Transcutaneous Pco₂ to Monitor Noninvasive Mechanical Ventilation in Adults*

Assessment of a New Transcutaneous Pco₂ Device

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The present study was designed to analyze the usability of a commercially available, transcutaneous Pco₂ (TcPco₂) sensor for monitoring noninvasive positive pressure ventilation (NPPV). Twenty-six hemodynamically stable patients with intra-arterial radial catheters were assessed. After stabilization of TcPco₂, arterial blood was analyzed and results were compared with TcPco₂ at time of sampling. To evaluate the drift of the signal, samples were taken hourly in five patients for 4 h while continuously recording TcPco₂. Finally, to assess for the response of the sensor to changes in PaC0₂, six patients underwent continuous TcPco₂ recording while initiating or interrupting NPPV; arterial samples were analyzed before the event, and 1, 3, 5, 7, 9, and 20 min afterwards.

Results: TcPco₂ and PaC0₂ were tested over a range of 26 to 71 mm Hg, and were found to be closely correlated (r=0.968, p<0.0001); mean bias was 0.75 mm Hg. There was no significant drift of TcPco₂ as compared with PaC0₂ over 4 h. The time of response of TcPco₂ to initiation or interruption of NPPV was <60 s. An estimation of the lag time averaged 5±3 min (range, 1 to 9 min).

Conclusion: TcPco₂ in hemodynamically stable adults was in excellent agreement with arterial measurements. The time of response to a change in ventilation was compatible with the aim of clinical monitoring of patients under NPPV.

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Key words: blood gas monitoring, transcutaneous; carbon dioxide tension; intermittent positive pressure ventilation

Abbreviations: d=bias; NPPV=nasal positive pressure ventilation; t90 %=90 % response time; TcPco₂=transcutaneous carbon dioxide partial pressure

Increasing evidence of the benefits of noninvasive ventilatory support in a variety of pulmonary disorders has led to the relatively widespread use of bi-level positive pressure support and noninvasive nasal positive pressure ventilation (NPPV). Assessment of PaC0₂ is essential for evaluation of the adequacy of alveolar ventilation in this setting. PaC0₂ can be measured on samples obtained by arterial puncture or from an indwelling catheter in the radial artery. Repeated sampling of arterial blood remains so far the “gold standard” for estimating the adequacy of ventilatory support. However, arterial catheterization needs costly equipment, specially trained personnel, and in most cases the environment of an ICU.¹ There is also a low but measurable morbidity associated with arterial catheterization.²

Noninvasive assessment of PaC0₂ can be performed by measuring transcutaneous CO₂ (TcPco₂) or peak expired CO₂. The latter, however, is a poor predictor of PaC0₂.³,⁴ Also, accurate measurement of peak expired CO₂ cannot be achieved during nasal continuous positive airway pressure ventilation because of continuous flow through the mask. Transcutaneous measurement of carbon dioxide theoretically appears more appropriate for monitoring PaC0₂. This measurement is based on the observation that CO₂ has a high tissue solubility and diffuses through the skin. It can be performed by two different types of sensors: the Severinghaus electrode⁵,⁶ and the Hewlett-Packard TcPco₂ device (Waltham, Mass), which uses an infrared transcutaneous capnometer.¹ Most capnometers use the Severinghaus electrode.
Available data as to the precision of TcPCO₂ have given conflicting results.3,7-17 Therefore, the present study was designed to evaluate a new device for the measurement of TcPCO₂. We wanted to test for agreement with arterial values, detect any possible drift of TcPCO₂ over time, and describe the response of TcPCO₂ when initiating or interrupting NPPV in hypercapnic patients.

