Transcutaneous Measurement of Carbon Dioxide Tension During Extended Monitoring: Evaluation of Accuracy and Stability, and an Algorithm for Correcting Calibration Drift

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BACKGROUND: When polysomnography is indicated in a patient with a presumed sleep disorder, continuous monitoring of arterial carbon dioxide tension (P_{aCO_2}) is desirable, especially if nocturnal hypoventilation is suspected. Transcutaneous CO_2 monitors (P_{tcCO_2}) provide a noninvasive correlate of P_{aCO_2} , but their accuracy and stability over extended monitoring have been considered inadequate for the diagnosis of hypoventilation. We examined the stability and accuracy of P_{tcCO_2} measurements and the performance of a previously described linear interpolation technique designed to correct for calibration drift. **METHODS:** We compared the P_{tcCO_2} values from 2 TINA TCM-3 monitors to P_{aCO_2} values from arterial blood samples obtained at the beginning, every 15 min of the first hour, and then hourly over 8 hours of monitoring in 6 hemodynamically stable, male, intensive care patients (mean age 46 ± 17 y). **RESULTS:** Time had a significant ($P = .002$) linear effect on the P_{tcCO_2} - P_{aCO_2} difference, suggesting calibration drift over the monitoring period. We found no differences between monitor type or interaction between time and monitor type. For the 2 monitors the uncorrected bias was 3.6 mm Hg and the limits of agreement were -5.1 to 12.3 mm Hg. Our linear interpolation algorithm improved the bias and limits of agreement to 0.4 and -5.5 to 6.4 mm Hg, respectively. **CONCLUSIONS:** Following stabilization and correction for both offset and drift, P_{tcCO_2} tracks P_{aCO_2} with minimal residual bias over 8 hours of monitoring. Should future research confirm these findings, then interpolated P_{tcCO_2} may have an increased role in detecting sleep hypoventilation and assessing the efficacy of treatment. *Key words:* carbon dioxide monitoring; polysomnography. [Respir Care 2011;56(4):442–448. © 2011 Daedalus Enterprises]

Introduction

When polysomnography is indicated in a patient with a presumed sleep disorder, continuous monitoring of arterial carbon dioxide tension (P_{aCO_2}) is desirable, especially if nocturnal hypoventilation is suspected. Arterial blood sampling provides direct assessment of blood gas tensions, but it is an invasive, expensive, potentially sleep-disruptive measure, and provides only periodic data. In contrast, transcutaneous monitoring of P_{CO_2} (P_{tcCO_2}) provides continuous data and is noninvasive. Transcutaneous CO_2 monitors employ a skin-surface Stow-Severinghaus electrode to measure CO_2 after heating and “arterialization” of the underlying tissue.

Although continuous P_{aCO_2} measurement is desirable in the monitoring and treatment of hypoventilation, transcu-

taneous CO_2 monitoring is specifically not recommended for the quantification of hypoventilation during a sleep study.¹ The limits of agreement between P_{aCO_2} and P_{tcCO_2} are considered too imprecise, particularly for extended monitoring where there is increased potential for electrode calibration drift. The methods used to quantify the change in P_{tcCO_2} - P_{aCO_2} difference (electrode drift) during sleep are not well established, and previous research has not clearly established whether electrode drift is systematic and therefore amenable to quantification and correction. Despite these limitations, both the absolute P_{tcCO_2} value and the P_{tcCO_2} change during sleep are frequently reported as a clinical measure of both disease severity and treatment efficacy, particularly in studies of the outcome of noninvasive ventilation.²⁻⁷ Although the minimum clinically important change that P_{tcCO_2} monitoring aims to measure is

not known, current consensus guidelines suggest that a rise of ≥ 10 mm Hg in P_{aCO_2} indicates sleep hypoventilation,¹ and recent publications have suggested that 7.6 mm Hg should be the accepted minimum clinically important difference between measured P_{aCO_2} and P_{tcCO_2} .^{8,9}

