Transcutaneous Oxygen and Carbon Dioxide Tensions Compared to Arterial Blood Gases in Normals

Lindell K Weaver MD

BACKGROUND: Most hyperbaric medicine centers do not monitor arterial oxygen (P_{aO_2}) and carbon dioxide (P_{aCO_2}) tensions during hyperbaric oxygen, but many can transcutaneously monitor oxygen (P_{tcO_2}) and carbon dioxide (P_{tcCO_2}). METHODS: We compared P_{tcO_2} and P_{tcCO_2} measurements to simultaneous P_{aO_2} and P_{aCO_2} measurements in 10 healthy volunteers to determine if P_{tcO_2} and P_{tcCO_2} measurements are surrogates for P_{aO_2} and P_{aCO_2} in the hyperbaric environment. We took blood samples via arterial catheter and took P_{tcO_2} and P_{tcCO_2} chest measurements while the subjects were compressed in a monoplace hyperbaric chamber at pressures between 0.85 atmospheres absolute (atm abs) (our local atmospheric pressure, at altitude 1,300 m) and 3.0 atm abs, while the subjects breathed air, then oxygen. RESULTS: The P_{tcO_2} correlated with P_{aO_2} ($r^2 = 0.99$). Under all the conditions, the P_{tcO_2} values were lower than P_{aO_2} values by approximately 10%. The P_{tcCO_2} was 2–6 mm Hg higher than the P_{aCO_2}, but the correlation was low ($r^2 = 0.21$). CONCLUSIONS: The P_{tcO_2} in normal humans may be used to estimate the P_{aO_2}. The P_{tcCO_2} may not be an adequate reflection of the P_{aCO_2}. It is unknown if P_{tcO_2} and P_{tcCO_2} measurements in critically ill patients can replace P_{aO_2} and P_{aCO_2} measurements. *Key words: transcutaneous oxygen, transcutaneous carbon dioxide, P_{aO_2}, P_{aCO_2}, hyperbaric oxygen, blood gas monitoring.* [Respir Care 2007;52(11):1490–1496.

Introduction

At our institution we routinely measure arterial oxygen (P_{aO_2}) and carbon dioxide (P_{aCO_2}) tensions in critically ill patients who are treated with hyperbaric oxygen and who have indwelling arterial catheters. Our techniques for drawing arterial blood and the confidence of the P_{aO_2} and P_{aCO_2} measurements have been discussed previously.[1,2] These patients often have lung injury from aspiration, atelectasis, or inflammation, and at any alveolar oxygen tension their P_{aO_2} will be considerably lower than if they had normal lung function.[3]

We perform P_{aO_2} measurements to titrate the chamber pressure and positive end-expiratory pressure. For example, if the P_{aO_2} is $<$ 1,000 mm Hg at 2.0 atmospheres absolute (atm abs), we increase chamber pressure in an attempt to raise P_{aO_2} to between 1,000 mm Hg and 1,500 mm Hg. Similarly, we titrate positive end-expiratory pressure to achieve P_{aO_2} between 1,000 mm Hg and 1,500 mm Hg if the P_{aO_2} is $<$ 1,000 mm Hg at 3 atm abs while the patient is breathing 100% oxygen.[4]

We also monitor the P_{aCO_2} and titrate ventilation during hyperbaric oxygen therapy. Hypercapnia is a risk factor for central nervous system oxygen toxicity.[5] Critically ill patients often have increased alveolar dead space from lung pathology. We attempt to normalize the P_{aCO_2}, which can be challenging because of ventilator limitations.[6–8]

Most hyperbaric medicine centers do not monitor arterial blood gas tensions during hyperbaric oxygen. They

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may not have blood gas instrumentation nearby that can accurately measure hyperbaric P_{aO_2}, or the patient may not have an indwelling arterial catheter. However, many hyperbaric medicine centers do have the equipment to transcutaneously measure oxygen (P_{tcO_2}). Therefore, we decided to compare P_{tcO_2} and transcutaneously measured carbon dioxide (P_{tcCO_2}) measurements to simultaneously measured P_{aO_2} and P_{aCO_2} measurements in a group of normal healthy volunteers during hyperbaric air and oxygen breathing, to determine if P_{tcO_2} and P_{tcCO_2} measurements are suitable surrogates for P_{aO_2} and P_{aCO_2} measurements.

Methods

Ten healthy, nonsmoking, volunteer subjects with no contraindications for hyperbaric oxygen agreed to placement of radial artery catheters and transcutaneous measurement of oxygen and carbon dioxide (TINA, Radiometer, Copenhagen, Denmark).

