

Comparison of Arterial and Venous Blood Gases and the Effects of Analysis Delay and Air Contamination on Arterial Samples in Patients with Chronic Obstructive Pulmonary Disease and Healthy Controls

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Key Words

Chronic obstructive pulmonary disease · Arterial blood gases · Venous blood gas

Abstract

Background: Arterial blood gases (ABGs) are often sampled incorrectly, leading to a 'mixed' or venous sample. Delays in analysis and air contamination are common. **Objectives:** We measured the effects of these errors in patients with chronic obstructive pulmonary disease (COPD) exacerbations and controls. **Methods:** Arterial and venous samples were analyzed from 30 patients with COPD exacerbation and 30 controls. Venous samples were analysed immediately and arterial samples separated into non-air-contaminated and air-contaminated specimens and analysed at 0, 30, 60, 90 and 180 min. **Results:** Mean venous pH was 7.371 and arterial pH was 7.407 ($p < 0.0001$). There was a correlation between venous and arterial pH ($r = 0.5347$, $p < 0.0001$). The regression equation to predict arterial pH was: arterial pH = $4.2289 + 0.43113 \cdot$ venous pH. There were no clinically significant differences in arterial PO₂ associated with analysis delay. A statistically significant decline in pH was detected at

30 min in patients with COPD exacerbation ($p = 0.0042$) and 90 min in controls ($p < 0.0001$). A clinically significant decline in pH emerged at 73 min in patients with COPD exacerbation and 87 min in controls. Air contamination was associated with a clinically significant increase in PO₂ in all samples, including those that were immediately analyzed. **Conclusions:** Arterial and venous pH differ significantly. Venous pH cannot accurately replace arterial pH. Temporal delays in ABG analysis result in a significant decline in measured pH. ABGs should be analysed within 30 min. Air contamination leads to an immediate increase in measured PO₂, indicating that air-contaminated ABGs should be discarded.

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Arterial blood gas (ABG) analysis is an essential tool for the assessment of patients with a wide range of clinical presentations. ABGs provide data relevant to acid-base status, ventilation, and arterial oxygenation and may help confirm a clinical diagnosis, ascertain the severity of a clinical condition (decompensated hypercapnic respiratory failure, metabolic acidosis, pulmonary embolism), the need for further interventions and the intensity

of monitoring required [1–3]. ABGs are used both in the acute setting and for assessing patients' gas exchange status during periods of clinical stability.

Arterial blood is obtained by percutaneous needle puncture or arterial cannulation of a palpable artery, typically the radial, brachial or femoral artery. Many factors may alter the accuracy of results, including the type of syringe used, the presence of air bubbles, specimen handling, delay in sample analysis, blood gas analyzer and storage temperature of the specimen [4–9]. Complications of the procedure include local pain, haematoma, arterial spasm and occlusion, vessel trauma, air or clotted-blood emboli, infection and needle stick injury to the sampler [10, 11].

In clinical practice, ABGs are often sampled incorrectly, leading to a 'mixed' or venous sample. Furthermore, it may be difficult to obtain arterial samples in certain patients. Pulse oximetry measurements of arterial oxygen saturation correlate well with oxygen saturations as measured by ABG, and transcutaneous carbon dioxide tension can provide a useful guide to the adequacy of ventilation [12]. However, neither can measure arterial pH. Cellular metabolism and organ function are optimally maintained over a relatively narrow range of pH, requiring close regulation of acid-base status. It would be of great clinical utility if venous blood pH could be used as a surrogate for ABG pH, protecting the patient from a painful and potentially hazardous investigation.

Studies performed in the emergency department have suggested that venous and arterial pH do not differ significantly and that venous pH can be used as a direct substitute for arterial pH in this setting [13, 14]. Studies performed in paediatric patients suggest that venous and arterial pH differ significantly, but that they are highly correlated [15–17]. Furthermore, a recent study suggests that venous PCO₂ can predict arterial hypercapnia [18].

Delayed analysis and air contamination of ABG specimens are common in clinical practice. Samples frequently remain in specimen trays at room temperature until hospital staff can transport the specimen to the laboratory and until the medical scientist can analyze the sample. Although the clinician may suspect aberrant results, the exact temporal effects of analysis delay and air contamination are not clear.