**Materials and Methods**

The study protocol was approved by the Ethics Committee of the University Hospital of Geneva. Patients who had been admitted to the ICU and equipped with an indwelling arterial catheter were considered for the present study. To be included, they had to be in hemodynamically stable condition without vasopressor amine treatment other than low-dose dopamine (≤0.2 mg/min) to avoid cutaneous hyperperfusion or vasoconstriction. Twenty-six patients were included (18 men, 8 women, aged 68±10 years). Diagnoses included respiratory failure in eight, monitoring after heart failure or myocardial infarction in eight, neurologic disorders in six, severe systemic hypertension in two, pulmonary hypertension in one, and sepsis in one. At the time of the protocol, systolic BP was 128±24 mm Hg, diastolic pressure was 61±13 mm Hg, and mean BP was 83±15 mm Hg. Mean body weight was 74±11 kg. Eight patients were under perfusion with a gas-permeable Teflon membrane. The collar of the sensor was softened with a gas-permeable gel: the membrane surface temperature was approximately 37°C. Arterial blood gases were determined with a gas-analyzer (ABL 520; Radiometer; Copenhagen, Denmark).

Correlation Between TcPCO₂ and PaCO₂ at Steady State

Twenty-six pairs of measurements were recorded from 26 different subjects. Because PaCO₂ may vary substantially even in patients in apparently stable condition,20 we ensured that arterial samples were taken after stabilization of TcPCO₂ values for at least 3 min. We also checked for the stability of these values afterwards: 3 min after blood sampling, changes in TcPCO₂ were minimal and ranged from 0 to 3 mm Hg (mean±SD: 1±1 mm Hg). For correlation studies, PaCO₂ values were compared with values of TcPCO₂ at time of sampling.

**Possible Drift of TcPCO₂ During Continuous Recording**

To detect any systematic drift of TcPCO₂ values as compared with PaCO₂, we continuously recorded TcPCO₂ in five patients for 4 h. We did not exceed 4 h to avoid any risk of skin burning as a consequence of the temperature of the sensor. Arterial samples were taken at the beginning of the recordings, then every hour for 4 h.

Response of Transcutaneous Measurements to Induced Changes in PaCO₂

In six hypercapnic patients, we examined the response of TcPCO₂ to a ventilatory event that was expected to induce a change in PaCO₂. The event consisted in initiating NPPV in five patients and interrupting NPPV in one. During the procedure, TcPCO₂ was recorded continuously. Arterial blood samples were drawn 3 and 1 min before the event, then 1, 3, 5, 7, 9, and 20 min afterwards. Values of PaCO₂ were paired with the corresponding values of TcPCO₂ at the time of arterial blood sampling. Values obtained 1 min before the event were considered as baseline. An exponential fit curve was computed for PaCO₂ and TcPCO₂ curves of each patient. The “90% response time” (t₉₀%) was then calculated for each curve (ie, the time at which PaCO₂ or TcPCO₂ had reached 90% of the final equilibrated value). The mean difference between t₉₀% for TcPCO₂ and t₉₀% for PaCO₂ was reported as an index of the TcPCO₂ response time itself. Results are reported as mean value (range).

**Statistical Analysis**

PaCO₂ and TcPCO₂ were correlated by linear regression, with calculation of Pearson’s coefficient of correlation (r).

We also calculated the bias and the limits of agreement between the parameters as described by Bland and Altman.22 The bias (d) is the mean difference between TcPCO₂ and PaCO₂ values; if s is the SD of d, the limits of agreement between the

**Figure 1.** The capnograph (Fastrac) with the sensor placed on the skin of the anterior abdomen.
two methods are defined as follows: $d \pm 2s$. Ninety-five percent of the values of $(TcPCO_2 - PaCO_2)$ are expected to be within the limits of agreement.

Exponential fits for curves of PaCO$_2$ and TcPCO$_2$ in response to induced changes were computed using software (GraphPad “Prism” Software (1994); GraphPad Software; San Diego, Calif).

Analysis of variance for repeated measurements and Student’s $t$ test were used for other comparisons.

