

## Transcutaneous versus blood carbon dioxide monitoring during acute noninvasive ventilation in the emergency department – a retrospective analysis

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### Summary

**QUESTIONS UNDER STUDY:** Transcutaneous measurement of carbon dioxide (PtCO<sub>2</sub>) has been suggested as an alternative to invasively obtained PaCO<sub>2</sub> for the monitoring of patients with hypercapnic respiratory failure during noninvasive ventilation (NIV). Current data on monitoring in hypoxaemic respiratory failure are scarce and show conflicting results in hypercapnic patients in the emergency department.

**METHODS AND SETTING:** We performed a retrospective comparison of real-time PtCO<sub>2</sub> (SenTec Digital Monitor) and arterial/venous carbon dioxide tension (PaCO<sub>2</sub>/PvCO<sub>2</sub>) measurements in patients with severe hypoxaemic and/or hypercapnic respiratory failure during NIV. Agreement between PtCO<sub>2</sub> and PaCO<sub>2</sub>/PvCO<sub>2</sub> was the primary endpoint. Bland-Altman analysis and linear regression were used.

**RESULTS:** 102 patients had at least one matched measurement of PtCO<sub>2</sub> and PaCO<sub>2</sub>/PvCO<sub>2</sub>. For patients with arterial blood gas analysis, the mean difference was 0.46 kPa at baseline (95% confidence interval [CI] 0.23 to 0.60, limits of agreement 95% CI –0.54 to 1.45) and 0.12 kPa after NIV (95% CI –0.04 to 0.29, limits of agreement 95% CI: –0.61 to 0.86). The linear regression analysis found a correlation  $R^2$  of 0.88 ( $p < 0.001$ ) at baseline and an  $R^2$  of 0.99 ( $p < 0.001$ ) after initiating NIV. For patients with venous blood gas analysis, the mean difference was 0.64 kPa at baseline (95% CI 0.04 to 1.24, limits of agreement 95% CI –0.72 to 2) and 0.80 kPa after NIV (95% CI 0.51 to 1.10, limits of agreement 95% CI 0.29 to 1.32),  $R^2$  0.78 ( $p < 0.001$ ) at baseline and  $R^2$  0.91 ( $p < 0.001$ ) after initiating NIV.

A PaCO<sub>2</sub>/PvCO<sub>2</sub> >8 kPa was associated with a lesser degree of agreement between the levels of PtCO<sub>2</sub> and PaCO<sub>2</sub>/PvCO<sub>2</sub> ( $p < 0.001$ ).

**CONCLUSION:** Transcutaneous PCO<sub>2</sub> monitoring shows a good concordance with PaCO<sub>2</sub> and is a reliable, feasible, patient-friendly and safe alternative to repeated blood gas analysis for patients with severe hypoxaemic and/or hypercapnic respiratory failure receiving emergency NIV in the emergency department. An initial blood gas analysis to evaluate the respiratory and metabolic state and to rule out

a significant discrepancy compared with the transcutaneous measurement is recommended.

Trials registration number: EKSG13/118

**Key words:** noninvasive ventilation; respiratory failure; transcutaneous CO<sub>2</sub> measurement; emergency

### Introduction

Transcutaneous measurement of carbon dioxide (PtCO<sub>2</sub>) offers constant monitoring of CO<sub>2</sub> and is a pain-free alternative to arterial blood gas analysis [1], which has more potential side effects (bleeding, haematoma, thrombosis [2]) and results in only a snapshot compared with continuous monitoring. In a number of studies, PtCO<sub>2</sub> monitoring has been shown to be a reliable alternative to measurement of arterial carbon dioxide tension (PaCO<sub>2</sub>) [3–13]. This was also demonstrated in hypotensive patients (mean arterial pressure <60 mm Hg) [14].

However, there are conflicting results in patients undergoing noninvasive ventilation (NIV) [3, 15, 16]. Only two studies were performed in patients with acute respiratory failure in the emergency department (ED) [15, 17]. The first study concluded that PtCO<sub>2</sub> levels had an unacceptably wide margin compared with PaCO<sub>2</sub>, whereas the second demonstrated a good correlation between the respective measurements. In previous studies [3, 15], a margin of 10 mm Hg (≈1.33 kPa) has been suggested as the maximum acceptable disagreement between techniques. However, all of these studies compare PtCO<sub>2</sub> with PaCO<sub>2</sub>, despite venous blood gases emerging as a potential replacement for arterial blood gases in emergency situations [18–20]. In our non-university hospital, we regularly and successfully use PtCO<sub>2</sub> monitoring backed up with an initial invasive blood gas analysis (BGA) to guide the adaptation of NIV settings in the ED. This approach is implemented in the German guidelines for NIV as a monitoring tool [21], and a recent study [17] in hypercapnic patients showed a good correlation between hypothetical NIV changes compared with true management.

We hypothesised that there would be no clinically significant difference ( $\leq 1$  kPa) at baseline and after acute NIV between transcutaneous and arterial measurements of  $\text{CO}_2$  in patients with severe respiratory failure. We also included patients who had only venous blood gas analysis, since it is essentially measured in every patient in our medical emergency department and we were sometimes unable to draw arterial blood for gas analysis. We arbitrarily choose 1 kPa as a clinically significant difference.