To better understand the relationships between P_{tcCO_2} and P_{aCO_2} , we assessed the stability and accuracy of P_{tcCO_2} readings from 2 TINA TCM-3 monitors (Radiometer Medical, Brønshøj, Denmark) during extended monitoring, and determined the limits of agreement between P_{tcCO_2} and P_{aCO_2} measured from arterial blood, to characterize the monitors' electrode drift over 8 hours. In our institution the accuracy of the P_{tcCO_2} values during a sleep study is established with an *in vivo* calibration against arterial blood samples taken at the start of the monitoring period. Any observed P_{tcCO_2} - P_{aCO_2} difference thereafter is assumed to behave as a fixed offset and to remain constant. A second arterial sample is taken at the end of the monitoring period, and the observed change in the P_{tcCO_2} - P_{aCO_2} difference is assumed to reflect electrode drift. To assess the validity of this procedure we also assessed the utility of the previously published methods for correction of offset and electrode drift in P_{tcCO_2} values.¹⁰

Methods

This research was conducted at Austin Hospital, Heidelberg, Australia. The study protocol was approved by the

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Human Ethics Committee of the Austin and Repatriation Medical Centre. All patients or their authorized representatives gave informed consent.

Subjects

It was considered unethical to conduct arterial puncture in otherwise healthy people undergoing a routine sleep study, so we recruited stable patients in our intensive care unit (ICU) to serve as a model for people in the sleep laboratory. We selected ICU patients because they typically have an indwelling arterial catheter. The potential impact of tissue hypoperfusion on the results was minimized by excluding patients on inotropic support, with temperature greater than 37.0°C, or who just had cardiac arrest or surgery.

Transcutaneous Carbon Dioxide Monitor

We measured P_{tcCO_2} simultaneously with 2 TINA TCM-3 monitors (Monitor 1 and Monitor 2). The TCM-3 monitor employs a dual transcutaneous carbon dioxide and oxygen electrode. We used a new transcutaneous electrode membrane for each study. The electrodes were re-membraned 12–24 hours prior to each study. The probes were heated to 43°C and the analog outputs recorded continuously on chart paper and were calibrated with the devices' 1-volt internal test signal.

The P_{tcCO_2} monitors were calibrated according to the manufacturer's instructions, using a one-point dry-gas calibration with 5% CO_2 . The skin was shaved if required, cleansed with alcohol, and dried. The P_{tcCO_2} electrodes were positioned approximately 2 cm apart on the patient's anterior chest wall, and attached with their respective adhesive rings and contact solutions. Additional tape was applied to secure the electrodes and minimize the risk of movement or dislodgment. The usual nursing, medical, and physiotherapy care of the patients was not altered during the monitoring period.

Arterial Blood Sampling

Arterial blood samples were obtained via a 3-way tap connected to the arterial line. Arterial samples were taken at the beginning, every 15 min for the first hour, and then hourly during the monitoring period. The clinical care of participants was managed by the usual ICU care team, but attention was given to ensure that the participants had no change in ventilation made immediately prior to any sample being drawn. Furthermore, all samples were drawn during periods of steady-state breathing and recording (defined as no P_{tcCO_2} change > 2 mm Hg for at least 3 min and no apparent respiratory instability associated with rolling over or with gross body movements). We recorded the

time of each arterial sample and the P_{tCO_2} value at that time of the arterial sample.

The accuracy of the blood-gas analyzer (ABL500, Radiometer Medical, Brønshøj, Denmark) was confirmed with standard quality-control solutions. Whole-blood tonometry was also performed twice during each study period to verify the accuracy of the blood-gas analyzer. The arterial blood samples were immediately placed in an ice-and-water slurry, and analyzed (in duplicate) within 20 min of drawing. If the 2 P_{aCO_2} values were more than 1 mm Hg different, a third P_{aCO_2} measurement was made. We used the average of the 2 closest P_{aCO_2} values.

Method for Correcting Electrode Drift and Offset

For each patient we matched the P_{aCO_2} values obtained 15 min after electrode application with the corresponding P_{tCO_2} and used the difference as the initial measurement of electrode offset. The P_{aCO_2} measurements at 8 hours were used as the final in vivo calibration points for the simultaneous P_{tCO_2} measurement. We used changes in that difference as an estimate of electrode calibration drift. We corrected the P_{tCO_2} values by subtracting the baseline offset alone, and then by subtracting both the baseline offset and the drift over the 8 hours.¹⁰ A spreadsheet example of the correction process is available in the supplementary materials at <http://www.rcjournal.com>.