We sought to determine whether venous blood gas values differ from arterial values in normal subjects and patients with acute chronic obstructive pulmonary disease (COPD) exacerbations and to determine the extent of any such differences, to determine changes in ABG measurements associated with analysis delay and with the pres-

Table 1. Characteristics of the patients

	Controls	COPD exacerbations
Age, years	68.1 ± 13.76	71.7 ± 9.49
Sex, M/F	21/9	22/8
Hb concentration, g/dl	12.9 ± 1.71	13.2 ± 1.39

ence of air contamination ('air bubble') in the specimen over time and to determine recommended time limits within which ABGs should be analyzed.

Methods

Study Design

Sixty subjects were recruited between November 2006 and December 2008 from the emergency department and wards of the Mercy University Hospital, Cork, Ireland. The study protocol was approved by the Clinical Research Ethics Committee of the Cork Teaching Hospitals and written informed consent was required for participation. Thirty subjects were admitted to hospital with COPD exacerbation. Thirty control subjects with no history of respiratory disease were recruited from the inpatient population, age and sex matched to the COPD group. Control subjects were recruited before elective surgical procedures. Current or recent smokers (1 month) and patients with non-respiratory acid-base disturbance, including renal impairment, diabetic ketoacidosis, lactic acidosis, ethanol intoxication, acetazolamide, metformin or diuretic use, vomiting or diarrhoea within 24 h and salicylate overdose were excluded from the study. Table 1 describes the patients' characteristics.

Methods

Two milliliters of venous blood and 10 ml of arterial blood were collected consecutively from the antecubital vein and radial artery in identical plastic heparin-coated syringes with 23-gauge needles. Arterial samples were immediately separated into 10 samples of 1 ml each in standard ABG syringes. Five samples were unaltered (non-air-contaminated) and 5 samples were reconstituted with 0.8 ml blood with 0.2 ml air (air contaminated). The venous sample, one sample from the non-air-contaminated group and one sample from the air-contaminated group were immediately analyzed (T = 0 min), and one sample from each arterial group was kept at room temperature and analyzed at 30, 60, 90 and 180 min using an AVL OMNI-3 blood gas analyzer (Roche Diagnostics, Graz, Austria). ABGs were collected and analyzed by doctors experienced in the procedure. All data pertaining to the study were recorded in an anonymized computerized database. A change in PO₂ and PCO₂ of 1 kPa (7.5 mm Hg) and a change in pH of 0.02 were considered clinically significant. The change in pH that was deemed clinically significant was chosen to reflect the fact that electrode accuracy during pH measurement may vary by up to 0.01.

Analysis

Kolmogorov-Smirnov testing was used to determine whether data were normally distributed. Normally distributed data were compared using two-tailed parametric testing. Non-normally distributed data were compared using two-tailed non-parametric testing. Correlations between values were determined using Pearson (standard) and Spearman (non-parametric) testing. Linear regression was used to determine prediction algorithms for venous versus arterial samples. Comparisons at time points between groups were made using two-tailed parametric testing or non-parametric testing, depending on whether data are normally distributed. Comparisons across time points were made using repeated measures analysis of variance (ANOVA). Statistical significance was assumed at $p < 0.05$. Data were analyzed using GraphPad Software (San Diego, Calif., USA).

Results

Comparison of Arterial and Venous Blood Gases

There was a statistically significant difference between venous and arterial blood gas measurements in all measured parameters, with the exception of base excess in patients with COPD (table 2).

Temporal Effects of Analysis Delay in ABG Samples in Patients with COPD Exacerbations and Healthy Controls

There was a statistically significant increase in PO_2 after 90 min ($p = 0.0378$) and 180 min ($p = 0.0004$) in the control group. However, these differences were not clinically significant. There were no statistically or clinically significant differences in PO_2 in the COPD exacerbation group (fig. 1a) (table 3).

Arterial pH declined in both groups and this decline was more pronounced in patients with COPD exacerbations. A statistically significant decline in pH was detected at 30 min in patients with COPD exacerbations ($p = 0.0042$) and 90 min in healthy controls ($p < 0.0001$). A clinically significant decline in pH was detected at 73 min in patients with COPD exacerbations and at 87 min in controls (fig. 1b).

A statistically significant (but not clinically significant) rise in PCO_2 was detected at 60 min in both groups that was sustained through to 180 min (fig. 1c).

