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Xavier Bobbia, Pierre-Géraud Claret, Ludovic Palmier, Michaël Robert, Romain Genre Granpierre, et al.. Erratum: Concordance and limits between transcutaneous and arterial carbon dioxide pressure in emergency department patients with acute respiratory failure: a single-center, prospective, and observational study. *Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine*, 2015, 23, pp.77. 10.1186/s13049-015-0154-7. hal-01909448

HAL Id: hal-01909448

<https://hal.science/hal-01909448>

Submitted on 8 Jun 2021

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ERRATUM

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Erratum: Concordance and limits between transcutaneous and arterial carbon dioxide pressure in emergency department patients with acute respiratory failure: a single-center, prospective, and observational study

Xavier Bobbia^{1†}, Pierre-Géraud Claret^{1*†}, Ludovic Palmier¹, Michaël Robert¹, Romain Genre Granpierre¹, Claire Roger¹, Justin Yan², Patrick Ray³, Mustapha Sebbane¹, Laurent Muller¹ and Jean-Emmanuel de La Coussaye¹

Abstract

After publication of this article (*Scand J Trauma Resusc Emerg Med* 23:40, 2015), it came to light that an earlier version had been published in error. This erratum contains the correct version of the article, which incorporates revisions made in response to reviewer comments. Additionally, one of the authors was inadvertently omitted from the author list. This author, Justin Yan, has been included in the corrected author list above.

Background: Transcutaneous CO₂ (PtCO₂) is a continuous and non-invasive measure recommended by scientific societies in the management of respiratory distress. The objective of this study was to evaluate the correlation between PtCO₂ and arterial partial pressure of CO₂ (PaCO₂) by arterial blood gas analysis in emergency patients with dyspnoea, and to determine the factors that interfere with this correlation.

Methods: From January to June 2014, all adult patients admitted to the RR with dyspnoea during business hours were included in the study if arterial blood gas measurements were indicated. A sensor measuring the PtCO₂ was attached to the ear lobe of the patient before the gas analysis. Anamnesis, clinical and laboratory parameters were identified.

Results: Ninety patients with dyspnoea were included (104 pairs of measurements). The median (IQR) age was 79 years (69 – 85). The correlation between PtCO₂ and PaCO₂ was $R^2 = .83$ ($p < .001$) but became lower for values of PaCO₂ above 60 mm Hg. The mean bias (\pm SD) between the two methods of measurement (Bland-Altman analysis) was -1.4 mm Hg (± 7.7) with limits of agreement from -16.4 to 13.7 mm Hg. In univariate analysis, PaO₂ interfered with this correlation. After multivariate analysis, temperature (OR = 3.01; 95 % CIs [1.16, 7.80]) and PaO₂ (OR = 1.22; 95 % CIs [1.02, 1.47]) significantly interfered with this correlation.

Conclusions: There is a significant correlation between PaCO₂ and PtCO₂ values for patients admitted to the emergency department for acute respiratory failure. One limiting factor to routine use of PtCO₂ measurements in the emergency department is the presence of hyperthermia.

Keywords: Emergency service, Blood gas monitoring, Transcutaneous, Carbon dioxide, Partial pressure

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Background

This is a corrected version of the previously published article [1]. Arterial blood gas monitoring is crucial for management of patients with respiratory failure [2]. The gold standard technique involves an arterial puncture which is invasive, time-consuming, and only gives results at one point in time [3, 4]. Moreover, the delay in waiting for the results of blood gas analysis does not allow for real-time adaptation of oxygen therapy or mechanical ventilation. Oxygen saturation by pulse oximetry (SpO₂) is widely used as a surrogate of arterial oxygen saturation (SaO₂) [5]. Similarly, end tidal CO₂ (EtCO₂) allows for an indirect, but reliable and continuous assessment of arterial pCO₂ for mechanically ventilated patients. However, for non-ventilated patients, assessment of EtCO₂ is more complex, less accurate, and often impossible. For these patients, the recently recommended [6, 7] transcutaneous monitoring of carbon dioxide (PtCO₂) could represent an alternative for immediate and continuous assessment of pCO₂. Numerous studies of both children [8, 9] and adults [10–12] have found a good correlation between PaCO₂ and PtCO₂. Yet in the specific setting of the emergency department (ED) resuscitation room (RR), PtCO₂ has been poorly studied. The main objective of this study was to investigate the relationship between measures of PtCO₂ and PaCO₂ for patients admitted to the ED RR. The secondary objective was to determine the variables that may disrupt the link between PtCO₂ and PaCO₂.