Our institutional review board approved this study, and all the volunteers gave informed consent prior to participation. All the subjects had a radial artery catheter (Arrow International, Reading, Pennsylvania) placed by hospital-certified technicians who followed hospital protocols and used sterile technique. The transcutaneous sensors were calibrated and placed on healthy skin on the subject's chest, per the manufacturer's recommendations. The sensor temperatures were maintained at 44°C. Initial baseline measurements from the transcutaneous sensor were obtained after 30 min. Arterial blood samples were collected and analyzed immediately in a blood gas analyzer (ABL330, Radiometer, Copenhagen, Denmark) with techniques described previously.[1,2] The P_{aO_2}, P_{aCO_2}, P_{tcO_2}, and P_{tcCO_2} values were recorded at baseline conditions, then at each hyperbaric compression condition.

The subjects were compressed with air in a monoplace hyperbaric chamber (model 2500B, Sechrist Industries, Anaheim, California) to 3.0, 2.5, 2.0, and 1.12 atm abs. Each chamber pressure was maintained for 10 min, arterial blood was sampled, and then the chamber pressure was lowered to the new pressure. The subjects then breathed room air at 0.85 atm abs (our local atmospheric pressure at 1,300 m altitude) for 30 min after the air compression. The subjects then breathed 100% oxygen, delivered via a demand regulator and mouthpiece, with a nose clip, for 30 min prior to compression with oxygen, to accelerate residual nitrogen elimination. They continued to breathe oxygen via the regulator, until the fractional oxygen concentration within the hyperbaric chamber was > 0.95. The subjects were then compressed to an identical profile with 100% oxygen, followed by 10 min breathing oxygen and 30 min breathing air at our local atmospheric pressure (0.85 atm abs).

We used 10-min intervals at each chamber pressure of inquiry, because in normal subjects we had observed that the transcutaneously measured oxygen and carbon dioxide values reached asymptotic values in 10 min. In addition, others have used 10 min as the time necessary to reach equilibrium.[9–11] In an experiment that addressed the time to equilibrium, the time for P_{tcO_2} to reach a plateau was 5–7 min.[12] Since the subjects incurred a decompression stop from their hyperbaric air exposures, we did not wish to lengthen the intervals under each condition until sample acquisition. If these intervals had been longer than 10 min, the decompression schedule would have been lengthened. We used 1.12 atm abs pressure while breathing air as a decompression period for our altitude (1,300 m), using Cross-corrections for altitude air decompression.[13]

For control purposes, we placed the transcutaneous sensors on the chest of a healthy volunteer and on a ceramic surface, and we inverted a sensor, placed an open tube over the adhesive ring, and filled the area with 1 mL of saline (open to the ambient atmosphere). P_{tcO_2} and P_{tcCO_2} values were recorded from atmospheric pressure (0.85 atm abs) to 2.5 atm abs, with calibration to room air at 0.85 atm abs, then exposed to chamber pressure with the chamber filled with 100% O_2. The volunteer made the adhesive seal leak when directed to do so.

We analyzed the data with mixed-effects regression for both P_{tcCO_2} and P_{tcO_2}, and allowed for different intercepts and slopes for each subject. When appropriate, the data are expressed as mean ± 1 standard deviation.

Results

Oxygen comparisons, by individual, are shown in Figure 1. For individual subjects, the regression lines were similarly oriented but had slightly different slopes ($p < 0.001$) (Fig. 2). The data are also presented in Bland-Altman plots[14] (Fig. 3). The transcutaneous oxygen measurements correlated highly with the P_{aO_2} (Fig. 4). The $P_{tcO_2} = -2.1 + 0.9\ P_{aO_2}$ ($r^2 = 0.99$, $p < 0.001$). While compressed with air at 2.5 atm abs, the P_{tcO_2} values of the group were approximately 50 mm Hg lower than the P_{aO_2} (Fig. 5). While breathing 100% oxygen between 3 atm abs and 2 atm abs, the P_{tcO_2} measurements were approximately 150 mm Hg lower than the P_{aO_2} values (see Fig. 5).

Carbon dioxide comparisons, by individual, are shown in Figure 6. For individual subjects the regression lines were strikingly different ($p < 0.001$) (Fig. 7). The data are also presented in Bland-Altman plots[14] (Fig. 8). Transcutaneous carbon dioxide measurements correlated poorly with P_{aCO_2} values ($r^2 = 0.21$) (Fig. 9). The P_{tcCO_2} overestimated the P_{aCO_2}, particularly at 3 atm abs while breathing 100% oxygen (Fig. 10).