## Material and methods

### Study setting and population

This was an observational and retrospective analysis. All patients undergoing NIV because of severe respiratory failure in the ED of our hospital (Kantonsspital St. Gallen, Switzerland) from 1 March 2012 to 28 February 2014 and who had at least one BGA, either venous (vBGA) or arterial (aBGA), and matching  $\text{PtCO}_2$  measurements were included in the study. At baseline, the time difference between BGA and start of the  $\text{PtCO}_2$  measurements was not defined; after initiation of NIV, BGA and  $\text{PtCO}_2$  were synchronously recorded. Severe respiratory failure was defined as hypoxaemic respiratory failure (respiratory rate [RR] above 25/min and an oxygen saturation [ $\text{SpO}_2$ ] of below 92% despite supplemental oxygen at 6 l/min) and/or hypercapnic respiratory failure (pH  $< 7.35$  and either hypercapnia [ $\text{PaCO}_2 > 6.0$  kPa] or severe hypercapnia [ $\text{PaCO}_2 > 7.4$  kPa]). We excluded patients who arrived intubated at the emergency department. For NIV, we used a portable homecare ventilator (Stellar<sup>TM</sup> 100 or Stellar<sup>TM</sup> 150, Resmed Ltd, Bella Vista, Australia).

### Endpoints

The primary endpoint was the difference between arterial or venous carbon dioxide tension ( $\text{PaCO}_2/\text{PvCO}_2$ ) and  $\text{PtCO}_2$  at admission and after up to 2 hours of NIV. We arbitrarily choose 1 kPa as a clinically significant difference. Secondary endpoints were feasibility of transcutaneous  $\text{PCO}_2$  monitoring, evolution of values of  $\text{PtCO}_2$  versus the evolution of values of  $\text{PaCO}_2$  (no change defined as within  $\pm 0.5$  kPa and change defined as  $> \pm 0.5$  kPa) and whether heart rate, respiratory rate, body temperature, body mass index (BMI), forced expiratory volume in 1 second (FEV1), haemoglobin levels,  $\text{PaCO}_2$  values, pH, underlying disease (acute cardiac pulmonary oedema [ACPE], lower respiratory tract infection [LRTI], exacerbated chronic obstructive pulmonary disease [COPD], COPD and LRTI, other) or the time gap between blood gas and  $\text{PtCO}_2$  measurement had any influence.

### Data collection and material

Data retrospectively collected from medical records included indication for NIV, heart rate (measured via electrocardiogram), respiratory rate (measured via electrocardiogram), blood pressure, failure of  $\text{PtCO}_2$  measurement, patient demographics, BMI, FEV1, haemoglobin levels and the time gap between drawing a blood sample and the measurement of  $\text{PtCO}_2$ . The latter was calculated as the interval from the point of time when a sample for BGA was

drawn to the point of time when the first value from the  $\text{PtCO}_2$  measurements was collected at baseline; during NIV the  $\text{PtCO}_2$  values were recorded simultaneously with the sampling for BGA. Venous or arterial BGA were analysed using a Radiometer ABL800 Flex (Thalwil, Switzerland) and the blood samples were drawn by the attending physicians of the ED.  $\text{PtCO}_2$  was measured using a SenTec Digital Monitor SDM (SMB SW 07.02.2, V-STATS/VCARE 3.01, Therwil, Switzerland) using a V-Sign sensor. V-Sign sensors use a Stow-Severinghaus type  $\text{PtCO}_2$  sensor measurement segment. In addition, V-Sign sensors also include  $\text{SpO}_2$  measurement and a heating segment. Probes were placed on the earlobe of the patients using a conductive lotion, as proposed by the manufacturer. The corresponding values were recorded as soon as the colour of the  $\text{PtCO}_2$  indicator turned from grey to green, marking a stable value (the earliest values were taken 5 min after sensor application). The sensor was attached solely by our team of respiratory therapists, consisting of nine people. Every 5 min,  $\text{PtCO}_2$  values,  $\text{SpO}_2$ , RR, heart rate and blood pressure were recorded. In addition, from 2014 onwards,  $\text{PtCO}_2$  measurements were saved as PDF files. Drift correction of  $\text{PtCO}_2$  was not performed, since we considered this to be negligible owing to the minimal changes reported earlier ( $< 0.1$  kPa/h) [3].

### Statistical analysis

The agreement between  $\text{PtCO}_2$  and  $\text{PaCO}_2/\text{PvCO}_2$  was assessed using the Bland-Altman procedure [22]. Estimated biases (mean difference between the two methods) are given together with their 95% confidence intervals (CIs). The 95% limits of agreement (2 x standard deviations of the difference) are also reported. In addition, linear regression was used and the estimate of the slope of the regression line is provided together with its standard error. A significance level of 5% was chosen. All data are reported as median with interquartile range. All analyses were done using the R statistical software [23].