Methods

Setting

We conducted this single-center prospective observational study from January to June 2014 in the ED of Nîmes University Hospital, France. This study was reviewed and approved by our Institutional Review Board (number: 13/06–02) and was declared to and approved by the national commission for data processing and civil liberties. All patients provided written informed consent. This study is in compliance with the Helsinki Declaration.

Study population

All adult patients admitted to the RR with dyspnoea during business hours (from 9:00 to 17:00, weekend excluded) were included in the study if arterial blood gas measurements were indicated. In our ED, patients are admitted to the RR if they are level 1 or level 2 according to the Canadian Triage and Acuity Scale (CTAS). Thus, patients with dyspnoea are admitted to the RR if they suffered from severe respiratory distress, asthma, or important dyspnoea. Definition of CTAS level 1 and level 2 for dyspnoea are specified in Appendix 1. Exclusion criteria were incorrect installation of the sensor, signal abnormality on the monitor, and backup error on the memory of the device.

Measurement

The PtCO₂ measurement was performed by a Stow-Severinghaus sensor (tc Sensor 92 by Radiometer™, Copenhagen, Denmark). The sensor heats skin to a temperature of 44 °C resulting in a dilatation of the capillary bed that allows for diffusion of gases (CO₂ and O₂) [13]. On the sensor, carbon dioxide reacts with water to form carbonic acid which dissociates into H⁺ and HCO₃⁻, thereby changing pH values. These pH changes are translated into PtCO₂ value through the Henderson-Hasselbalch formula [14]. Medical and paramedical staff were trained in the operation and maintenance of the PtCO₂ TOSCA monitor (Radiometer™, Copenhagen, Denmark) before the study commenced. For included patients, the PtCO₂ sensor was attached to the ear lobe of the patient allowing for continuous measurement of PtCO₂. After stabilization of the monitor to obtain a good signal, arterial blood gases and PtCO₂ measurements were performed simultaneously. The medical team was blinded to the value of PtCO₂ measured.

Outcomes

The primary outcome was concordance between the simultaneous PaCO₂ and PtCO₂ values. The sample size calculation was based on the anticipated variation in the differences between the measurements and the required precision. Using a previous study [15] for an estimate of the variation between the differences, a sample size of 50 patients gave a precision of ± 0.19 kPa as the limits of agreement. The secondary outcome was to determine the factors that interfere with this correlation. PtCO₂ values were automatically saved every ten seconds by the monitor. Medical patient data were collected and entered into an electronic database after initial collection on paper case report forms (CRF). Blood pressure, heart rate, respiratory rate, blood oxygen saturation, Glasgow coma scale, temperature, time to completion of arterial blood gases, catecholamine use, and non-invasive ventilation or tracheal intubation were recorded by the attending physician. Characteristics of patients such as ED arrival modalities, hospital length of stay, and biological data were collected on the CRF.

Statistical analysis

Patient characteristics were described using qualitative (frequencies and percentages) or quantitative variables (means and standard deviations or median with interquartile ranges - depending on type of distribution) where appropriate. The concordance between PtCO₂ and PaCO₂ was evaluated by linear regression (correlation coefficients) and Bland-Altman analysis, which determined bias, precision, and agreement of PtCO₂ and PaCO₂, taking the automated analysis in the laboratory as the reference standard. The Pearson correlation

coefficient was used to demonstrate the presence or absence of a relationship between PtCO₂ and PaCO₂. Relationships between measurement differences (| PaCO₂ - PtCO₂ |) and patient characteristics were investigated by regression analysis. Variables related to the difference between PtCO₂ and PaCO₂ in the univariate analysis (defined by $p < .1$, forward selection) were further analyzed in a multivariate model (analysis of covariance). We included PaCO₂ in this model but did not include pH or PtCO₂ to avoid a collinear bias. Overall model fit was assessed using the Hosmer-Lemeshow test. All statistical tests were two sided. A p -value less than .05 was considered significant for all analyses.

Analyses were performed with the use of R 3.0.2 (R Core Team 2013, R: A language and environment for statistical computing, R Foundation for Statistical Computing, Vienna, Austria). The authors had full access to and take full responsibility for the integrity of the data.