Effects of Air Contamination in ABG Samples in Patients with COPD Exacerbations and Healthy Controls

Air contamination was associated with a clinically and statistically significant increase in PO_2 in all samples, including those that were immediately analysed (fig. 2a). Over 180 min, there was a progressive rise in PO_2 as time

Table 2. Comparison of arterial and venous blood gases

	Venous blood gas	Arterial blood gas	p value
PO_2 , kPa			
COPD	5.142	9.022	<0.0001
Controls	4.412	10.677	<0.0001
pH			
COPD	7.367	7.399	<0.0001
Controls	7.376	7.415	0.0002
PCO_2 , kPa			
COPD	7.428	6.626	<0.0001
Controls	6.461	5.433	<0.0001
Bicarbonate, mmol/l			
COPD	31.06	29.66	<0.0001
Controls	27.50	25.53	<0.0001
Base excess, mmol/l			
COPD	4.47	4.14	0.0771
Controls	1.59	0.92	0.0007
O_2 saturation			
COPD	64.36	92.06	<0.0001
Controls	51.58	96.35	<0.0001

These differences were similar within the COPD exacerbation and control subgroups. Mean venous pH was 7.371 and mean arterial pH was 7.407 ($p < 0.0001$) (fig. 3). There was a statistically significant correlation between venous and arterial pH ($r = 0.5347$, $p < 0.0001$) (fig. 4). The regression equation to predict arterial pH from venous pH was: arterial pH = $4.2289 + 0.43113 \cdot$ venous pH.

evolved in air-contaminated samples that was more pronounced in healthy controls than in patients with COPD exacerbation. Air contamination was associated with attenuation of the rate of decline in arterial pH compared with non-air-contaminated samples in healthy controls and patients with COPD exacerbation that was clinically and statistically significant at 180 min (fig. 2b). Air contamination was associated with a statistically significant attenuation of the progressive rise in PCO_2 that was noted in analysis delay samples. However, there were no clinically significant differences in PCO_2 across all time points (fig. 2c).

Discussion

Venous pH cannot be used as a surrogate for arterial pH. Temporal delays in ABG analysis result in a decline in measured pH values that is detectable after 30 min in patients with COPD exacerbation and becomes clinically significant after 42 min. For this reason, the au-

Table 3. Effects of analysis delay in ABGs

	0 min	30 min	60 min	90 min	180 min	ANOVA, p
PO ₂ , COPD, kPa	9.02 ± 2.470	8.94 ± 2.214	8.80 ± 1.984	8.77 ± 1.891	8.84 ± 1.753	0.5670
PO ₂ , control, kPa	10.68 ± 1.307	10.79 ± 1.400	10.76 ± 1.229	11.09 ± 1.401	11.19 ± 1.335	0.0011
pH, COPD	7.399 ± 0.0455	7.394 ± 0.0457	7.382 ± 0.0449	7.375 ± 0.0438	7.365 ± 0.0448	<0.0001
pH, Control	7.415 ± 0.0307	7.411 ± 0.0304	7.411 ± 0.0529	7.393 ± 0.0303	7.381 ± 0.0351	<0.0001
PCO ₂ , COPD, kPa	6.63 ± 1.901	6.63 ± 1.884	6.75 ± 1.842	6.84 ± 1.883	6.87 ± 1.805	<0.0001
PCO ₂ , control, kPa	5.43 ± 0.532	5.42 ± 0.557	5.50 ± 0.542	5.60 ± 0.598	5.64 ± 0.584	<0.0001
Bicarbonate, COPD, mmol/l	29.66 ± 6.287	29.34 ± 6.238	29.13 ± 5.990	28.98 ± 5.966	28.45 ± 5.799	<0.0001
Bicarbonate, control, mmol/l	25.53 ± 2.397	25.25 ± 2.389	25.17 ± 2.480	24.97 ± 2.426	24.61 ± 2.464	<0.0001
Base excess, COPD, mmol/l	4.14 ± 5.230	3.68 ± 4.990	3.25 ± 4.866	2.90 ± 4.736	2.17 ± 4.689	<0.0001
Base excess, control, mmol/l	0.92 ± 2.287	0.60 ± 2.200	0.27 ± 2.239	0.01 ± 2.183	-0.40 ± 2.320	<0.0001
O ₂ saturation, COPD	92.06 ± 5.726	91.98 ± 5.746	91.73 ± 5.389	91.40 ± 5.412	91.61 ± 5.625	0.4570
O ₂ saturation, control	96.35 ± 1.260	96.64 ± 0.855	96.28 ± 1.194	96.46 ± 1.343	96.57 ± 1.170	0.0938

thors recommend that ABG samples should be analysed within a maximum of 30 min and samples delayed beyond this time should be discarded and remeasured. Air contamination results in an immediate (22% in the control group) increase in PO₂ in ABG samples that is both clinically and statistically significant and that rises further as time passes, culminating in a 54% rise after 180 min.