### Ethics

The study was approved by the Ethics Committee of the Canton of St. Gallen and was registered under EKSG13/118. Informed patient consent was waived by the institutional ethics board.

## Results

Baseline characteristics of the patients are shown in table 1. The patient flow chart is shown in figure 1. Thirteen of the 102 patients treated with NIV were subsequently intubated. In figure 2, two examples of NIV using  $\text{PtCO}_2$  measurement are demonstrated, showing a good correlation with the respective aBGA and also showing how quickly  $\text{PtCO}_2$  changes.

Of a total of 121 identified cases, 102 had  $\text{PtCO}_2$  and  $\text{PaCO}_2/\text{PvCO}_2$  measurements, 100 at baseline and 95 before and after intervention. In the remaining 19 patients, either no BGA or no measurement of  $\text{PtCO}_2$  was recorded. For patients with aBGA, a Bland-Altman analysis and a linear regression analysis are depicted in figure 3A–D. The mean difference was 0.46 kPa at baseline (95% CI 0.23 to

0.60, limits of agreement 95% CI -0.54 to 1.45) (fig. 3A) and 0.12 kPa after NIV (95% CI -0.04 to 0.29, limits of agreement 95% CI -0.61 to 0.86) (fig. 3C). The linear regression analysis found a correlation of  $R^2$  0.88 ( $p < 0.001$ )

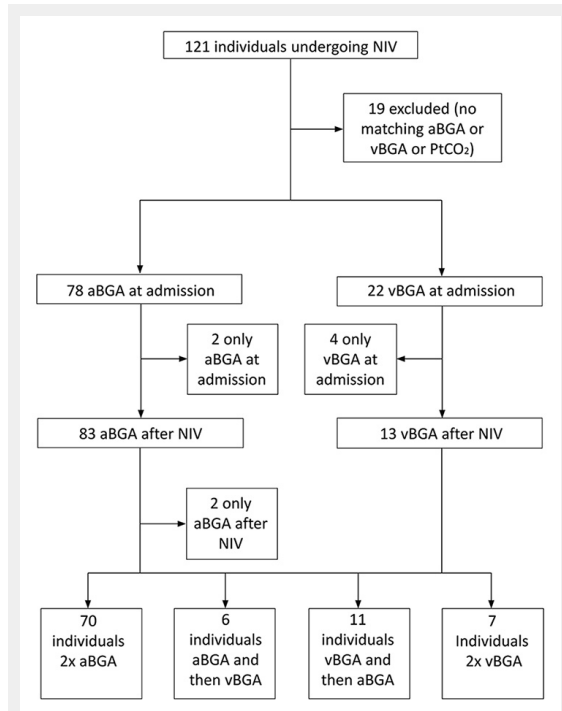


Figure 1

Study flow diagram.

aBGA = arterial blood gas analysis; NIV = noninvasive ventilation; PtCO<sub>2</sub> = transcutaneous partial pressure of CO<sub>2</sub>; vBGA = venous blood gas analysis

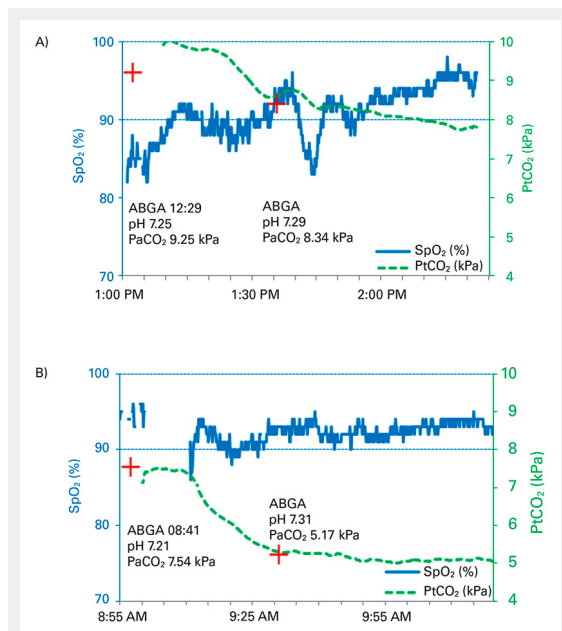


Figure 2

Real-time PtCO<sub>2</sub> monitoring of two patients undergoing noninvasive ventilation. (A) Exacerbated chronic obstructive pulmonary disease. (B) Acute cardiac pulmonary oedema. The red cross indicates the PaCO<sub>2</sub> values from the arterial blood gas analysis, PtCO<sub>2</sub> values (dashed lines) and SpO<sub>2</sub> values (solid lines) are demonstrated. ABGA = arterial blood gas analysis; PaCO<sub>2</sub> = arterial partial pressure of CO<sub>2</sub>; SpO<sub>2</sub> = oxygen saturation

at baseline (fig. 3B) and of  $R^2$  0.99 ( $p < 0.001$ ) after initiating NIV (fig. 3D). In patients with a time gap  $< 60$  min between aBGA and PtCO<sub>2</sub> measurement at baseline, the mean difference was 0.52 kPa (95% CI 0.22 to 0.81, limit of agreement 95% CI -0.55 to 1.57),  $R^2$  0.88 ( $p < 0.001$ ). Subgroup analysis for patients with aBGA revealed a similar good correlation for the different disease groups (ACPE, LRTI, acute exacerbated COPD, COPD and LRTI and other, data not shown).