For the control experiment with the healthy volunteer, the P_{tcO_2} and P_{tcCO_2} were physiologic, with values within

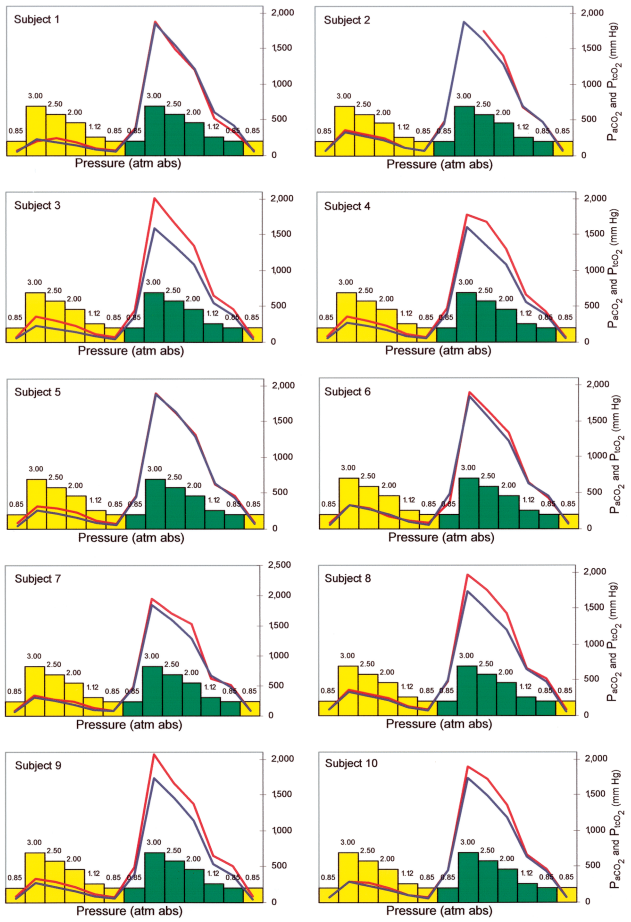

Fig. 1. Arterial oxygen tension measured from arterial blood samples (P_{aO_2}) (red lines) and transcutaneously measured oxygen tension (P_{tcO_2}) (blue lines) values from the individual subjects at each condition. Yellow denotes air breathing. Green denotes 100% oxygen breathing. 1.12 atm abs was chosen as a decompression stop for the prior air pressurization at our altitude (1,300 m). Measurements were obtained after 10 min at each pressure, except initially, the air period at 0.85 atm abs following hyperbaric air compression and 100% oxygen at 0.85 atm abs prior to compression on oxygen. Under those conditions the time to measurement was 30 min.

the expected ranges. When the volunteer caused the adhesive seal to leak, immediately the P_{tcO_2} rose to values close to the ambient O_2 level in the chamber and the P_{tcCO_2} fell to a nonphysiologic level (< 10 mm Hg) (Table 1). After re-sealing the adhesive, the P_{tcO_2} dropped to the physiologic, expected level and the P_{tcCO_2} rose back to a physiologic level.

For the control experiments with a sensor placed on a ceramic surface, the O_2 values were 140 mm Hg (predicted value at 0.85 atm abs = 134 mm Hg), from 0.85 atm abs to 2.5 atm abs. The CO_2 values were zero from 0.85 atm abs to 2.5 atm abs (see Table 1).

For the control experiments with a sensor inverted and covered with 1 mL of saline and open to the chamber

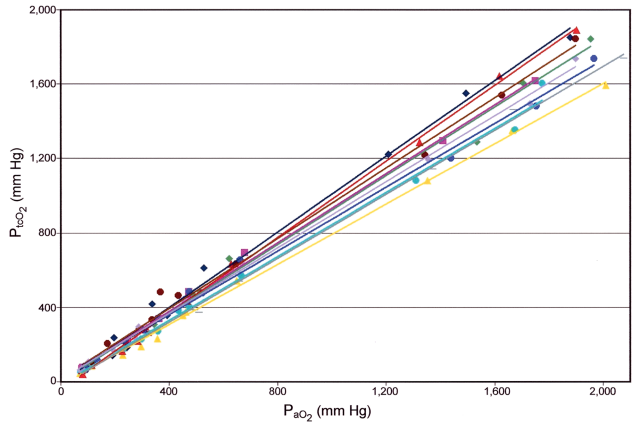

Fig. 2. Transcutaneously measured oxygen tension (P_{tcO_2}) versus arterial oxygen tension measured from arterial blood samples (P_{aO_2}), with regression lines, by subject. When we take individual subjects into account, the regression lines are similarly oriented but had slightly different slopes ($p < 0.001$).

atmosphere, the O_2 value at 0.85 atm abs (air) was 140 mm Hg and the CO_2 value was zero. At 2.0 atm abs the O_2 value was 1,582 mm Hg, and at 2.5 atm abs it was 1,980 mm Hg (predicted at 2.0 atm abs = 1,520 mm Hg; predicted at 2.5 atm abs = 1,900 mm Hg). The CO_2 value remained zero from 0.85 atm abs to 2.5 atm abs (see Table 1).