Statistical Analysis

Given the potential for variable or systematic P_{tCO_2} - P_{aCO_2} differences, we employed several strategies to analyze P_{tCO_2} offset and drift. We analyzed for offset and drift with linear regression of each subject's P_{tCO_2} recording. We analyzed the P_{tCO_2} - P_{aCO_2} differences and patterns of P_{tCO_2} change (drift) in the group data with repeated-measures analysis of variance, incorporating polynomial trend analysis to investigate systematic linear versus higher-order polynomial effects over time. We made post hoc pair-wise comparisons of mean differences at each time, incorporating a Bonferroni correction for multiple comparisons. We examined the benefit of using corrected values with a linear mixed-effects model analysis,¹¹ with the P_{tCO_2} - P_{aCO_2} difference as the response, subject as a random effect with separate intercept, and monitor (Monitor 1 or Monitor 2) and correction type (uncorrected, corrected for baseline only, and corrected for both baseline and drift) as factors. We conducted the analyses with statistics software (PASW Statistics 18.0, SPSS, Chicago, Illinois), and we ran the fully saturated model first, followed by removal of nonsignificant interaction terms. We also determined instrument bias (average) and limits of agreement (1.96 standard deviations) in the group data, according to the methods of Bland and Altman.¹² Results are presented

Fig. 1. P_{aCO_2} and transcutaneously measured P_{CO_2} (P_{tCO_2}) measured with 2 P_{tCO_2} monitors, over 8 hours in 6 subjects.

as mean $\pm$ SD unless otherwise indicated. $P < .05$ was regarded as significant.

Results

We recruited 6 male patients (mean age 46 ± 17 y, range 26–72 y), whose diagnoses were trauma, post-neurosurgery, C3/4 spinal injury, C4/5 spinal injury, subarachnoid hemorrhage, and asthma. Four subjects were intubated and sedated to various degrees, but none were pharmacologically paralyzed. The mean P_{aCO_2} of all 72 arterial samples was 45.6 ± 12.8 mm Hg (range 31.9–77.7 mm Hg). All samples were drawn at the scheduled sample times, and all the recording periods were 8 hours. We found no cutaneous irritation or burning associated with the electrodes.

Figure 1 shows the P_{aCO_2} and P_{tCO_2} values, and Figure 2 shows the P_{tCO_2} - P_{aCO_2} differences. Table 1 shows the minimum and maximum P_{aCO_2} , drift, and intercept data, and the linear regression correlation coefficients. In the individual recordings, statistically significant baseline offset and linear drift were common: each affected 5 of 12 records (see Table 1), and 4 of 6 patients had drift of

Fig. 2. Raw and corrected differences between P_{aCO_2} and transcutaneously measured P_{CO_2} (P_{tcCO_2}) measured with 2 P_{tcCO_2} monitors, over 8 hours in 6 subjects.

Table 1. P_{aCO_2} , Drift, Intercept, and Slope Data

Subject	Diagnosis	P_{aCO_2} (mm Hg)		Drift* (mm Hg)		Intercept† (mm Hg)		Slope‡ (mm Hg/h)		r^2 ‡	
		Min	Max	Monitor 1	Monitor 2	Monitor 1	Monitor 2	Monitor 1	Monitor 2	Monitor 1	Monitor 2
1	Multiple trauma	42.1	50.9	12.3§	2.3	2.1 ± 2.1	4.3 ± 2.2	$1.2 \pm 0.5 $	0.3 ± 0.5	0.483	0.056
2	Post-neurosurgery	31.9	39.5	0.3	1.3	-1.5 ± 0.7	-1.3 ± 0.6	0.2 ± 0.2	0.2 ± 0.1	0.160	0.273
3	C3/4 spinal injury	40.7	48.3	7.1§	0.1	$-1.8 \pm 0.7 $	1.5 ± 0.7	$1.3 \pm 0.2 $	0.3 ± 0.2	0.905	0.245
4	C4/5 spinal injury	36.7	46.5	3.1	8.1§	0.2 ± 0.3	$3.9 \pm 0.4 $	$0.2 \pm 0.1 $	$0.8 \pm 0.1 $	0.539	0.913
5	Subarachnoid hemorrhage	33.8	37.0	0.6	3.6	-0.6 ± 0.6	$1.4 \pm 0.4 $	0.0 ± 0.1	$0.3 \pm 0.1 $	0.003	0.450
6	Asthma	53.5	77.6	7.7§	-2.3	$14.4 \pm 1.4 $	$7.5 \pm 1.1 $	-0.5 ± 0.3	-0.1 ± 0.2	0.269	0.033