The pathway for an arterial blood sample can be tortuous. In the ideal clinical scenario, an experienced practitioner would take the arterial sample, expel air in the ABG syringe and analyse the sample immediately. In contrast, the sample may be obtained by an inexperienced doctor or nurse resulting in a 'mixed' or venous sample, inadequately prepared resulting in the presence of air bubbles and delayed in its transportation to a laboratory. Furthermore, pneumatic transport may exacerbate the effects of air contamination [19]. Any or all of these factors might potentially compromise the accuracy of results in ABG samples which, in turn, may alter treatment decisions in individual patients. A mixed arterial/venous sample or a delayed analysis sample (with a lower than accurate pH) may for example result in inappropriate non-invasive ventilatory support for a patient with COPD exacerbation whereas an air-contaminated sample (with a higher than accurate PO₂) may result in inappropriate reassurance and discharge in a hypoxemic patient with a pulmonary embolus.

The clinical scenario of an ABG result for which the clinician suspects the sample is a mixed arterial/venous or purely venous sample is common. While there is consensus that such samples are unreliable for assessment of arterial PO₂ or PCO₂, published studies have suggested

that arterial and venous pH values are comparable [13–17]. Other studies have suggested that calculation of arterial acid-base and blood gas status can be made from measurements in the peripheral venous blood [20, 21]. International guidelines for instituting non-invasive ventilation for patients with decompensated hypercapnic respiratory failure emphasize the acid-base status of a patient and the level of hypercapnia [22]. In patients with COPD exacerbation, using venous pH as a surrogate for arterial pH would be extremely useful and would spare the patient repeated ABG sampling or arterial line insertion and their potential complications. This study demonstrates that there is a substantial difference between venous and arterial pH that is both statistically and clinically significant. Furthermore, while there was a statistically significant correlation between venous and arterial pH, the correlation was relatively weak ($r = 0.5347$) and there were substantial 'outliers' from this correlation (fig. 3), suggesting that this correlation, and the resulting prediction equation (arterial pH = $4.2289 + 0.43113 \cdot$ venous pH), while statistically significant, is insufficient for the accurate prediction of arterial pH from venous pH in clinical practice. Outliers from the correlation line between venous and arterial pH (fig. 4) are much more marked than noted in previous studies, suggesting that there is no venous substitute for ABG analysis [14, 20]. Where there is a suspicion of mixed arterial/venous or venous samples, we suggest that it is mandatory to obtain an arterial sample to determine arterial pH.

Accurate data on the effects of analysis delay on ABG samples, particularly with regard to arterial pH, are extremely limited [5, 9, 23]. The decline in arterial pH