Discussion

In normal healthy subjects we found that chest P_{tcO_2} measurements correlated with P_{aO_2} measurements. However, that correlation may be misleading.[14,15] The Bland-Altman graph suggests that P_{tcO_2} does not accurately reflect P_{aO_2}, especially during hyperbaric oxygen conditions. Nevertheless, under all conditions measured, the P_{tcO_2} values were lower than P_{aO_2} values, but the magnitude of

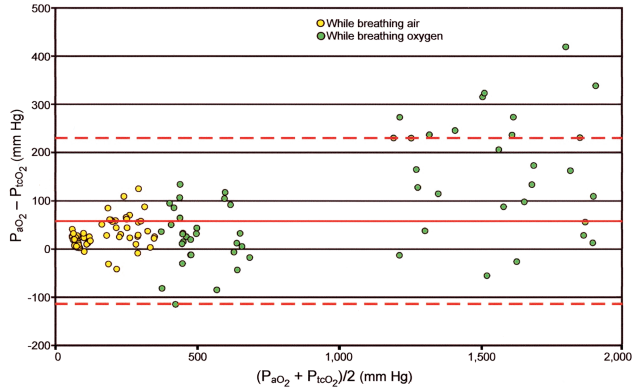

Fig. 3. Bland-Altman plot of arterial oxygen tension measured from arterial blood samples (P_{aO_2}) and transcutaneously measured oxygen tension (P_{tcO_2}). The limits of agreement are the mean difference ± 1.96 standard deviations.

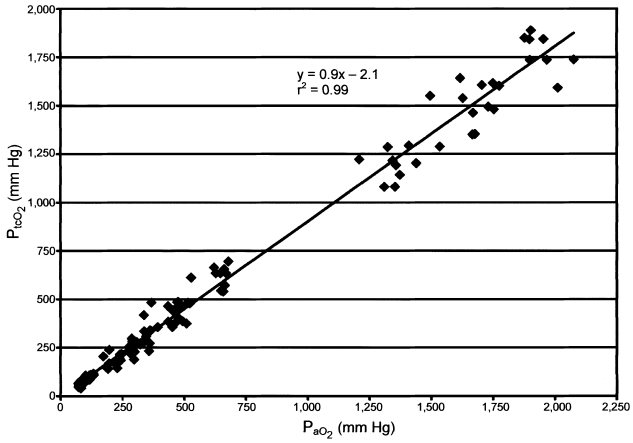

Fig. 4. Transcutaneously measured oxygen tension (P_{tcO_2}) versus arterial oxygen tension measured from arterial blood samples (P_{aO_2}).

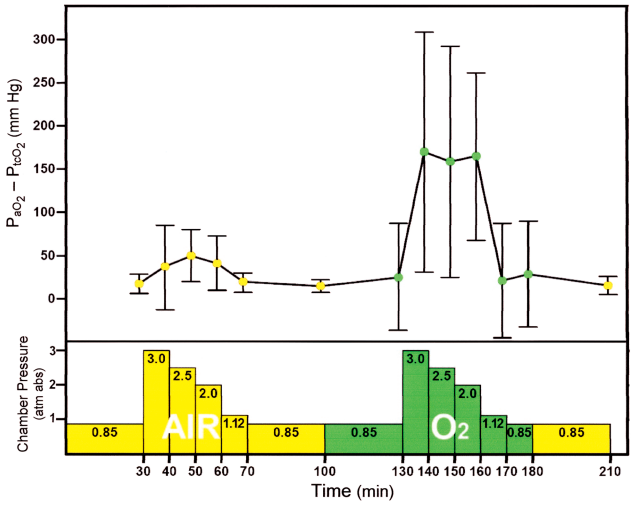

Fig. 5. Difference graph of arterial oxygen tension measured from arterial blood samples (P_{aO_2}) minus transcutaneously measured oxygen tension (P_{tcO_2}). Yellow denotes air breathing. Green denotes 100% oxygen breathing. The measurements were obtained after 10 min at each pressure, except initially, the air period at 0.85 atm abs following hyperbaric air compression and 100% oxygen at 0.85 atm abs prior to compression on oxygen. Under those conditions the time to measurement was 30 min. Data are expressed as mean ± 1 standard deviation.