* Drift is the magnitude of the difference between transcutaneously measured P_{CO_2} (P_{tcCO_2}) and P_{aCO_2} at the beginning and end of the recording period.

† Intercept and slope are coefficients $\pm$ standard error of the estimate.

‡ r^2 calculated from linear regression of the P_{tcCO_2} - P_{aCO_2} difference over time in each subject.

§ Values where the observed drift was higher than half of the reported minimal clinically important difference of 7.6, which highlights the need for drift and offset correction when examining P_{tcCO_2} data.

|| Difference statistically significant.

> 4 mm Hg in at least one monitor. In the group data there was a significant positive intercept ($P = .01$) and linear effect of time on the P_{tcCO_2} - P_{aCO_2} difference ($P = .002$), indicating systematic bias and drift, increasing on average from 1.7 ± 1.6 mm Hg at baseline to 5.4 ± 3.5 mm Hg at the conclusion of the recording period. No higher-order polynomial effects were observed, which supports the finding that the drift was predominantly linear. There was no difference in overall performance between the 2 monitors in raw P_{tcCO_2} - P_{aCO_2} difference scores ($P = .82$) and no significant interaction between time and monitor type ($P = .97$).

Offset and drift correction decreased the bias and limits of agreement of the P_{tcCO_2} - P_{aCO_2} difference (see Figs. 2

and 3). Mixed-model analysis showed statistically significant main effects of time ($P < .001$), monitor ($P = .01$), and correction type ($P < .001$), and a significant monitor-by-correction interaction ($P = .01$), but no further interaction effects. The monitor main and interaction effects indicated that, overall, Monitor 1 had greater bias than Monitor 2 (2.4 ± 1.5 mm Hg vs 1.6 ± 1.5 mm Hg), $P = .01$, in post hoc tests, revealing greater residual bias in Monitor 1 than in Monitor 2 with a baseline correction alone, but no further differences between the monitors. The correction-type effect showed incremental reductions in P_{tcCO_2} - P_{aCO_2} difference with each correction, and that the combined correction was superior. Overall bias was reduced by 1.7 ± 1.1 mm Hg with baseline correction

Fig. 3. Bland-Altman plot of raw and corrected (interpolated) P_{aCO_2} and transcutaneously measured P_{CO_2} (P_{tcCO_2}) (combined P_{tcCO_2} values from the 2 monitors). Before correction the bias is 4 mm Hg and the limits of agreement are -9 to 9 mm Hg. After correction the bias is less than 1 mm Hg, and the limits of agreement are -6 to 6 mm Hg.

alone ($P = .001$), and by a further 1.5 ± 0.9 mm Hg with combined baseline and drift correction ($P < .001$, Table 2).

Discussion

We have demonstrated that while P_{tcCO_2} monitoring can track trends in P_{aCO_2} during an extended (8-hour) monitoring period, significant electrode drift is common, but tends to be linear when P_{tcCO_2} is stable (ie, no change > 2 mm Hg over the preceding 3 min). Consequently, a 2-point in vivo calibration using the P_{tcCO_2} offset and drift, versus arterial P_{aCO_2} measurements at the beginning and end of recording, allowed for substantial compensation for P_{tcCO_2} drift. Simple baseline adjustment alone reduced bias, but ignores significant drift, and was inferior to 2-point baseline and drift correction. While the requirement for arterial blood measurements at the beginning and end of extended recording may limit the clinical utility of this

method, more frequent arterial sampling is often impractical, particularly in a sleep laboratory setting, where P_{tcCO_2} monitoring is common and potentially most useful.