Absolute changes of PtCO<sub>2</sub> correlated in 73% of patients with no change in PaCO<sub>2</sub> ( $\pm 0.5$  kPa) and in 76% with changes in PaCO<sub>2</sub>  $> \pm 0.5$  kPa.

For patients with vBGA, the mean difference was 0.64 kPa at baseline (95% CI 0.04 to 1.24, limits of agreement 95% CI -0.72 to 2) and 0.80 kPa after NIV (95% CI 0.51 to 1.10, limits of agreement 95% CI 0.29 to 1.32),  $R^2$  0.78 ( $p < 0.001$ ) at baseline and  $R^2$  0.91 ( $p < 0.001$ ) after initiating NIV.

Univariate analysis of the effect of confounders on PtCO<sub>2</sub> revealed that only a PaCO<sub>2</sub>/PvCO<sub>2</sub>  $> 8$  kPa ( $n = 48$  at baseline) was independently associated with a widened margin of PtCO<sub>2</sub> ( $p < 0.001$ ). Residual standard error varied from 0.52 below 8 kPa up to 1.42 above 8 kPa. No other correlations were found (see table 2).

Problems with the PtCO<sub>2</sub> measurement probe occurred in three cases: once because of a very thick earlobe, once because of probe malfunction – the probe had to be replaced – and once owing to insufficient peripheral perfusion most possibly secondary to low blood pressure.

Table 1: Characteristics of patients at baseline.

Age (n = 102)	69 (59–77)
SBP (mm Hg) (n = 102)	138 (117–158)
MAP (mm Hg) (n = 102)	95 (86–108)
HR (beats/min) (n = 100)	95 (79–112)
BMI (kg/m <sup>2</sup> ) (n = 81)	28.4 (22.6–35)
FEV <sub>1</sub> (%) (n = 55)	43 (34.0–60)
RR (breaths/min) (n = 101)	28 (24.0–32)
Saturation of oxygen (%) (n = 102)	90 (86–94)
Oxygen supply (l/min) (n = 102)	3 (1–7)
pH overall (n = 100)	7.32 (7.25–7.39)
pH in hypercapnic patients (PaCO <sub>2</sub> $> 6$ kPa) (n = 53)	7.29 (7.24–7.34)
Temp (°C) (n = 91)	37 (36.5–37.4)
Hb (g/dl) (n = 101)	13.6 (12.1–15.2)
Time difference between BGA and PtCO <sub>2</sub> measurement (min) (n = 100)	35 (20–58)
PaCO <sub>2</sub> (kPa) (n = 78)	7.64 (5.31–9.25)
Underlying disease:	
AECOPD (n)	35
ACPE (n)	19
LRTI (n)	19
COPD+LRTI (n)	7
Other (n)	22

ACPE = acute cardiac pulmonary oedema; AECOPD = acute exacerbated COPD; BGA = blood gas analysis; BMI = body mass index; COPD = chronic obstructive pulmonary disease; Hb = haemoglobin; HR = heart rate; LRTI = lower respiratory tract infection; MAP = median blood pressure; PaCO<sub>2</sub> = arterial carbon dioxide tension; PtCO<sub>2</sub> = transmembraneous carbon dioxide tension; RR = respiratory rate; SBP = systolic blood pressure; Temp = temperature  
Values are reported as median with inter-quartile range.

## Discussion

To our knowledge, this is the largest study in patients with severe respiratory failure undergoing NIV in an ED, monitored with real time PtCO<sub>2</sub> measurements [3, 5, 16], and the only study to include patients with hypoxaemic failure. We created a unique setting by interpreting real-time PtCO<sub>2</sub>

Table 2: Confounding factors of concordance of PtCO <sub>2</sub> vs PvCO <sub>2</sub> /PaCO <sub>2</sub> measurement.	
Underlying disease:	
AECOPD (n = 35)	p = 0.46
ACPE (n = 19)	p = 0.96
LRTI (n = 19)	p = 0.91
COPD+LRTI (n = 7)	p = 0.28
Other (n = 22)	p = 0.56
PaCO <sub>2</sub> >8 kPa (n = 48)	p <0.001
Heart rate (beats/min) (n = 100)	p = 0.29
Time difference between BGA and PtCO <sub>2</sub> measurement (maximum 385 min) (n = 86)	p = 0.66
SBP (n = 102)	p = 0.97
SBP <90 mm Hg (n = 11)	p = 0.44
MAP (n = 102)	p = 0.77
MAP <60 mm Hg (n = 8)	p = 0.31
BMI (n = 81)	p = 0.11
Temp. (n = 91)	p = 0.42
FEV <sub>1</sub> (n = 55)	p = 0.90
RR (breaths/min) (n = 101)	p = 0.06
Age (n = 102)	p = 0.61
pH	p = 0.97