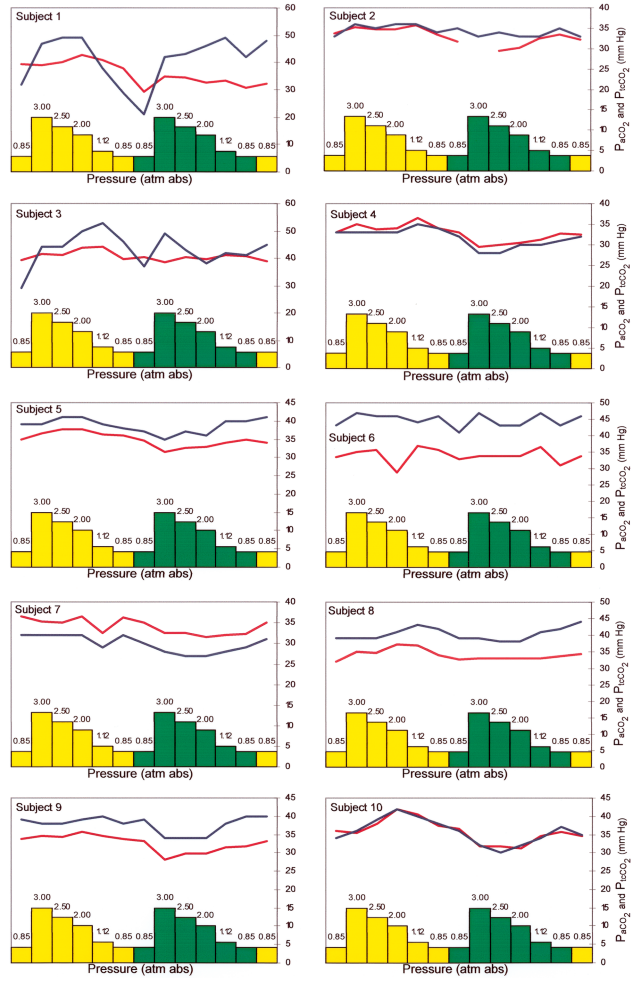

Fig. 6. Arterial carbon dioxide tension measured from arterial blood samples (P_{aCO_2}) (red lines) and transcutaneously measured carbon dioxide tension (P_{tcCO_2}) (blue lines) values from individual subjects at each condition. Yellow denotes air breathing, green denotes 100% oxygen breathing. 1.12 atm abs was chosen as a decompression stop for the prior air pressurization at our altitude (1,300 m). The measurements were obtained after 10 min at each pressure, except initially, the air period at 0.85 atm abs following hyperbaric air compression and 100% oxygen at 0.85 atm abs prior to compression on oxygen. Under those conditions the time to measurement was 30 min.

differences were relatively small (< 10%). Although Brown et al did not test under hyperbaric conditions, they also found that the P_{tcO_2} was lower than the predicted P_{aO_2} with normal subjects breathing oxygen at sea level pressure.[12] They found $P_{tcO_2} = 0.78 \times P_{aO_2} + 1.65$ ($r = 0.99$, $p < 0.01$, $n = 20$), and the difference between the transcutaneous oxygen measurements and arterial oxygen tensions was $P_{tcO_2} - P_{aO_2} = 0.22\ P_{aO_2} - 1.65$ ($r = 0.93$, $p < 0.01$, $n = 20$).[12]

Our results suggest that P_{tcO_2} measurements in normal humans could be used to crudely estimate the P_{aO_2}. Measuring the P_{aO_2} or the P_{tcO_2} in patients with normal cardiopulmonary systems is unnecessary and unimportant. However, measuring the P_{aO_2} or the P_{tcO_2} in patients with compromised lung or heart function may be important. For any given patient treated at a specified chamber pressure there will be a wide range of P_{aO_2} values, dependent upon whether the patient's lungs are normal or dysfunctional, and the P_{aO_2} of these patients cannot be predicted based upon sea level P_{aO_2} measurements.[3] In addition, hypoxemia can occur while breathing air under hyperbaric conditions, and during these air-breathing periods, P_{aO_2} monitoring may be important.[16] If the P_{aO_2} during hyperbaric