RESULTS

Agreement between transcutaneous and arterial values for CO$_2$ was tested over a range of 26 to 71 mm Hg. TcPCO$_2$ as a function of PaCO$_2$ is shown on
FIGURE 2. Both measurements were highly correlated (r=0.968, p<0.001), and linear regression was close to the identity line (TcPCO₂ = 1.116 × PaCO₂ - 0.46). Figure 2 indicates that at high PaCO₂ values, there was a trend for TcPCO₂ to slightly overestimate PaCO₂. Figure 3 shows the d and limits of agreement between the TcPCO₂ and PaCO₂. The d was 0.75 mm Hg; SD of d was 2.6 mm Hg; limits of agreement were therefore as follows: -4.5 to +6 mm Hg. Eight of 26 patients were receiving low-dose dopamine (≤0.2 mg/min). In these cases, correlation between TcPCO₂ and PaCO₂ was not altered and appeared as close as for the rest of the group (Fig 2).

In five patients with prolonged, continuous recording, the mean values for (TcPCO₂-PaCO₂) showed no significant difference at 0, 1, 2, 3, and 4 h (p=0.873). Therefore, no significant drift of TcPCO₂ could be detected over this period; maximal difference between TcPCO₂ and PaCO₂ reached 6 mm Hg after 4 h (Fig 4).

Figure 5 shows simultaneous recordings of TcPCO₂ and PaCO₂ in all six patients, while initiating or interrupting NPPV. TcPCO₂ followed the same trend as PaCO₂, with a few minutes of delay, as expected. In all six patients, the first changes in TcPCO₂ were observed during the first 60 s following the ventilatory event. In four patients, a new steady state for PaCO₂ was reached after starting or interrupting NPPV, and lasted for up to 20 min. We computed an exponential fit for PaCO₂ and TcPCO₂ values, and calculated the t90% for each curve. The difference between t90% for PaCO₂ and TcPCO₂ indicates the lag time of the latter over the former. This lag time averaged 5±3 min (range, 1 to 9 min). In two patients (cases 4 and 6), PaCO₂ did not become sufficiently stable after the start of NPPV and this precluded computation of an exponential fit with calculation of the lag time. However, it can be estimated from the graph on Figure 5 that the response time of TcPCO₂ in case 6 did not lag over PaCO₂ by more than a few minutes. In case 4, there was an initial drop in TcPCO₂ without concomitant change in PaCO₂, for the first 5 min; then both graphs followed a similar trend.

**DISCUSSION**

We have shown that TcPCO₂ values, measured with the capnograph (Fastrac), showed a good agreement with simultaneous arterial measurements of PaCO₂, without significant drift after 4 h of continuous recording.

A review of published studies, as summarized in Table 1, shows conflicting results regarding agreement between TcPCO₂ and PaCO₂. Three studies, however, gave excellent correlation, two of them indicating Pearson’s coefficient of correlation (r) equal to 0.97, and three others reporting bias values of 1.5 mm Hg with SDs of 3.5 mm Hg. Thus, the results that we obtained in the present study are among the best reported so far, and they seem appropriate for clinical application.

By contrast, in some other studies, the limits of agreement are such that measurement of TcPCO₂ would appear unfit for clinical purposes such as monitoring NPPV. In some cases, it seems to be related to a particular capnograph. However, other factors such as calibration before all measurements, frequent change of membranes, allowing sufficient time for stabilization of TcPCO₂ signals, and adequate cutaneous perfusion are important in ensuring sufficient precision of transcutaneous measurements. TcPCO₂ measurements are reported to be adversely affected by cutaneous vasoconstriction due to low cardiac output or vasoconstricting agents, hypothermia, and elevated cutaneous vascular resistance due to hypovolemic or cardiogenic hypotension. A cardiac index below 1.5 L/min can dramat-
phically increase the \( \text{TcPco}_2/\text{PaCO}_2 \) gradient.\(^1\) However, Palmisano and Severinghaus\(^6\) did not find any significant effect of use of vasopressors on bias, limits of agreement, or regression slope. Similarly we found that low-dose dopamine did not alter measurement of \( \text{TcPco}_2 \). Finally, it is interesting to note that the error of \( \text{TcPco}_2 \) is the same in fat and lean subjects, and is unrelated to body mass index.\(^3,10\)