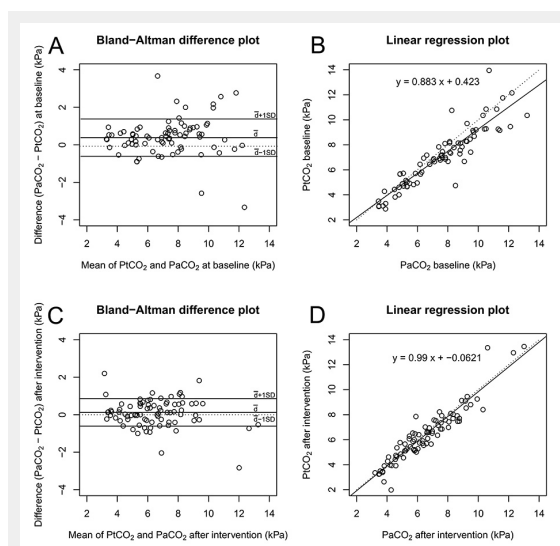
ACPE = acute cardiac pulmonary oedema; AECOPD = acute exacerbated COPD; BGA = blood gas analysis; BMI = body mass index; COPD = chronic obstructive pulmonary disease; FEV<sub>1</sub> = forced expiratory volume in 1 second; HR = heart rate; LRTI = lower respiratory tract infection; MAP = median blood pressure; PaCO<sub>2</sub> = arterial carbon dioxide tension; PtCO<sub>2</sub> = transmembraneous carbon dioxide tension; PvCO<sub>2</sub> = venous carbon dioxide tension; RR = respiratory rate; SBP = systolic blood pressure; Temp. = temperature

measurements and did not use the drift correction during acute NIV. At baseline, transcutaneous PCO<sub>2</sub> could not be obtained because of problems with the sensor in 3 (3%) cases. In all cases, the detection failure could be corrected within 1 h. In our setting, we found a good correlation of PtCO<sub>2</sub> and arterial PCO<sub>2</sub> at baseline, which was even better after intervention (fig. 3). The correlation was within the predefined clinically significant difference of 1 kPa in 75% of cases at baseline, which is not sufficient without prior validation with an aBGA. The better correlation after NIV is possibly directly related to the fact that CO<sub>2</sub> levels were reduced (often <8 kPa, fig. 3), thus enabling the device to operate in the optimised range. We likewise noted a good correlation between the trends for PtCO<sub>2</sub> and PaCO<sub>2</sub> before and after intervention, especially in patients with a change in PaCO<sub>2</sub> of ≥1 kPa. The former finding was also observed by Storre et al. [3] and Janssens and colleagues [16], but has never been formally investigated in patients undergoing acute NIV in the ED. The latter has been recently shown by the group of Van Oppen [17], although our trends of PtCO<sub>2</sub> and PaCO<sub>2</sub> were less well-correlated, probably as a result of the shorter observational time (up to 2 hours vs 12 hours) and because Van Oppen and colleagues included only hypercapnic patients in a different setting (medical high dependence unit). Therefore, this might be a good tool for real-time surveillance in this group of patients while in the emergency department but not a tool to measure the exact values of CO<sub>2</sub>.

Before this study, end-tidal CO<sub>2</sub> measurement was available in the ED but was rarely used. In comparison with transcutaneous PCO<sub>2</sub>, end-tidal CO<sub>2</sub> measurements are often misleading in this setting [24] owing to the frequently used vented systems of NIV, additional oxygen application or air leakage of facial masks. We thus believe that PtCO<sub>2</sub> could replace end-tidal CO<sub>2</sub> for monitoring patients in severe respiratory failure undergoing NIV in the ED of other hospitals as well, since it is patient-friendly, feasible, reliable and safe in emergency situations.

The difference between PaCO<sub>2</sub> and PtCO<sub>2</sub> increased with higher levels of PCO<sub>2</sub> (≥8 kPa). This is in agreement with the findings of other studies [13, 15]. We believe that this might be directly related to higher levels of CO<sub>2</sub> within the tissue leading to acidosis and vasoconstriction, thus potentially resulting in reduced blood flow and, therefore, interfering with the measurement. However, despite increasing differences between arterial and transcutaneous CO<sub>2</sub> levels, we found the use of PtCO<sub>2</sub> very helpful in patients with excessive hypercarbia because of the correct real-time trend supplied by the device.

The Sentec Digital Monitor offers a relatively short time to stabilisation and the sensor can be disconnected from the machine without the need for an additional calibration. With these advances, PtCO<sub>2</sub> monitoring could prove to be helpful in emergency situations and may even improve NIV guidance, because of the real-time documentation of ventilatory changes, especially in hypercapnic patients. Moreover, repeated aBGA may not be necessary to assess the success of NIV. Since PtCO<sub>2</sub> measurement is an indirect measure of PCO<sub>2</sub>, a synchronous BGA at baseline is recommended to validate the respective levels and appears to be mandatory in cases of PtCO<sub>2</sub> values ≥8 kPa. Evidently,



**Figure 3**

Comparison of transcutaneous and invasive PaCO<sub>2</sub> at baseline and after intervention. Measurements were compared using both a Bland-Altman plot (A, C) and a linear regression plot (B, D). A and B at baseline, C and D after NIV. The Bland-Altman plot displays the mean bias and limits of agreement (solid lines). The linear regression plot displays the line of best fit (solid line) and the identity line (dashed line).