Results

Between January 2014 and June 2014, 102 patients were screened for eligibility. Ninety patients were included and analyzed with 104 PtCO₂ values (Fig. 1). Table 1 shows the patient characteristics corresponding to the 104 measurements. After linear regression analysis of 104 couples of measurements, we found a significant correlation between PaCO₂ and PtCO₂ with $R^2 = .83$ ($p < .001$) (Fig. 2). The linear regression equation between the two variables was $PaCO_2 = (0.81 \times PtCO_2) + 10.86$. The Bland-Altman analysis is shown in Fig. 3. The mean bias was -1.4 mm Hg (± 7.7) and the limits of agreement (bias ± 1.96 SD) between the two techniques were -16.4 mm Hg and 13.7 mm Hg. The Pearson’s correlation coefficient was

.94 (95 % CIs [0.87, 0.94]; $p < .001$). For the group with PaCO₂ < 60 mm Hg, $R^2 = .70$ ($p < .001$) and the mean bias was -3.5 mm Hg (± 5.0). For the group with PaCO₂ > 60 mm Hg, $R^2 = .57$ ($p < .001$) and the mean bias was 4.1 mm Hg (± 10.2).

In the univariate analysis, the only factor associated with a difference between PaCO₂ and PtCO₂ was PaO₂ (Table 2). In multivariate analysis with three explanatory variables (PaCO₂, PaO₂, temperature), we found the temperature and the PaO₂ to be significantly associated with a large difference between PaCO₂ and PtCO₂ (Table 2). The higher the temperature of the patient, the greater the difference between PaCO₂ and PtCO₂ (Fig. 4). We developed this model on a data set of 93 measurements (11 observations were excluded due to missingness). This model had a non-significant Hosmer-Lemeshow chi-square goodness-of-fit statistic.

Discussion

To our knowledge, our study is the largest cohort of PtCO₂ measurements conducted in the ED. The mean bias was -1.4 mm Hg (± 7.7) and the limits of agreement (bias ± 1.96 SD) between the two techniques were -16.4 mm Hg and 13.7 mm Hg. There was a significant correlation between PaCO₂ and PtCO₂ ($R^2 = .83$; $p < .001$). Because most of our patients were non-intubated, our results highlight the feasibility and the potential benefit of measuring PtCO₂ since EtCO₂ cannot easily be monitored in non-intubated patients. The correlation coefficient in our study was comparable to what was shown in a previous intensive care study ($R^2 = .86$; $p < .01$) [10]. However, other studies have found a stronger correlation (R^2 coefficient, ranged between .91 and .99 [16–18]).