We confirm that the accuracy and the agreement of the monitors was too imprecise for uncorrected P_{tcCO_2} measurements to substitute directly for P_{aCO_2} in adults, as has been previously described.^{4,13-18} The reasons for the observed differences between P_{aCO_2} and P_{tcCO_2} have previously been reviewed.¹⁹ However, we would attest that the aim of continuous CO_2 measurement during extended sleep monitoring is to detect sleep-related changes that suggest the presence or adequate control of hypoventilation, rather than as a direct measure of P_{aCO_2} per se (ie, monitoring changes during sleep, rather than measuring absolute values), so noninvasive devices that can detect change over an extended monitoring period with sufficient accuracy and minimal artifactual drift have substantial clinical utility.

Limitations

The generalizability of our findings is limited by our use of only one model of P_{tcCO_2} monitor and the small number and type of patients studied. However, the performance of the TINA-TCM3 monitors was similar to that in other studies that used the TINA-TCM3 monitor^{2,4} suggesting that our results are valid for this device. Although we studied only 6 participants, the P_{aCO_2} values ranged widely, from 31.9 mm Hg to 77.6 mm Hg, and that wide range has been suggested as desirable in studies such as this.⁴ As shown in Figure 3 and in previous literature,^{13,15,20} the correlation between P_{aCO_2} and P_{tcCO_2} decreases as P_{aCO_2} increases, particularly beyond 50 mm Hg, which reinforces the need for both offset and drift correction to maximize the utility of a continuous P_{tcCO_2} trace, and it cautions against P_{tcCO_2} as the sole method of quantifying severe hypoventilation.¹ Our study therefore both demonstrates the utility of the offset and drift correction method and identifies circumstances where its performance may be compromised.

We used ICU patients as “models” of patients in a sleep laboratory, because we could not conduct arterial punctures in otherwise well sleep patients. Additionally, our experience suggested that taking arterial blood samples from sleeping participants typically resulted in arousal from sleep, large changes in ventilation, and, thus, non-steady-state comparisons of transcutaneous and arterial measurements. While all care was taken to include participants who best modeled the “sleep patient,” future validation studies of the interpolation method with multiple simultaneous measurements over an extended monitoring period should be with sleeping patients who experience a wide range of P_{aCO_2} .

Table 2. Effect of Offset and Drift Correction on the Bias and Limits of Agreement* of Transcutaneously Measured P_{CO_2}

	Uncorrected (mm Hg)		Baseline Corrected (mm Hg)		Baseline and Drift Corrected (mm Hg)	
	Mean $\pm$ SD Bias	Limits of Agreement	Mean $\pm$ SD Bias	Limits of Agreement	Mean $\pm$ SD Bias	Limits of Agreement
Monitor 1	3.3 $\pm$ 5.3	−7.1 to 13.7	3.0 $\pm$ 4.8	−6.5 to 12.5	0.9 $\pm$ 3.7	−6.3 to 8.0
Monitor 2	3.9 $\pm$ 3.3	−2.7 to 10.4	0.9 $\pm$ 2.7†	−4.3 to 6.1	0.0 $\pm$ 2.2	−4.3 to 4.3
Combined data	3.6 $\pm$ 4.4	−5.1 to 12.3	1.9 $\pm$ 4.0‡	−6.0 to 9.8	0.4 $\pm$ 3.0‡§	−5.5 to 6.4

* Limits of agreement (mean $\pm$ 1.96 SD) of P_{tcCO_2} - P_{aCO_2} differences from raw (uncorrected) values, values adjusted for P_{tcCO_2} - P_{aCO_2} difference at baseline (baseline corrected), and values adjusted for baseline offset and drift via linear interpolation between initial and final P_{tcCO_2} - P_{aCO_2} differences (baseline and drift corrected).

† Different from Monitor 1.