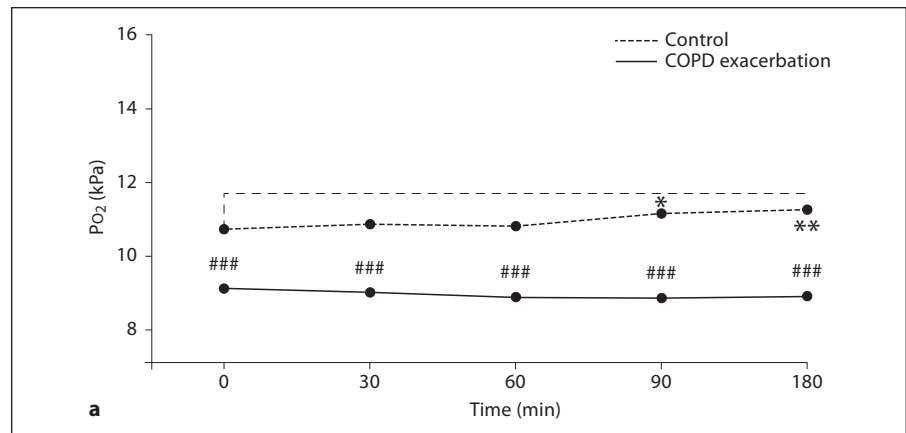


Fig. 1. Analysis delay led to a gradual rise in measured PO₂ that became statistically significant in the control group at 90 min. However, this difference was not clinically significant at any time point (clinical significance is represented by the dashed line). The PO₂ in the COPD exacerbation group was unchanged at all time points. There was a statistically and clinically significant difference in measured PO₂ between control and COPD exacerbation groups at all time points (a). There was a progressive decline in pH associated with analysis delay in both control and COPD exacerbation groups that was more marked in the latter. Statistically significant differences in pH compared to baseline were detected at 60 min in the control group and 30 min in the COPD exacerbation group. Using a change in pH of 0.02 to represent a clinically significant change (dashed line), a significant decline in pH was detected at 72.5 min in patients with COPD exacerbations and at 86.6 min in controls. Although there were clinically significant differences in measured pH between control and COPD exacerbation groups at all time points, these differences were only statistically significant at 60 min ($p = 0.02$) (b). Analysis delay led to a gradual rise in measured PCO₂ that became statistically significant in both groups at 60 min. However, this difference was not clinically significant at any time point (dashed line). There was a statistically and clinically significant difference in measured PCO₂ between control and COPD exacerbation groups at all time points (c). Statistically significant differences compared with baseline values are represented by * $p < 0.05$, ** $p < 0.01$, *** $p < 0.0001$. Statistically significant differences between controls and COPD exacerbations are represented by # $p < 0.05$, ## $p < 0.01$, ### $p < 0.0001$.