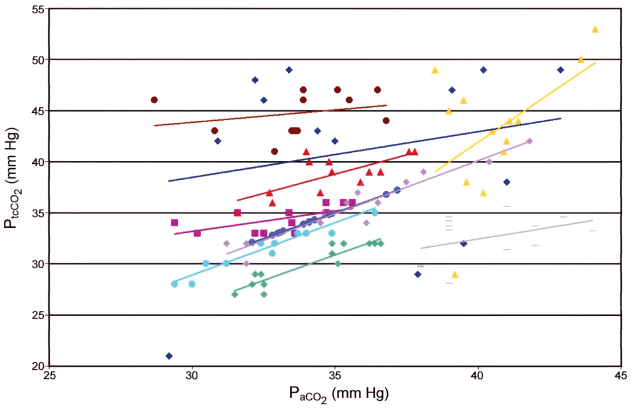

Fig. 7. Transcutaneously measured carbon dioxide tension (P_{tcCO_2}) versus arterial carbon dioxide tension measured from arterial blood samples (P_{aCO_2}), with regression lines, by subject. The regression lines are strikingly different ($p < 0.001$).

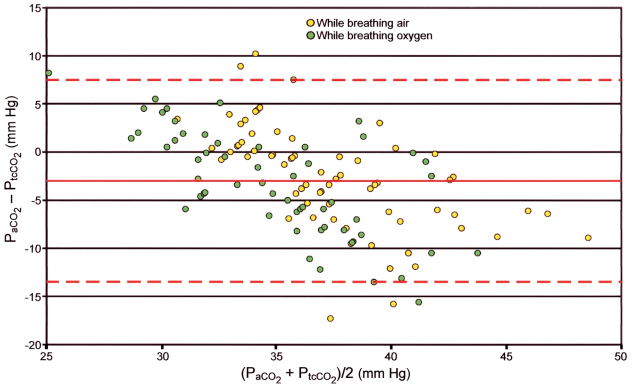

Fig. 8. Bland-Altman plot of arterial carbon dioxide tension measured from arterial blood samples (P_{aCO_2}) and transcutaneously measured carbon dioxide tension (P_{tcCO_2}). The limits of agreement are mean difference ± 1.96 standard deviations.

oxygen is considerably lower than expected, the risk versus the benefit of therapy should be considered. Since hyperbaric P_{aO_2} monitoring is not practical or possible at some hyperbaric services, monitoring the P_{tcO_2} during hyperbaric oxygen therapy, especially during air-breathing periods, may be helpful. For example, P_{tcO_2} monitoring of a child with gas embolism prompted measuring the P_{aO_2}, which demonstrated hypoxemia.[16]

In patients with normal chest reference sites who do not have edema or microvascular derangements, the P_{tcO_2} would be expected to be similar to our results. However, in patients the P_{tcO_2} may or may not correlate with P_{aO_2} measurements. If the patient has chest wall edema, microvascular blood-flow abnormalities, perfusion abnormalities from severe sepsis, fluid resuscitation, or use of vasoactive drugs, the P_{tcO_2} may not correlate with the P_{aO_2}.[12] Hyperoxic vasoconstriction could also reduce the P_{tcO_2} values, compared to the P_{aO_2}.[17]

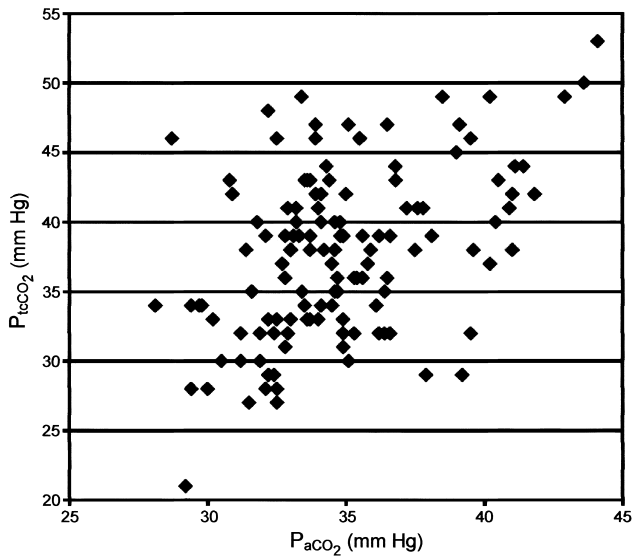

Fig. 9. Transcutaneously measured carbon dioxide tension (P_{tcCO_2}) versus arterial carbon dioxide tension measured from arterial blood samples (P_{aCO_2}).