A drawback of the method is the necessity to change the skin site after 4 h to avoid skin burning—and to recalibrate the capnograph. One might consider decreasing the temperature to allow a longer time at the same site. However, temperatures of approximately 43°C are required to maintain adequate perfusion at the skin site: lower sensor temperatures have been associated with longer response times\(^23\) and would not be desirable. The limit of 4 h at 43°C must be considered as a rule of safety, but other investigators have reported no skin burn after periods of 6 to 8 h at sensor temperatures of 43 to 44°C.\(^7,9,24,25\) Furthermore, although we found no significant drift of \( \text{TcPco}_2 \) vs \( \text{PaCO}_2 \) over a 4-h period, one case did show a \( \text{PaCO}_2/\text{TcPco}_2 \) difference of 6 mm Hg after 4 h of continuous recording. Others have reported, over periods of 6 to 7 h, a drift of 0.1 to 0.8 mm Hg/h.\(^7,9,24,25\)

The aim of our study was to evaluate if a \( \text{TcPco}_2 \) capnograph that appears suitable for measurements under steady-state conditions would also be suitable for monitoring noninvasive, intermittent ventilation. We found that when a ventilatory event was induced by initiating or interrupting NPPV in hypercapnic patients, a change in \( \text{TcPco}_2 \) was detected within <60 s, and that the trend accurately reflected the change in \( \text{PaCO}_2 \). Moreover, by 3 min, at least half of the amplitude of the change in \( \text{PaCO}_2 \) had been displayed by \( \text{TcPco}_2 \) recording. An estimation of the lag time averaged 5±3 min (range, 1 to 9 min).

Our results therefore suggest that the apparatus would appear suitable for monitoring NPPV, with some important limitations that the clinician must bear in mind: there is a distinct possibility of occasional errant \( \text{TcPco}_2 \) values: Figure 3, for instance, shows two values overestimating \( \text{PaCO}_2 \) by 8 to 12 mm Hg; the high correlation depicted between \( \text{TcPco}_2 \) and \( \text{PaCO}_2 \) values was obtained after recalibration for each individual measurement—a 10-min procedure that may appear time-consuming in clinical practice. Furthermore, although \( \text{TcPco}_2 \) did accurately detect acute changes in \( \text{PaCO}_2 \) in five of six cases, case 4 (Fig 5) showed an initial drop in \( \text{TcPco}_2 \) without any concomitant change in \( \text{PaCO}_2 \). Substantial changes in \( \text{TcPco}_2 \) should probably be confirmed by arterial blood gas determination, once a new steady state is reached. Also, the slow response time, although inferior to 1 min in our study, prevents \( \text{TcPco}_2 \) from detecting short, transient changes in \( \text{PaCO}_2 \) that can be associated with brief apneas or hypopneas. This point has been well illustrated by Lanigan and coworkers,\(^8\) with healthy subjects breathing a hypercapnic gas mixture that will induce almost instantaneous changes on \( \text{PaCO}_2 \). The lag time of various \( \text{TcPco}_2 \) devices in these studies ranged from 31 to 56 s.\(^8\) Recording \( \text{TcPco}_2 \)

Table 1—Correlation Between \( \text{TcPco}_2 \) and \( \text{PaCO}_2 \), Bias, and Limits of Agreement for Different Capnographs According to Available Published Data\(^6\)