BGA also offers additional information such as pH or lactate concentration. As an alternative, repeated vBGA might be useful as previously noted [18].

As shown in earlier studies, heart rate, blood pressure [14, 15], BMI [5] and skin temperature [25] had no significant influence on levels of PtCO<sub>2</sub>. The latter finding is in contrast to that of Bobbia and colleagues [13]. Furthermore, respiratory rate, FEV<sub>1</sub> and underlying disease did not influence PtCO<sub>2</sub> in our mixed patient group.

Since implementing PtCO<sub>2</sub> measurements, NIV has been used more often on the emergency department in our hospital, and from our perspective the demand for beds in the intensive care unit has diminished, since some of the patients could be directly transferred to the pneumology ward in our hospital.

There are some limitations to this study – especially its observational and retrospective nature. We cannot rule out a selection bias and also the time between BGA and measurement of PtCO<sub>2</sub> at baseline was not standardised. However, as a result of a high degree of adherence to a standardised NIV protocol in the ED in our hospital, we are optimistic that we have provided sufficient data quality for the current analysis. This is a single-centre study and we used only one type of sensor device, which limits the transferability of the results to other centres or other transcutaneous measurement devices.

## Conclusion

Transcutaneous PCO<sub>2</sub> monitoring shows a good concordance with PaCO<sub>2</sub> and is a reliable, feasible, patient-friendly and safe alternative to repeated blood gas analysis for patients with severe hypoxaemic and/or hypercapnic respiratory failure receiving emergency NIV in the ED. In addition, it can be used to adapt NIV settings in real time. An initial BGA to evaluate the respiratory and metabolic state and to rule out a significant discrepancy compared with the transcutaneous measurement is recommended.