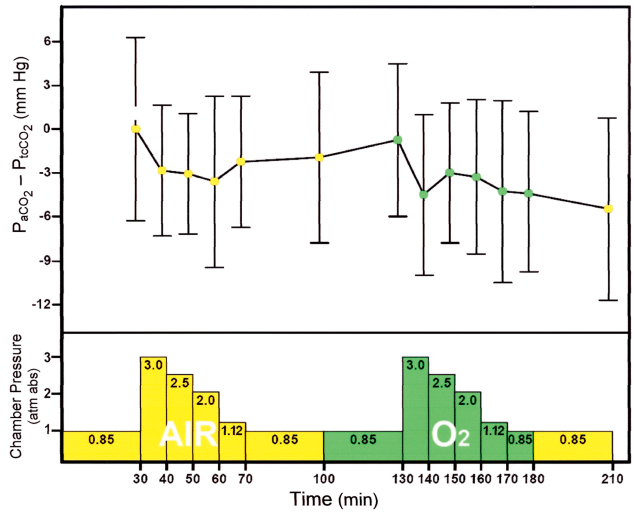

Fig. 10. Difference graph of arterial carbon dioxide tension measured from arterial blood samples (P_{aCO_2}) minus transcutaneously measured carbon dioxide tension (P_{tcCO_2}). Yellow denotes air breathing. Green denotes 100% oxygen breathing. The measurements were obtained after 10 min at each pressure, except initially, the air period at 0.85 atm abs following hyperbaric air compression and 100% oxygen at 0.85 atm abs prior to compression on oxygen. Under those conditions the time to measurement was 30 min. Data are expressed as mean ± 1 standard deviation.

The P_{tcO_2} values may have been lower than the P_{aO_2} values because the 10-min stabilization time at each condition did not permit transcutaneous oxygen equilibrium. We chose 10 min at each test condition for arterial blood to reach gaseous equilibrium.[9–12] Also, the interval that subjects could remain at each test condition was limited by the amount of time the subjects could tolerate the exper-

Table 1. Measurements From Control Experiments

Probe Location and/or Status	Chamber Pressure (atm abs)		
	0.85	2.0	2.5
Probe open to chamber			
P_{tcO_2}			
Predicted	134	1,520	1,900
Measured	140	1,582	1,980
P_{tcCO_2}			
Predicted	0	0	0
Measured	0	0	0
Probe on ceramic surface			
P_{tcO_2}			
Predicted	134	134	134
Measured	140	140	140
P_{tcCO_2}			
Predicted	0	0	0
Measured	0	0	0
Probe on healthy volunteer			
P_{tcO_2}			
Predicted	134	1,520	ND
Measured	86	1,458	ND
P_{tcCO_2}			
Predicted	38	36	ND
Measured	27	27	ND
Leaking probe on healthy volunteer			
P_{tcO_2}			
Predicted	NA	1,520	ND
Measured	NA	1,500	ND
P_{tcCO_2}			
Predicted	NA	36	ND
Measured	NA	6	ND

P_{tcO_2} = transcutaneously measured partial pressure of oxygen
P_{tcCO_2} = transcutaneously measured partial pressure of carbon dioxide
NA = not applicable
ND = no data collected

iment and the decompression risk and decompression period necessary for the hyperbaric air exposures.

In these normal healthy subjects we found that chest P_{tcCO_2} correlated poorly with P_{aCO_2} measurements, and P_{tcCO_2} values were not interchangeable with P_{aCO_2} values. In some individuals the P_{tcCO_2} correlated well with the P_{aCO_2}, but in other subjects there was no correlation, for reasons that are unclear. The Bland-Altman graph shows the lack of relationship between the P_{aCO_2} and the P_{tcCO_2}.

When they compared P_{tcCO_2} to P_{aCO_2} at sea level pressure, Nishiyama et al also found that P_{tcCO_2} exceeded the P_{aCO_2}.[18] The P_{tcCO_2} may exceed the P_{aCO_2} because of the effect of inhalation of high partial pressure of oxygen under the experimental conditions. Under hyperoxic breathing there is less desaturation of hemoglobin, which results in higher amounts of dissolved carbon dioxide in tissue and blood. In addition, the P_{tcCO_2} increases by 4% for every 1°C rise in electrode temperature[19] and there is increased CO_2 production by the skin, which can also increase the P_{tcCO_2}.[20] Therefore, the P_{tcCO_2} is expected to be higher than the P_{aCO_2}.[16] In patients, the P_{tcCO_2} could be influenced by underlying disease, tissue edema, and use of vasoactive drugs, so it is doubtful that P_{tcCO_2} measurements could replace P_{aCO_2} measurements. Nevertheless, a "semi-physiologic" P_{tcCO_2} value gives evidence that the P_{tcO_2} value has not been influenced by a leaking seal.

The control experiments confirm that the sensor seals did not leak during our study and that the sensors were not influenced by the chamber atmosphere gas pressures. We therefore conclude that our data were not influenced by artifact.