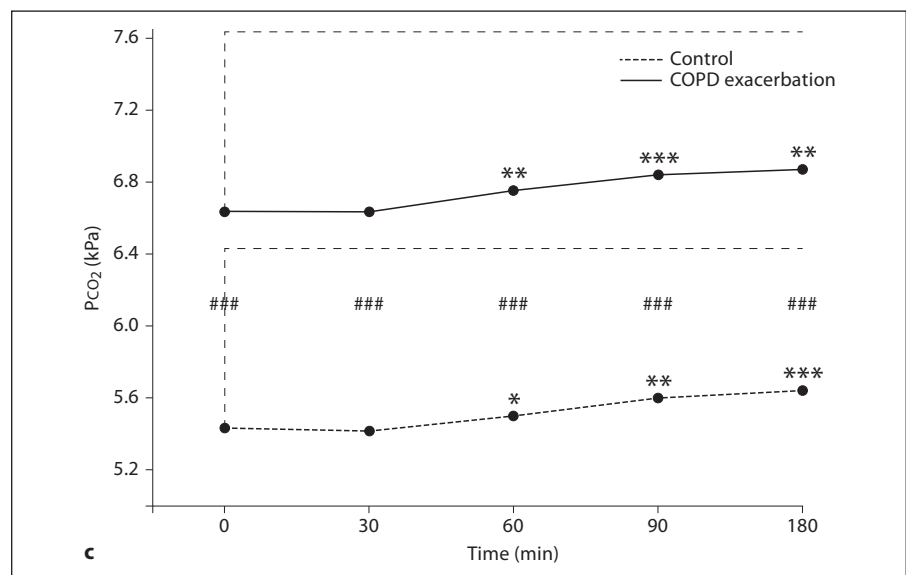
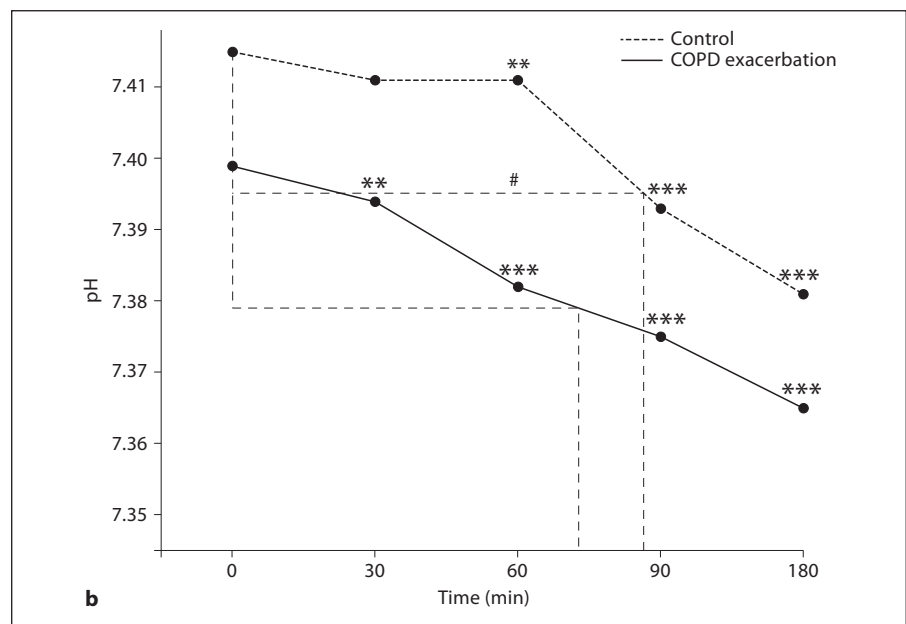
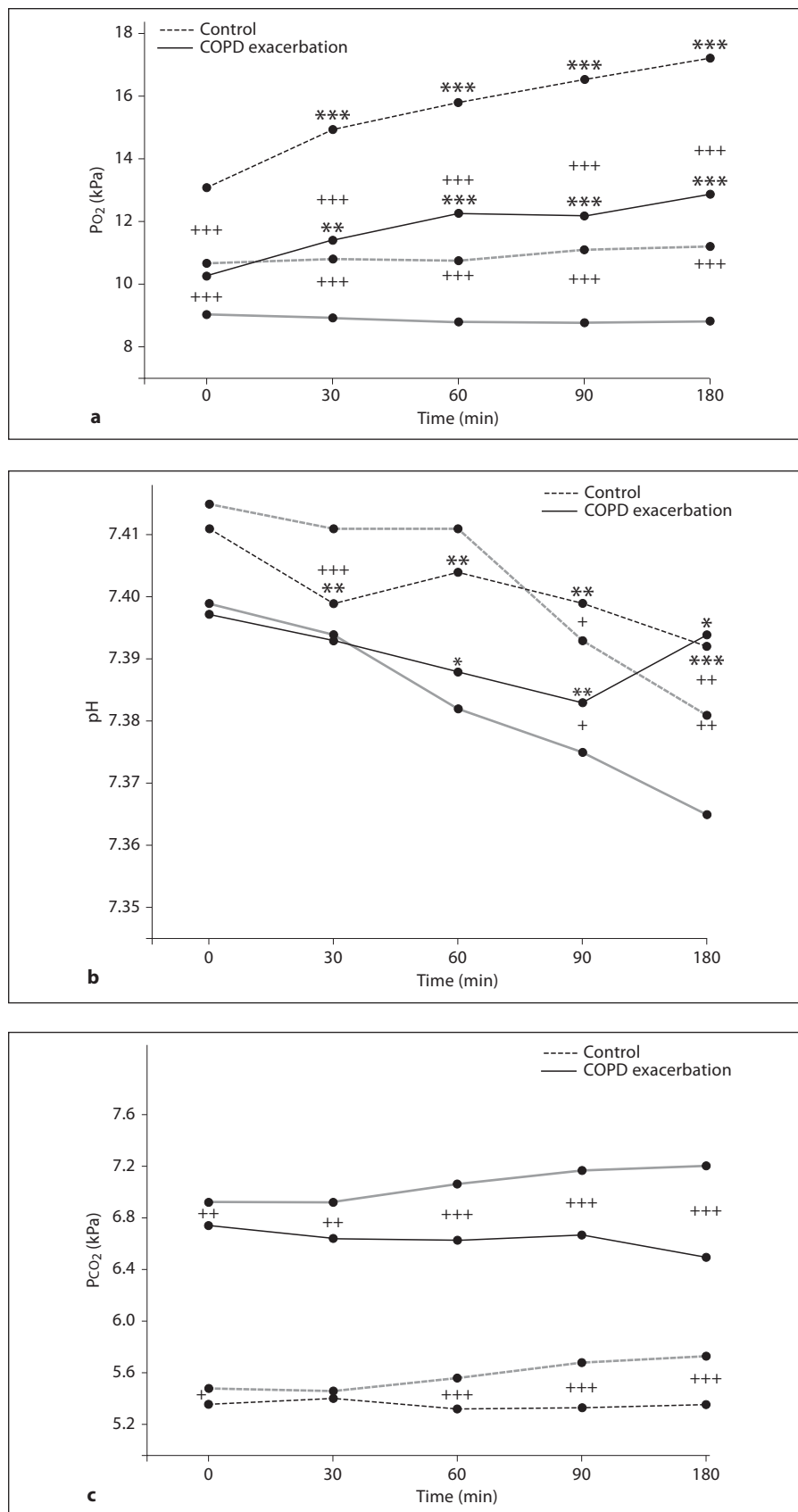