‡ Combined data significantly different from uncorrected.

§ Difference statistically significant from baseline corrected.

P_{tcCO_2} = transcutaneously measured P_{CO_2}

Similar to transcutaneous carbon dioxide monitoring, end-tidal carbon dioxide (P_{ETCO_2}) monitoring is a noninvasive method of continuously measuring carbon dioxide over an extended period. In healthy awake individuals, P_{ETCO_2} is essentially equivalent to alveolar (and thus arterial) carbon dioxide values. However, data obtained during sleep indicated that P_{ETCO_2} is insufficiently accurate for diagnostic use in the able-bodied because of the normal reduction in tidal volume and the associated difficulty in detecting an alveolar plateau in the P_{ETCO_2} trace.²⁰ Furthermore, patients with underlying lung disease and associated ventilation-perfusion inhomogeneity are likely to have additional dissociations between P_{ETCO_2} and alveolar CO_2 . The American Academy of Sleep Medicine guidelines¹ provide a recommendation level of D for P_{ETCO_2} monitoring, and C for P_{tcCO_2} , and recommend that further research examine the ability of P_{tcCO_2} to monitor change over extended periods. Those guidelines make no recommendation for further investment in P_{ETCO_2} monitoring during sleep. Unfortunately, there is currently no ideal noninvasive measure of CO_2 during sleep; in the present study we sought to explore whether the imperfect transcutaneous measurement can be made better through the addition of arterial blood sampling at the start and end of the recording period, and by offset and drift correction.

While the detection of a P_{aCO_2} difference of 7.6 mm Hg has been suggested to be the clinically important threshold value,^{8,9} that value is arbitrary and may not represent a clinically relevant limit. There appear to be no published, prospective data that relate a given change in P_{aCO_2} (much less P_{tcCO_2}) to clinical decision making or to clinical outcomes. However, Rodriguez et al demonstrated good discriminant ability of a P_{tcCO_2} monitor to detect a 7.6 mm Hg threshold,¹⁸ and the American Academy of Sleep Medicine recommendations suggest that an overnight P_{aCO_2} change of ≥ 10 mm Hg indicates sleep hypoventilation.¹ Therefore, although 7.6 mm Hg may be arbitrary, it is a pragmatic limit that may be expected to be sensitive to

clinically important change. However, longitudinal studies of a 7.6 mm Hg change are required to confirm the effect of that difference on clinical decision making or patient outcomes.

Conclusions

Even though P_{tcCO_2} has been considered inadequate for the diagnosis or the measurement of response to treatment of hypoventilation, it is used clinically and frequently reported as a measurement of both disease severity and treatment efficacy, particularly in studies of noninvasive ventilation.²⁻⁷ This apparent contradiction suggests that clinicians use the P_{tcCO_2} signal, most likely in combination with other polysomnographic traces and periodic P_{aCO_2} measurements, in the care of their patients, while acknowledging that it may not accurately represent P_{aCO_2} at all times.

The present study provides the first prospective evidence that if corrections are implemented for both offset and drift, P_{tcCO_2} tracks P_{aCO_2} with minimal residual bias over 8 hours of monitoring. Further research, particularly in subjects with less stable ventilation during sleep, is required before P_{tcCO_2} is likely to be recommended for routine clinical use. However, should further research support that recommendation, the present results suggest that the bias and limits of agreement of the interpolated CO_2 values are likely to be sufficiently precise to both detect sleep hypoventilation and to assess the efficacy of treatment.

REFERENCES

1. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999;22(5):667-689.
2. Rosner V, Hannhart B, Chabot F, Polu J. Validity of transcutaneous oxygen/carbon dioxide pressure measurement in the monitoring of