Conclusions

In normal human subjects the P_{tcO_2} measurements during hyperbaric oxygen exposure seem to be reasonable surrogates for P_{aO_2} measurements. The P_{tcCO_2} measurements may not agree with P_{aCO_2} and should not be used as surrogates for the P_{aCO_2}. It is unknown if P_{tcO_2} and P_{tcCO_2} measurements in critically ill patients can replace P_{aO_2} and P_{aCO_2} measurements, respectively.

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REFERENCES

1. Weaver LK, Howe S, Berlin SL. Normobaric measurement of oxygen tension of blood and saline tonometered under hyperbaric oxygen conditions. J Hyperbaric Med 1990;5(1):29–38.
2. Weaver LK, Howe S. Normobaric measurement of arterial oxygen tension in subjects exposed to hyperbaric oxygen. Chest 1992;102(4): 1175–1181.
3. Weaver LK, Howe S. Arterial oxygen tension of patients with abnormal lungs treated with hyperbaric oxygen is greater than predicted. Chest 1994;106(4):1134–1139.
4. Churchill S, Weaver LK. Unexpected arterial oxygen tension (PaO2) with super PEEP during HBO2 therapy. Undersea Hyperb Med 2001;28 (Suppl):38.
5. Clark JM, Thom SR. Oxygen under pressure. In: Brubakk AO, Neuman TS, editors. Bennett and Elliott's physiology and medicine of diving, 5th ed. Cornwall: Elsevier Science 2003;358–418.
6. Weaver LK, Greenway L, Elliott CG. Performance of the Sechrist 500A hyperbaric ventilator in a monoplace hyperbaric chamber. J Hyperbaric Med 1988;3(4):215–225.
7. Churchill S, Weaver LK, Haberstock D. Performance of the Omnivent mechanical ventilator for use with the monoplace hyperbaric chamber. Undersea Hyperb Med 1999;26(Suppl):70–71.
8. Weaver LK. Management of critically ill patients in the monoplace hyperbaric chamber. In: Kindwall EP, Whelan HT, editors. Hyperbaric medicine practice, 2nd ed. Flagstaff: Best;1999;245–294.

9. Huch A, Huch R, Hollmann G, Hockerts T, Keller HP, Seller D. Transcutaneous P_{O2} of volunteers during hyperbaric oxygenation. Biotelemetry 1977;4(2):88–100.
10. Wattel FE, Mathieu DM, Rossati P, Neviere RR, Coget JM. Hyperbaric oxygen in the treatment of diabetic foot lesions. J Hyperbaric Med 1991;6(4):263–268.
11. Fife CE, Buyukcakir C, Otto GH, Sheffield PJ, Warriner RA, Love TL, Mader J. The predictive value of transcutaneous oxygen tension measurement in diabetic lower extremity ulcers treated with hyperbaric oxygen therapy: a retrospective analysis of 1,144 patients. Wound Repair Regen 2002;10(4):198–207.
12. Brown JT, Schur MS, McClain BC, Kafer ER. In vivo response time of transcutaneous oxygen measurement to changes in inspired oxygen in normal adults. Can Anaesth Soc J 1984;31(1):91–96.
13. Bell RL, Borgwardt RE. The theory of high-altitude corrections to the US Navy standard decompression tables: the cross corrections. Undersea Biomed Res 1976;3(1):1–23
14. Bland JM, Altman DG. Comparing methods of measurement: why plotting difference against standard method is misleading. Lancet 1995;346(8982):1085–1087.
15. Bland JM, Altman DG. Measuring agreement in method comparison studies. Stat Methods Med Res 1999;8(2):135–160.
16. Weaver LK, Churchill S. Hypoxemia with air breathing periods in US Navy treatment table 6. Undersea Hyperb Med 2006;33(1):11–15.
17. Ratzenhofer-Komenda B, Kovac H, Smolle-Jüttner FM, Friehs GB, Schwarz G. Quantification of the dermal vascular response to hyperbaric oxygen with laser-Doppler flowmetry. Undersea Hyperb Med 1998;25(4):223–227
18. Nishiyama T, Nakamura S, Yamashita K. Comparison of the transcutaneous oxygen and carbon dioxide tension in different electrode locations during general anaesthesia. Eur J Anaesthesiol 2006;23(12):1049–1054.
19. Franklin ML. Transcutaneous measurement of partial pressure of oxygen and carbon dioxide. Respir Care Clin North Am 1995;1(1):119–131.
20. Wimberley PD, Grønlund-Pedersen K, Olsson J, Siggaard-Abderson O. Transcutaneous carbon dioxide and oxygen tension measured at different temperatures in healthy adults. Clin Chem 1985;31(10):1611–1615.