Fig. 2. Air contamination led to an immediate increase in measured PO₂ (even at T = 0 min) in both groups that was both statistically and clinically significant. The increase in PO₂ was progressive and occurred at a faster rate and to a greater degree in controls than in those with COPD exacerbations (a). In the air-contaminated samples, there was a progressive decline in measured pH associated with analysis delay in both control and COPD exacerbation groups. Statistically significant differences in pH compared to baseline were detected at 30 min in the control group and 60 min in the COPD exacerbation group. However, there were no clinically significant changes detected at any time in either group (using a fall in pH of 0.02 to represent a clinically significant change). When comparing with the non-air-contaminated samples, baseline pH was comparable in both groups. There was an initial statistically significant sharper decline in pH in control samples. However, as time went by, this decline was attenuated and was less than non-air-contaminated samples at 90 and 180 min. In the COPD exacerbation group, the rate of decline in pH was similar in the air-contaminated samples and in the non-aircontaminated samples up to 60 min. However, as time went by, this decline was attenuated and was less than non-air-contaminated samples at 90 and 180 min (b). In the air-contaminated samples, analysis delay attenuated the gradual rise in measured PCO₂ that was seen in the non-air-contaminated samples. There were no statistically or clinically significant differences in measured PCO₂ at any time point. When comparing with the non-aircontaminated samples, measured PCO₂ was significantly lower at most time points in the air-contaminated samples (c). Air-contaminated samples are represented by black lines, non-air-contaminated samples by grey lines. Statistically significant differences compared with baseline values in air-contaminated samples are represented by * p < 0.05, ** p < 0.01, *** p < 0.0001. Statistically significant differences between air-contaminated and non-air-contaminated samples at the same time points are represented by + p < 0.05, ++ p < 0.01, +++ p < 0.0001.



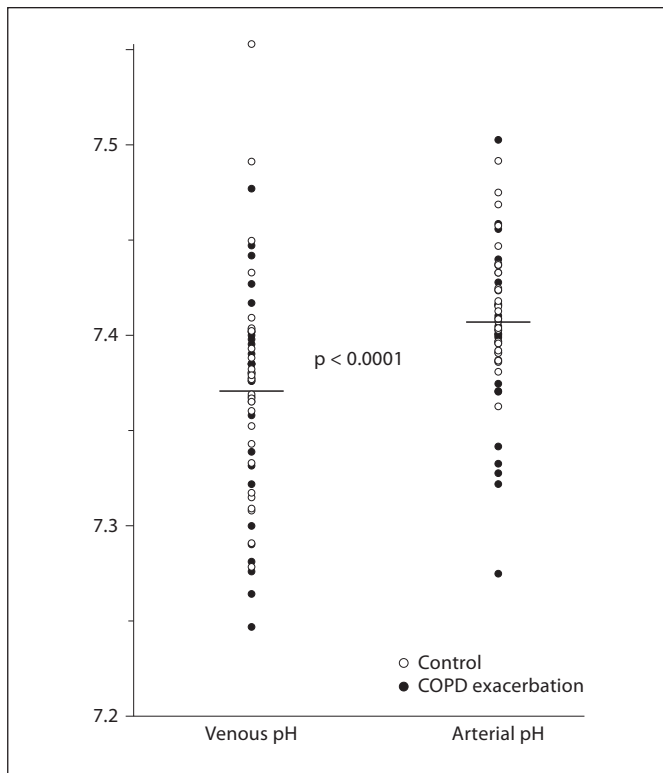


Fig. 3. There was a statistically significant difference between simultaneously measured venous pH (7.371) and arterial pH (7.407) ($p < 0.0001$) that was considered highly clinically significant (difference of >0.02 pH).