- mechanical ventilation in stable chronic respiratory failure. *Eur Respir J* 1999;13(5):1044-1047.
3. Cox M, Kemp R, Anwar S, Athey V, Aung T, Moloney ED. Non-invasive monitoring of CO₂ levels in patients using NIV for AE-COPD. *Thorax* 2006;61(4):363-364.
  4. Cuvelier A, Grigoriu B, Molano LC, Muir J-F. Limitations of transcutaneous carbon dioxide measurements for assessing long-term mechanical ventilation. *Chest* 2005;127(5):1744-1748.
  5. Ward S, Chatwin M, Heather S, Simonds AK. Randomised controlled trial of noninvasive ventilation (NIV) for nocturnal hypoventilation in neuromuscular and chest wall disease patients with daytime normocapnia. *Thorax* 2005;60(12):1019-1024.
  6. Guo YF, Sforza E, Janssens JP. Respiratory patterns during sleep in obesity-hypoventilation patients treated with nocturnal pressure support: a preliminary report. *Chest* 2007;131(4):1090-1099.
  7. Meecham Jones DJ, Paul EA, Jones PW, Wedzicha JA. Nasal pressure support ventilation plus oxygen compared with oxygen therapy alone in hypercapnic COPD. *Am J Respir Crit Care Med* 1995;152(2):538-544.
  8. Bolliger D, Steiner LA, Kasper J, Aziz OA, Filipovic M, Seeberger MD. The accuracy of noninvasive carbon dioxide monitoring: A clinical evaluation of 2 transcutaneous systems. *Anaesthesia* 2007;62(4):394-399.
  9. Bendjelid K, Schutz N, Stotz M, Gerard I, Suter PM, Romand JA. Transcutaneous P_{CO₂} monitoring in critically ill adults: clinical evaluation of a new sensor. *Crit Care Med* 2005;33(10):2203-2206.
  10. O'Donoghue FJ, Catcheside PG, Ellis EE, Grunstein RR, Pierce RJ, Rowland LS, et al. Sleep hypoventilation in hypercapnic COPD: prevalence and associated factors. *Eur Respir J* 2003;21(6):977-984.
  11. Laird NM, Ware JH. Random-effects models for longitudinal data. *Biometrics* 1982;38(4):963-974.
  12. Bland JM, Altman DG. Statistical methods for assessing agreement between 2 methods of clinical measurement. *Lancet* 1986;1(8476):307-310.
  13. Janssens JP, Howarth-Frey C, Chevrolet JC, Abajo B, Rochat T. Transcutaneous P_{CO₂} to monitor noninvasive mechanical ventilation in adults: assessment of a new transcutaneous P_{CO₂} device. *Chest* 1998;113(3):768-773.
  14. Janssens JP, Laszlo A, Uldry C, Titelion V, Picaud C, Michel JP. Noninvasive (transcutaneous) monitoring of P_{CO₂} (TcP_{CO₂}) in older adults. *Gerontology* 2005;51(3):174-178.
  15. Janssens JP, Perrin E, Bennani I, de Muralt B, Titelion V, Picaud C. Is continuous transcutaneous monitoring of P_{CO₂} (TcP_{CO₂}) over 8 h reliable in adults? *Respir Med* 2001;95(5):331-335.
  16. Nishiyama T, Nakamura S, Yamashita K. Effects of the electrode temperature of a new monitor, TCM4, on the measurement of transcutaneous oxygen and carbon dioxide tension. *J Anesth* 2006;20(4):331-334.
  17. Senn O, Clarenbach CF, Kaplan V, Maggiorini M, Bloch KE. Monitoring carbon dioxide tension and arterial oxygen saturation by a single earlobe sensor in patients with critical illness or sleep apnea. *Chest* 2005;128(3):1291-1296.
  18. Rodriguez P, Lellouche F, Aboab J, Buisson CB, Brochard L. Transcutaneous arterial carbon dioxide pressure monitoring in critically ill adult patients. *Intensive Care Med* 2006;32(2):309-312.
  19. Clark JS, Votteri B, Ariagno RL, Cheung P, Eichhorn JH, Fallat RJ, et al. Noninvasive assessment of blood gases. *Am Rev Respir Dis* 1992;145(1):220-232.
  20. Sanders MH, Kern NB, Costantino JP, Stiller RA, Strollo PJ Jr, Studnicki KA, et al. Accuracy of end-tidal and transcutaneous P_{CO₂} monitoring during sleep. *Chest* 1994;106(2):472-483.

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