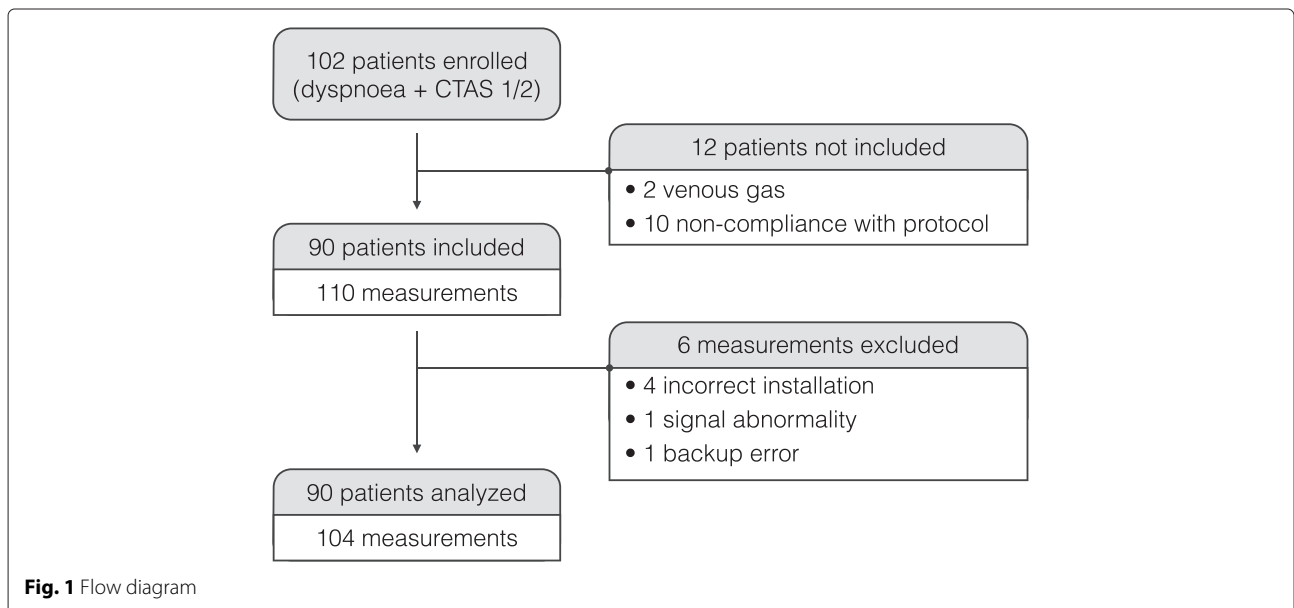


Fig. 1 Flow diagram

Table 1 Patients' characteristics

Male sex, no. (%)	51 (57)
Age, mean (± SD) - year	76 (15)
Past medical history, no. (%)	
Acute pulmonary edema	27 (29)
Chronic obstructive pulmonary disease	27 (29)
Ischemic heart disease	21 (23)
Home oxygen	16 (17)
Clinical data at admission, median (IQR)	
Heart rate - beats/min.	94 (80–110)
Systolic blood pressure - mm Hg	122 (106–144)
Diastolic blood pressure - mm Hg	69 (60–78)
Respiratory rate - breaths/min.	24 (19–28)
Glasgow coma scale	15 (14–15)
Temperature - °C	37.0 (36.2–37.6)
Laboratory values, median (IQR)	
PaCO ₂ - mm Hg	46.2 (37.6–66.8)
PtCO ₂ - mm Hg	47.2 (42.1–60.0)
PaO ₂ - mm Hg	73.5 (63.0–89.0)
pH	7.37 (7.30–7.43)
HCO ₃ - mEq/L	26.0 (22.8–29.7)
Base excess - mmol/L	1.9 (-1.9–5.8)
Lactate - mmol/L	1.3 (0.7–2.2)
Hemoglobin - g/dL	12.3 (10.9–13.8)
White blood cells - G/L	12.4 (7.9–15.5)
C-reactive protein	41 (8–122)
Glycemia - g/L	1.4 (1.2–1.7)
Brain natriuretic peptide - ng/L	1704 (579–6200)
Diagnosis, no. (%)	
Heart failure	25 (27)
COPD	14 (15)
Pneumonia	42 (46)
Pulmonary embolism	5 (5)
Outcome, no. (%)	
Noninvasive ventilation required	41 (45)
Intubation required	4 (4)
Admitted to hospital	61 (66)
Admitted to ICU	19 (21)
Discharged from ED	10 (11)
Death at the ED	2 (2)
Inpatient mortality	9 (10)

We assume this difference is not a consequence of the use of various devices since most of the studies were completed with a Radiometer™ device. This difference can be explained by the selection of our patients, as only those in the RR with acute respiratory failure were included.

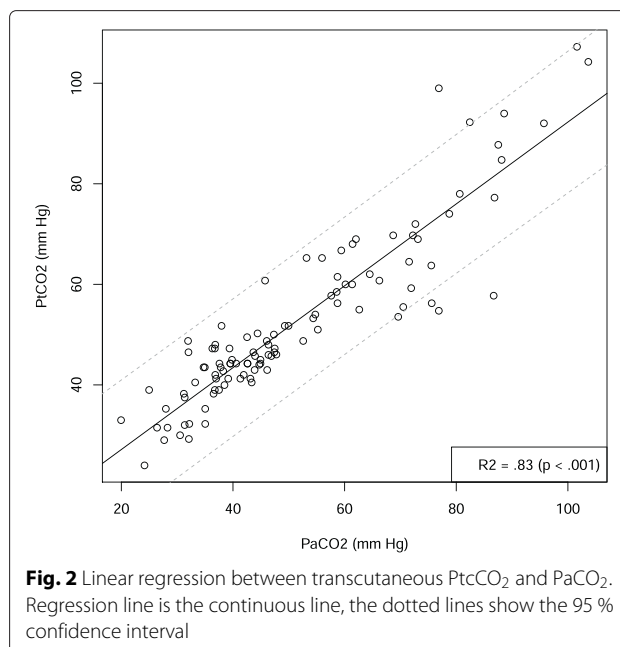


Fig. 2 Linear regression between transcutaneous PtcCO₂ and PaCO₂. Regression line is the continuous line, the dotted lines show the 95 % confidence interval