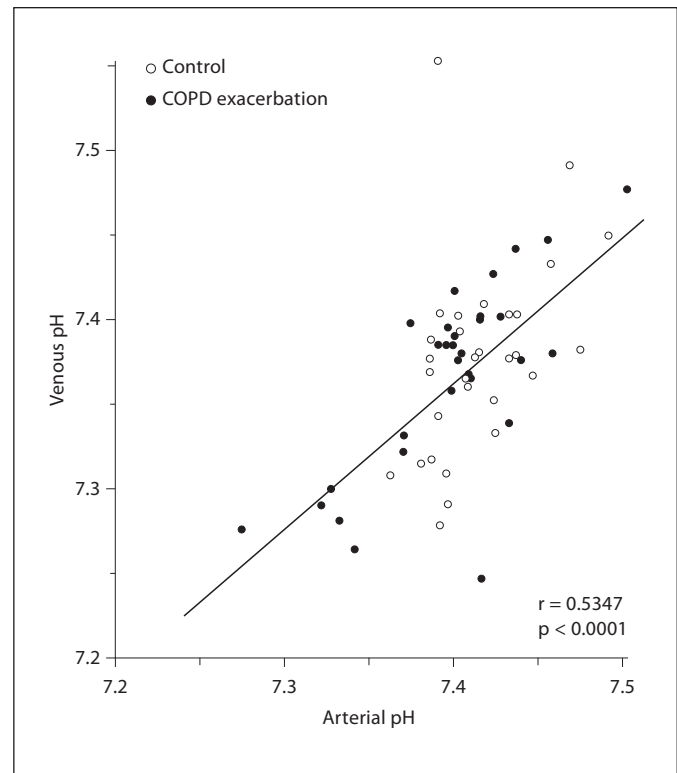


Fig. 4. There was a statistically significant correlation between venous and arterial pH ($r = 0.5347$, $p < 0.0001$). The regression equation to predict arterial pH from venous pH was: arterial pH = $4.2289 + 0.43113 \cdot$ venous pH.

associated with analysis delay that this study has demonstrated is very significant. It is notable that the decline in pH is more rapid in patients with COPD exacerbation than in controls (fig. 1b). Uninhibited anaerobic metabolism and a higher baseline PCO_2 are the likely causes of this decline in arterial pH. While patients with COPD had a substantially higher PCO_2 than controls at all time points of analysis delay and PCO_2 increased progressively over 180 min in both groups, the rate of increase was comparable between COPD exacerbations and controls (fig. 1c). However, this rate of increase brought PCO_2 from 6.63 to 6.87 kPa (49.7 to 51.5 mm Hg) in the COPD group, compared with 5.43 to 5.64 kPa (40.7 to 42.3 mm Hg) in the control group. Therefore, this increase is more likely to lead to a decompensated acid-base balance and a consequent sharper decline in pH. Furthermore, PO_2 was higher at all time points in the control group compared with the COPD group and indeed rose further after 90 min, lessening the tendency to anaerobic metabolism (fig. 1a).

Studies now more than 30 years old have demonstrated that air contamination with gas bubbles whose relative volume is 0.5–1% or more that of the liquid in the collection device may be a potential source of significant error [24]. A recent study from an intensive care unit setting suggested that 40% of ABG samples contain air bubbles or froth [23]. The immediacy with which air contamination leads to a clinically and statistically significant rise in measured PO_2 suggests that samples that are air contaminated are of no value with respect to PO_2 measurement.

Our study had several limitations. Firstly, it is possible that the process of separating arterial samples into air-contaminated and non-air-contaminated samples may, in itself, air contaminate the latter. However, the authors were careful to keep air from the original syringe used to obtain arterial blood as the samples were separated. Secondly, several aspects of this study have been previously investigated. However, many of these studies are several decades old and analysis systems have changed substan-

tially since. Furthermore, this is a comprehensive analysis of several aspects of ABG temporal kinetics, and direct comparisons between COPD exacerbation patients and controls have not been previously made. Thirdly, extrapolation of the clear differences demonstrated in this study between arterial and venous samples to mixed arterial/venous samples obtained through aberrant ABG sampling does not clarify whether such samples are clinically useful. It is impossible in this clinical scenario to determine the proportion of the sample that is arterial, and in such cases, the authors suggest a repeat accurate sample from another artery. Finally, this is the first study to demonstrate significant differences in patients with COPD and healthy controls, emphasizing the need for expediency and care in the very patients in whom ABG analysis is most clinically relevant.

The authors conclude that there is a significant difference between venous and arterial pH. Despite a significant correlation between venous and arterial pH, allowing the prediction of arterial pH from venous pH, the presence of outliers means that there is no alternative to the measurement of ABGs in clinical practice. Temporal delays in ABG analysis result in a decline in measured pH values that is detectable after 30 min and becomes clinically significant after 42 min. Air contamination leads to an immediate clinically significant increase in measured PO₂. Venous pH cannot be used as a surrogate for arterial pH. ABG samples should be analysed within a maximum of 30 min. Air-contaminated ABG samples should be discarded and remeasured.

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