<table>
<thead>
<tr>
<th>First Author</th>
<th>No. of Patients</th>
<th>r</th>
<th>( d_1 ) mm Hg</th>
<th>( s_1 ) mm Hg</th>
<th>Limits of Agreement</th>
<th>Capnograph*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>( d -2 ) s</td>
<td>d+2 s</td>
</tr>
<tr>
<td>Sanders(^3)</td>
<td>17</td>
<td>0.45</td>
<td>10.5</td>
<td>15.1</td>
<td>-19.7</td>
<td>40.7</td>
</tr>
<tr>
<td>Lanigan(^6)</td>
<td>12</td>
<td>0.52</td>
<td>0.0</td>
<td>7.9</td>
<td>-15.7</td>
<td>15.7</td>
</tr>
<tr>
<td>Sanders(^3)</td>
<td>15</td>
<td>0.67</td>
<td>6.8</td>
<td>11.5</td>
<td>-16.2</td>
<td>29.8</td>
</tr>
<tr>
<td>Sanders(^3)</td>
<td>17</td>
<td>0.80</td>
<td>0.2</td>
<td>10.9</td>
<td>-21.7</td>
<td>22.0</td>
</tr>
<tr>
<td>Hoffmann(^13)</td>
<td>9</td>
<td>0.84</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Lanigan(^6)</td>
<td>12</td>
<td>0.85</td>
<td>-6.6</td>
<td>3.4</td>
<td>-13.4</td>
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<tr>
<td>Healey(^12)</td>
<td>20</td>
<td>0.87</td>
<td>7.5</td>
<td>5.6</td>
<td>-3.8</td>
<td>18.7</td>
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<tr>
<td>Kesten(^14)</td>
<td>20</td>
<td>0.90</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
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<td>Lanigan(^8)</td>
<td>12</td>
<td>0.91</td>
<td>-0.5</td>
<td>2.8</td>
<td>-6.2</td>
<td>5.2</td>
</tr>
<tr>
<td>Reid(^15)</td>
<td>22</td>
<td>0.92</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Palmisano(^6)</td>
<td>251</td>
<td>0.93</td>
<td>1.3</td>
<td>3.9</td>
<td>-6.5</td>
<td>9.1</td>
</tr>
<tr>
<td>Mahutte(^9)</td>
<td>47</td>
<td>0.93</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Pilsbury(^7)</td>
<td>28</td>
<td>0.97</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
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<tr>
<td>Hanly(^11)</td>
<td>5</td>
<td>0.97</td>
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<td>NA</td>
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<td>NA</td>
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<tr>
<td>Sridhar(^17)</td>
<td>24</td>
<td>NA</td>
<td>0.2</td>
<td>1.0</td>
<td>-1.7</td>
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<td>Blanchette(^10)</td>
<td>30</td>
<td>NA</td>
<td>-0.5</td>
<td>5.2</td>
<td>-11.0</td>
<td>9.9</td>
</tr>
</tbody>
</table>

*Limits of agreement: \( d -2 \) s; \( d +2 \) s (expressed as described by Bland and Altman.\(^22\))

NA=not available from original publication.

*Sensormedics, Yorba Linda, Calif; Novametrix, Wallingford, Conn; Radiometer, Copenhagen; Kontron, St. Quentin En Yvelines, France.
would appear therefore unsuitable for monitoring patients with obstructive sleep apnea, for example.

In patients with NPPV, however, the correction of long-lasting periods of hypoventilation or hyperventilation, particularly during sleep, represents a major goal. Recently, three groups of investigators have used TcPCO₂ monitoring to estimate the mean level of PaCO₂ during the night. Naughton and colleagues³⁵ found that treatment of Cheyne-Stokes breathing with nasal continuous positive airway pressure increased mean nocturnal TcPCO₂ and decreased the number of apneas, underscoring the role of hypocapnia in the pathogenesis of periodic breathing. In a different setting, Piper and Sullivan²⁶ found that treatment with nasal continuous positive airway pressure in patients with combined obstructive sleep apneas and hypoventilation resulted in gradual decrease of mean nocturnal TcPCO₂. Finally, Meecham-Jones and coworkers²⁷ found that treatment with nocturnal nasal pressure support significantly decreased mean TcPCO₂ in selected patients with COPD, and that this change was associated with improved daytime PaCO₂.

Our results suggest that TcPCO₂ monitoring may give more detailed information than just the computation of mean nocturnal PaCO₂ as reported by others.²⁵,²⁷ The responses that we observed after initiating or interrupting NPPV show that the method can identify ventilatory events, as far as they last >1 min, and may permit us to undertake the necessary adjustments in ventilatory support. This might be particularly important in patients who receive combined NPPV and O₂ supplementation, in whom monitoring by pulse oximetry alone has limited value.

References