Indeed, the high and extreme PaCO₂ values were reported as possibly interfering with the correlation between PtCO₂ and PaCO₂ [10, 19–21]. In a study by Delorme et al. [11], patients had a lower PaCO₂ than in our study (39 mm Hg vs. 46, respectively). Secondly, this difference may result from the use of the device by many physicians. Calibration, sensor placement and latency to reach the plateau value of PtCO₂ may differ from one physician to another. Because some operators used the monitor less frequently, this may have led to poorer reproducibility. However, it also reflects our center's daily practice and

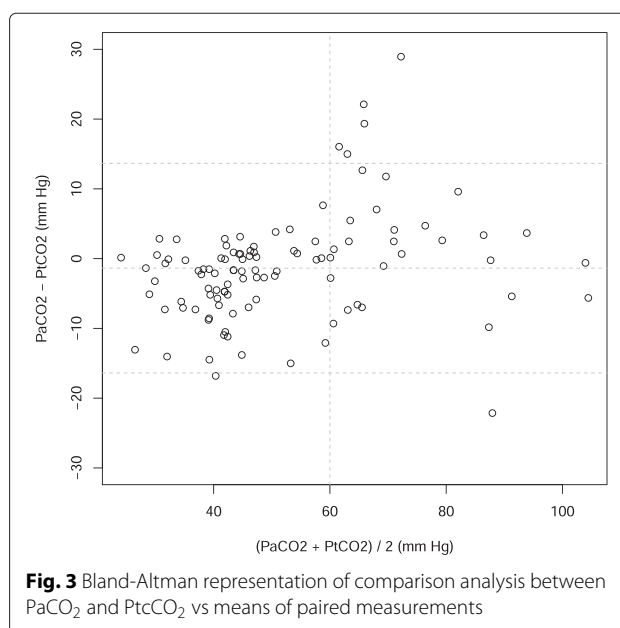


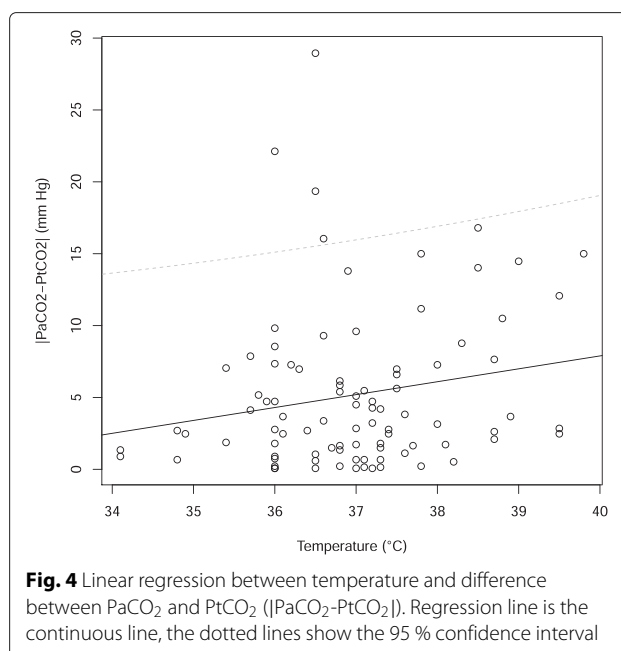
Fig. 3 Bland-Altman representation of comparison analysis between PaCO₂ and PtcCO₂ vs means of paired measurements

Table 2 Relationships between measurement differences ($|\text{PaCO}_2\text{-PtCO}_2|$) and patient characteristics using univariate and multivariate analysis (ANCOVA)