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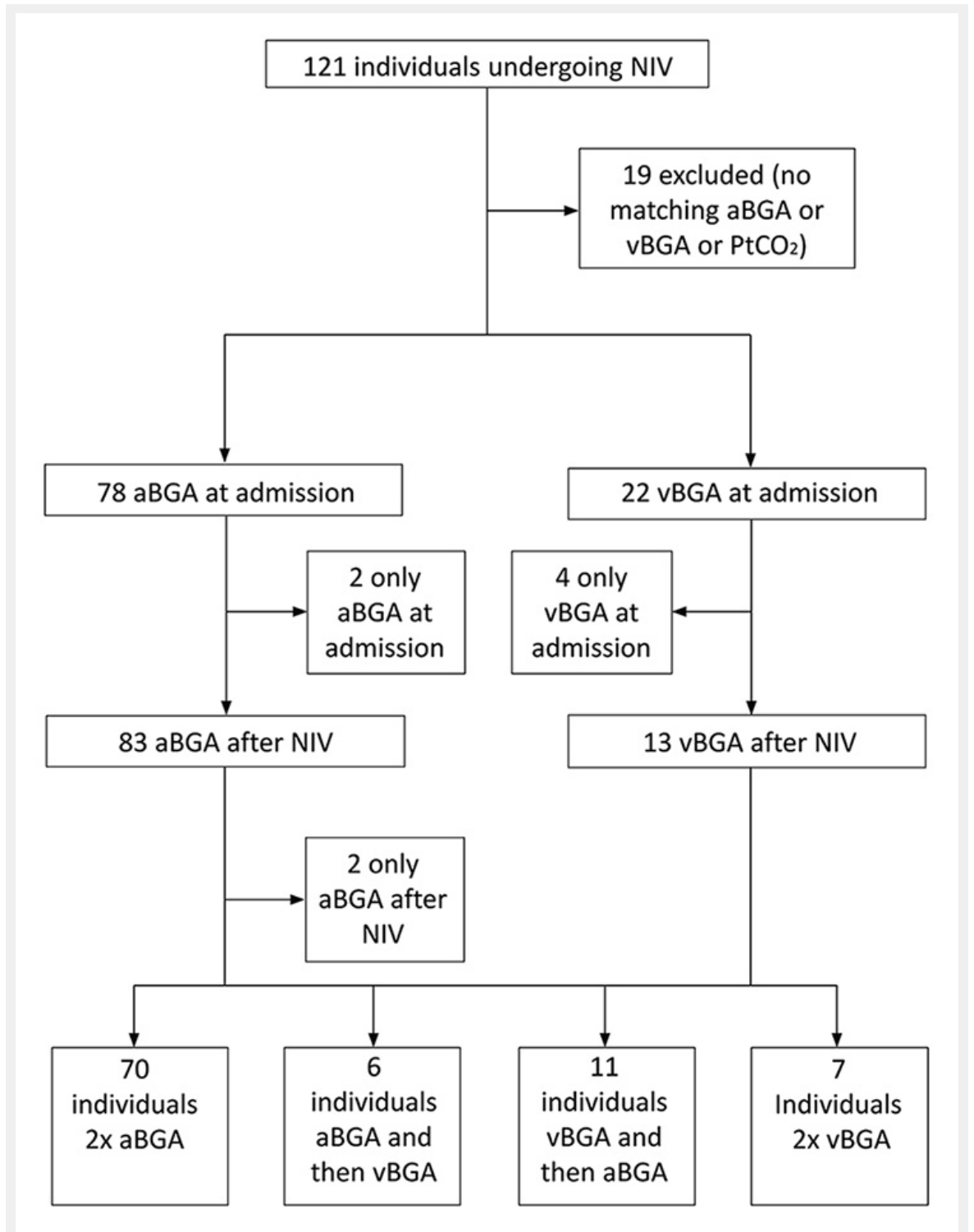
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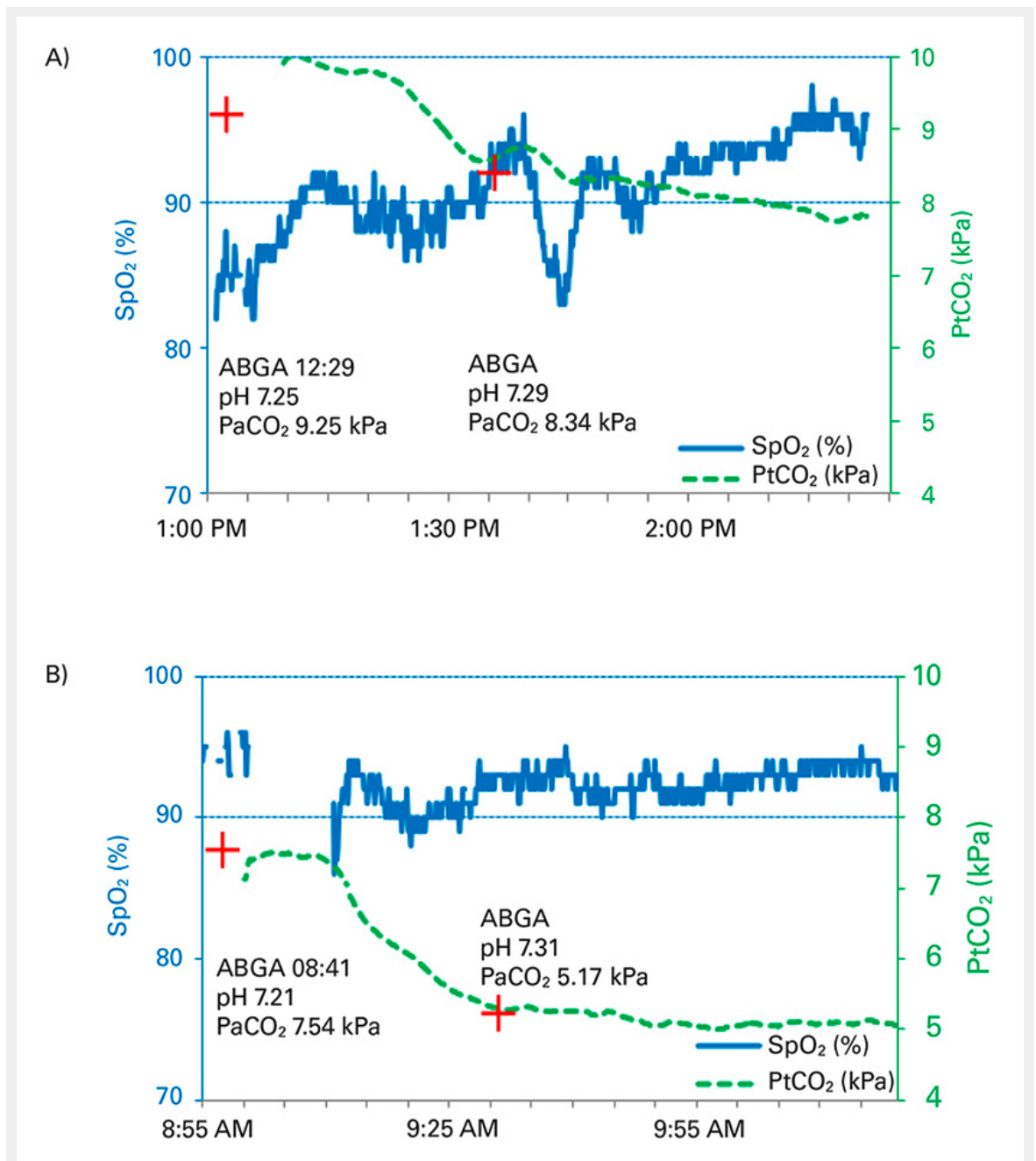
Figures (large format)



**Figure 1**

Study flow diagram.

