia using 4 Radiometer electrodes simultaneously in each subject. They chose to calibrate with a 6 mm Hg skin metabolism offset factor, derived from the data shown in Table 2, indicating that the mean residual offset bias in the PCO_2 range of 40 to 60 mm Hg was 2 mm Hg⁻ when a 4 mm Hg skin metabolism factor was used. Epstein et al [11] later obtained the same offset. With this offset, $P_s\text{CO}_2$ agreed optimally with PaCO_2 when electrodes were calibrated to 52.0 mm Hg with 10.33% CO_2 in the "skin" switch position. The appropriate temperature correction factor computed from these data would be 3.0% per degree Celsius. This reduction of the temperature factor occurred because, after 1982, Radiometer electrodes were made less sensitive to cooling of the sensor surface by skin [13]. To calibrate correctly the present equipment in the "gas" switch position, with its 4.5% per degree Celsius factor, using a 6 mm Hg offset, it is necessary to increase the assigned value of the calibration gas CO_2 concentration by the factor 1.11. For example, at 760 mm Hg Pb and 44°C, using 10.0% CO_2 , the instruments would be set to read $0.111 (\text{Pb}-60) = 77.7$ mm Hg.

The circumstances in which $P_s\text{CO}_2$ may be useful are often those in which PETCO_2 is the only alternative and is either impractical or inaccurate. In neonates, McEvedy et al [4] reported that $P_s\text{CO}_2$ was a more accurate monitor than PETCO_2 or PaCO_2 , even when PETCO_2 was obtained from the distal end of an endotracheal tube. This was true in neonates with normal lungs, but even more so in those with pulmonary abnormalities. Phan et al [3] reached the same conclusion in a study of 24 anesthetized adults.

The ability of $P_s\text{CO}_2$ electrodes to track PaCO_2 accurately has been documented in subjects over 60 years of age as well as in younger adults [12,14]. $P_s\text{CO}_2$ is sufficiently accurate at all ages for most clinical monitoring purposes, and it may be particularly helpful in patients in whom respiratory depression may occur but in whom monitoring of PETCO_2 is inaccurate, difficult, or impossible.

The relatively greater accuracy of $P_s\text{CO}_2$ than of PsO_2 had been noted by 1982. PsO_2 is reasonably accurate in neonates at low PO_2 , but in older patients it is useful only to indicate changes of PaO_2 and then only in states of adequate blood flow. PsO_2 values are never temperature corrected because, whereas skin metabolism lowers skin surface PO_2 , heating raises it, and

these two approximately cancel each other in neonates at low PO_2 levels. The effect of temperature on blood PO_2 varies from 1.3% per degree Celsius in fully saturated blood to 7.2% per degree Celsius when SaO_2 is less than 90%. This accounts for the nonlinearity evident in Figure 3, with a larger error when PO_2 is greater than about 80 mm Hg.

The scatter illustrated by Figure 3 confirms the general impression that PsO_2 is of rather limited value, especially now that pulse oximetry is readily available, without need of calibration or of membrane changes or skin heating and with remarkable accuracy in comparison with PsO_2 . Fortunately, skin PCO_2 appears far more dependable than the skin PO_2 , since no other method for noninvasively following blood PCO_2 is feasible in a large array of clinical situations.

An abstract of this work was presented previously [15].

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