Variable	Univariate analysis		Multivariate analysis	
	OR [95 % CIs]	P-value	OR [95 % CIs]	P-value
Sex	1.61 [0.18, 14.25]	.66		
Past medical history				
Acute pulmonary edema	0.29 [0.03, 2.96]	.29		
COPD	0.60 [0.06, 6.18]	.67		
Ischemic heart disease	0.75 [0.06, 8.91]	.82		
Home oxygen	1.01 [0.06, 16.77]	.99		
Heart rate	0.98 [0.94, 1.03]	.40		
Systolic blood pressure	1.01 [0.97, 1.05]	.60		
Diastolic blood pressure	0.97 [0.91, 1.03]	.33		
Respiratory rate	1.01 [0.88, 1.18]	.85		
Temperature	2.45 [0.93, 6.49]	.07	3.01 [1.16, 7.80]	.03
PaCO ₂	1.05 [1.00, 1.12]	.06	1.06 [1.00, 1.12]	.05
PtCO ₂	1.06 [1.00, 1.13]	.06		
PaO ₂	1.21 [1.01, 1.45]	.04	1.22 [1.02, 1.47]	.03
HCO ₃	0.96 [0.80, 1.15]	.64		
Base excess	0.96 [0.82, 1.12]	.60		
Lactate	1.44 [0.43, 4.79]	.54		
Hemoglobin	1.20 [0.74, 1.95]	.45		
White blood cells	0.91 [0.74, 1.12]	.38		
C-reactive protein	1.00 [0.99, 1.01]	.72		
Glycemia	4.70 [0.62, 35.58]	.13		
Brain natriuretic peptide	1.00 [1.00, 1.00]	.82		

this issue may occur with any change of device. Thirdly, we did not correct the arterial blood gases according to the patient's temperature and this may explain a portion of the increased difference. Finally, our population was more likely to have significant dyspnoea and therefore agitation or diaphoresis leading to movement of the sensor may have led to inaccurate measurements. Indeed, in the Gancel et al. study [12] where the difference was lower, the exclusion criteria were very rigorous. Authors did not study patients with status epilepticus, confusion or agitation. According to the authors, these criteria may have led to the exclusion of some patients with severe hypercapnia.

Our study found that PtCO₂ values were generally greater than PaCO₂ values. Indeed, our linear regression equation is $\text{PaCO}_2 = (0.81 \times \text{PtCO}_2) + 10.86$. This overestimation is in accordance with available literature

**Fig. 4** Linear regression between temperature and difference between PaCO₂ and PtCO₂ ($|\text{PaCO}_2\text{-PtCO}_2|$). Regression line is the continuous line, the dotted lines show the 95 % confidence interval

[10, 22, 23] and may have implications for patients requiring non-invasive ventilation and with no arterial blood gas reference. Thus, the recommendations highlight the need to conduct an arterial blood gas analysis to support the correlation between PaCO₂ and PtCO₂ values [6]. This issue is important given that the Bland-Altman analysis reveals a poorer correlation for PtCO₂ values above 60 mm Hg. The value of the mean bias reported in our study corresponds to those found in the literature (-1.4 to 4.6 mm Hg) [16, 24, 25]. The decrease of the correlation for high PaCO₂ values has been previously reported. The accuracy of PtCO₂ seems to be better for patients with PaCO₂ values below 56 mm Hg [26]. One explanation for this poor correlation is that the clinical manifestations of hypercapnia (excessive sweating and vasodilatation) leads to a lower diffusion of carbon dioxide [26]. In our study, after multivariate analysis, temperature was associated with a poor correlation between PaCO₂ and PtCO₂ (OR = 3.01; 95 % CIs [1.16, 7.80]; $p = .03$). The issue that the temperature can influence the correlation has been raised by Rodriguez et al. [27]. Our linear regression analysis revealed that the higher the body temperature, the greater the difference between PaCO₂ and PtCO₂ values. This poor correlation can be explained by the fact that as the patient's temperature increased, the difference between the patient and the temperature sensor (44 °C) decreased, resulting in small changes in local perfusion and production of CO₂. This hypothesis follows directly from the operating principle of the sensor [6]. It could also be hypothesized that a high body temperature promotes sweating and vasodilatation making the sensor's

measurement more inaccurate. Finally, a low blood pressure could also be a cause of a poor correlation between PaCO₂ and PtCO₂ [28]. Unfortunately, this hypothesis cannot be confirmed by our data because few patients had shock criteria. Similarly, the assumption that the pH may explain a poor correlation [21] cannot not be confirmed in our study with the multivariate analysis.