aBGA = arterial blood gas analysis; NIV = noninvasive ventilation; PtCO<sub>2</sub> = transcutaneous partial pressure of CO<sub>2</sub>; vBGA = venous blood gas analysis

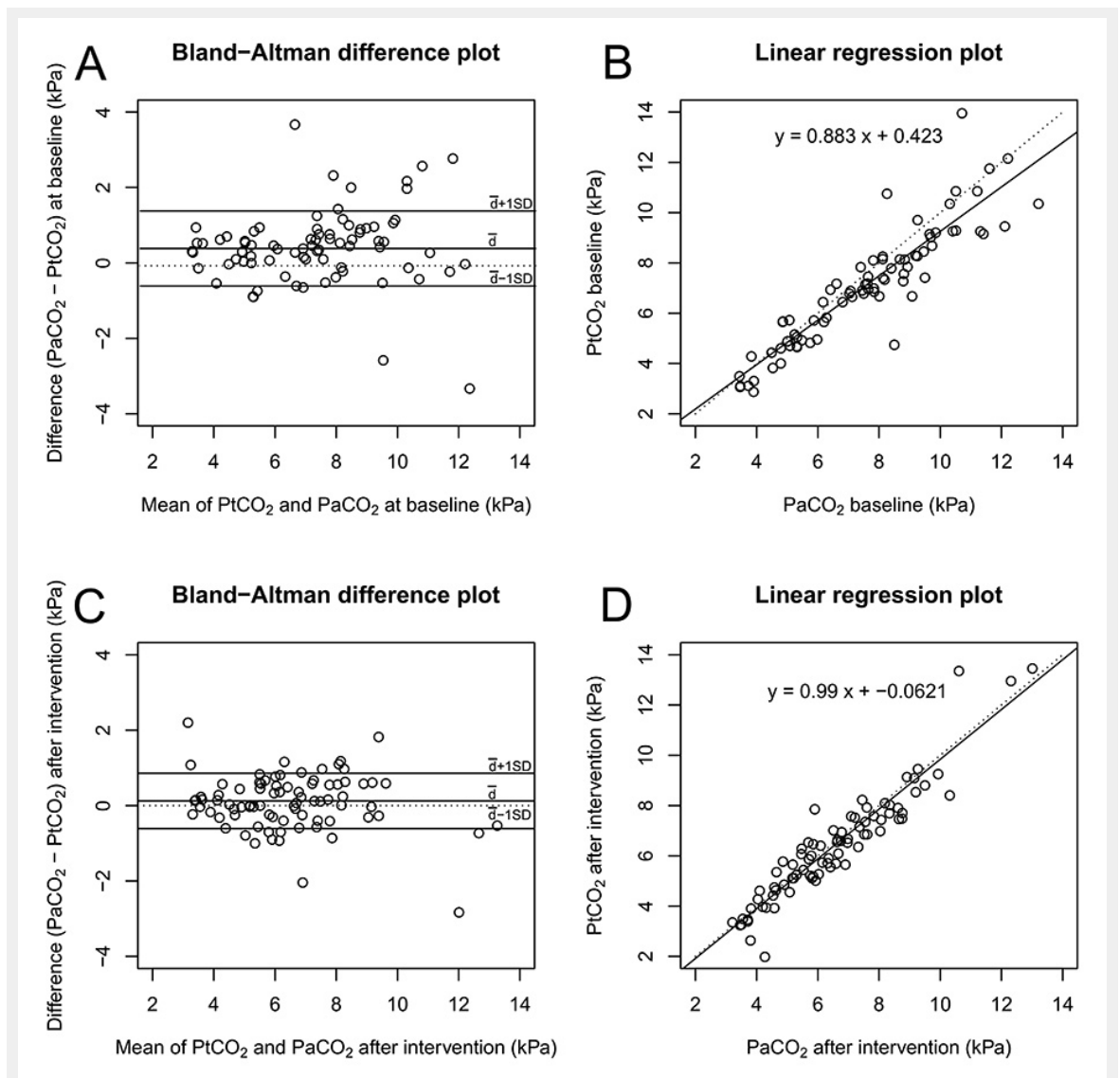


**Figure 2**

Real-time PtCO<sub>2</sub> monitoring of two patients undergoing noninvasive ventilation. (A) Exacerbated chronic obstructive pulmonary disease, (B) Acute cardiac pulmonary oedema. The red cross indicates the PaCO<sub>2</sub> values from the arterial blood gas analysis, PtCO<sub>2</sub> values (dashed lines) and SpO<sub>2</sub> values (solid lines) are demonstrated.

ABGA = arterial blood gas analysis; PaCO<sub>2</sub> = arterial partial pressure of CO<sub>2</sub>; SpO<sub>2</sub> = oxygen saturation





**Figure 3**

Comparison of transcutaneous and invasive PaCO<sub>2</sub> at baseline and after intervention. Measurements were compared using both a Bland-Altman plot (A, C) and a linear regression plot (B, D). A and B at baseline, C and D after NIV. The Bland-Altman plot displays the mean bias and limits of agreement (solid lines). The linear regression plot displays the line of best fit (solid line) and the identity line (dashed line).