Limitations

Although several studies have found a poor correlation between PaCO₂ and PtCO₂ in patients with shock who are treated with catecholamines [10], we did not analyze this particular relationship. Indeed, we included few patients with hemodynamic instability requiring the administration of intravenous fluids or vasopressor support. It is therefore difficult to assess the impact of decreased circulation on the correlation between PaCO₂ and PtCO₂. Several studies have shown that the correlation is not affected by catecholamines but by dermal vasoconstriction secondary to a state of shock [23, 27].

Secondly, body mass index (BMI) was not measured in our study. Several studies reported conflicting conclusions regarding the influence of skin thickness, indirectly estimated by BMI, on the CO₂ diffusion to the skin and therefore on the PaCO₂ values [10, 23, 25, 26]. However, there is no correlation between BMI and the skin on the earlobe, where the sensor was fixed [29].

Finally, one subject that remains to be explored is the intra-individual correlation. Most of the patients had only one arterial blood gas measurement during their management in the RR, which was inadequate for obtaining intra-individual correlations between different PtCO₂ and PaCO₂ values. This analysis would be important to predict PaCO₂ values from continuous measurement of PtCO₂, especially for patients requiring several hours of monitoring [27, 30].

Conclusions

There is a significant correlation between PaCO₂ and PtCO₂ values for patients admitted to the ED for acute respiratory failure. This correlation is particularly accurate for values below 60 mm Hg. One limiting factor to routine use of PtCO₂ measurements in the ED is the presence of hyperthermia.

Key messages

- There is a significant correlation between PaCO₂ and PtCO₂ values for patients admitted to the emergency department for acute respiratory failure.
- This correlation is comparable to that which has been shown in intensive care.
- One limiting factor to the use of PtCO₂ measurements in the ED is the presence of hyperthermia.

Appendix 1: Definition of Canadian triage and acuity scale (CTAS) level 1 and level 2 for dyspnoea Patients with dyspnoea and CTAS Level 1:

Severe respiratory distress: serious intracranial events, pneumothorax, near death asthma (unable to speak, cyanosis, lethargic/confused, tachycardia/bradycardia, arterial oxygen saturation below 90 %), chronic obstructive pulmonary disease exacerbations, cardiac heart failure, anaphylaxis and severe metabolic disturbances (renal failure, diabetic keto-acidosis).

Patients with dyspnoea and CTAS Level 2:

Asthma: severe asthma defined with a combination of objective measures and clinical factors which relate to the severity of symptoms, vital signs and history of previous severe episodes. If the forced expiratory volume in 1 second or peak expiratory flow rate are below 40 % predicted or previous best, the patient is considered severe.

Dyspnea: this is subjective and may correlate poorly with lung function or deficits in oxygen uptake and delivery. Depending on the age, previous history and physical assessment one may not be able to distinguish between asthma chronic obstructive pulmonary disease, cardiac heart failure, pulmonary embolism, pneumothorax, pneumonia, croup, epiglottitis, anaphylaxis or a combination of problems.

Abbreviations

ANCOVA: Analysis of covariance; BMI: Body mass index; CI: Confidence interval; CTAS: Canadian triage and acuity scale; COPD: Chronic obstructive pulmonary disease; CRF: Case report form; ED: Emergency department; EtCO₂: End tidal CO₂; ICU: Intensive care unit; IQR: Interquartile range; OR: Odds ratio; RR: Resuscitation room; PaCO₂: Partial pressure of carbon dioxide in the blood; PtCO₂: Transcutaneous partial pressure of carbon dioxide in the blood; PaO₂: Partial pressure of dioxygen in the blood; SaO₂: Arterial oxygen saturation; SpO₂: Oxygen saturation by pulse oxymetry.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

XB, PGC, LP and MR conceived the study and wrote the manuscript. JEdLC was the director of this research project and participated to the writing of this manuscript. LP and MR performed the exams. PGC was the responsible of the statistical analysis. XB, PGC, LP, MR, RGG, CR, JY, PR, MS, LM and JEdLC have been involved in drafting the manuscript or revising it critically for important intellectual content. XB, PGC, LP, MR, RGG, CR, JY, PR, MS, LM and JEdLC have given final approval of the version to be published and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Authors' information

Xavier Bobbia and Pierre-Géraud Claret have contributed equally and are both first authors.

Acknowledgements

The authors acknowledge all the emergency physicians at Nîmes University Hospital who recruited the patients, the residents who helped in this process, and other members of our research team for their help.

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Received: 7 September 2015 Accepted: 7 September 2015

Published online: 06 October 2015

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