Prediction of Arterial Blood Gas Values from Venous Blood Gas Values in Patients with Acute Exacerbation of Chronic Obstructive Pulmonary Disease

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Arterial blood gas (ABG) analysis has an important role in the clinical assessment of patients with acute exacerbation of chronic obstructive pulmonary disease (AECOPD). However, arterial puncture or insertion of an arterial catheter has many drawbacks. The aim of this study was to evaluate whether venous blood gas (VBG) values of pH, partial pressure of carbon dioxide (PCO₂), and oxygen (PO₂), bicarbonate (HCO₃⁻), and oxygen saturation (SO₂) can reliably predict ABG levels in patients with AECOPD. One hundred and thirty-two patients with a prior diagnosis of COPD presenting with acute exacerbation according to AECOPD criteria were included in this prospective study. AECOPD is defined as a recent increase in cough, wheezing, the volume and purulence of sputum or shortness of breath necessitating a change in regular medication, including corticosteroids or antibiotics. ABG samples were taken immediately after venous sampling, and both were analyzed. Linear regression analysis was performed and equations were established for the estimation of arterial values. The Pearson correlation coefficients for pH, PCO₂, HCO₃⁻, PO₂, and SO₂ were 0.934, 0.908, 0.927, 0.252, and 0.296, respectively. There was a significant correlation between ABG and VBG values of pH, PCO₂, and HCO₃⁻ (p < 0.001). Linear regression equations for the estimation of pH, PCO₂, and HCO₃⁻ were as follows: arterial pH = 1.004 × venous pH; arterial PCO₂ = 0.873 × venous PCO₂; and arterial HCO₃⁻ = 0.951 × venous HCO₃⁻. VBG analysis can reliably predict the ABG values of pH, PCO₂, and HCO₃⁻ in patients with AECOPD. ——— blood gas; arterial; venous; chronic obstructive pulmonary disease

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Arterial blood gas analysis is the gold standard to obtain information about oxygenation, ventilation and acid base status of the body. However, arterial puncture carries the risk of complications such as local hematoma, infection, arterial thrombosis or embolization with consequent ischemic injury to the digits. The procedure itself can be technically difficult and several attempts may be required. It is also painful, particularly when performed on the radial artery at the wrist. Peripheral venous blood gas (VBG) sampling may be a useful alternative to arterial blood gas (ABG) sampling. It is easier to obtain venous blood, the procedure is less painful, and the sample may be drawn during sampling for other laboratory tests. Venous sampling reduces the risk of arterial hematoma, dissection and thrombosis.

Over the years, researchers have searched for alternatives to ABG sampling. There are numerous studies comparing arterial and venous blood gas values that show a good correlation among arterial, capillary and venous samples in both humans and animals (Harrison and Galloon 1965; Long et al. 1971; Harrison et al. 1997). However, there are also contradictory reports (Klingstrom et al. 1976; Brashear et al. 1979). One of these studies was carried out in subjects with respiratory disease (Kelly et al. 2002), but none has specifically investigated the population of patients with acute exacerbation of chronic obstructive pulmonary disease (AECOPD) in the emergency department. The determination of arterial blood gas values is considered essential in the emergency department evaluation of patients with AECOPD.

This prospective study aimed to investigate the relationship between VBG and ABG values, using correlation analysis and to evaluate whether ABG values can be estimated from VBG values in AECOPD patients, accurately enough to be used in clinical practice.

**Materials and Methods**

This study was conducted in the Department of Emergency Medicine, after the approval of the Ethical Committee at Selçuk University, Faculty of Medicine Hospital. The department has an annual census of more than 20,000 visits. The hospital has 1,000-beds and is situated at approximately 1,020 meters above sea level. In our hospital, the reference ranges for ABG parameters are: pH, 7.34-7.46; PCO$_2$, 35-45 mmHg; PO$_2$, 76-100 mmHg; HCO$_3$, 21-26 mmol/l; and SO$_2$, 94-99%.

One hundred and thirty-two patients with a prior diagnosis of COPD presenting with acute exacerbation according to AECOPD criteria (Burge and Wedzicha 2003) were included in this prospective study. AECOPD is defined as a recent increase in cough, wheezing, the volume and purulence of sputum or shortness of breath necessitating a change in regular medication, including corticosteroids or antibiotics. The requirement of blood gas analysis was determined by the emergency department physician responsible for the initial treatment of the patient following informed consent of the patient.

Patients were excluded if verbal consent could not be obtained, hemodynamic status was unstable and there was an obvious alternative cause for their respiratory symptoms (such as pneumothorax, pulmonary emboli, congestive heart failure, restrictive pulmonary disease etc.). The demographic data, vital parameters, and relevant investigations of all the subjects were recorded on a specified proforma. The same investigator took all the blood samples. Venous blood samples were taken at the time of intravenous line placement without a tourniquet and immediately after that, arterial blood samples were taken from the radial artery by direct puncture. The arterial punctures were done by a 24 or 26 gauge needle with 2-ml syringes which contained 0.1 ml sodium heparinate as an anticoagulant. All the samples were sent immediately to the laboratory and analyzed in the Blood Gas/Electrolyte Analyzer (Gem premier 3000, model 5700; Instrumentation laboratory, Lexington, MA, USA) at the temperature of 37°C ± 0.1°C in 5 min.

All the documented data were analyzed using SPSS 13 package program. The means and 95% confidence intervals (CIs) were calculated for each arterial and venous variables and for the differences between them. The strength of the relationship between arterial and venous gas values was assessed with the Pearson product-moment correlation coefficient test. The estimation of arterial values from the venous values were performed by using linear regression equations. $P < 0.05$ was accepted as significant.

**Results**

One hundred and forty four patients were enrolled in the study. Seven patients with primary
metabolic problems were excluded. Three patients were also excluded because both of their samples were of venous origin. In addition, there were two patients with pneumothorax as well as AECOPD. The remaining 132 sample-pairs were studied.

Of the 132 patients, 93 were male and 39 were female. The mean age of the patients was 63.9 ± 10.1 years (range: 27-85 years). The mean arterial pressure of patients was 97.6 ± 11.9 mmHg (range: 73-128 mmHg). The mean pulse rate per minute was 97.8 ± 15.9 (range: 72-145) and the mean respiratory rate per minute was 33.9 ± 4.6 (range:18-40).

The detailed information on blood gas values are given in Table 1. The mean differences (and 95% CIs) between arterial and venous values were as follows: pH, 0.031 (0.026-0.036); PCO₂, 6.6 (5.7-7.5) mmHg; HCO₃⁻, 1.39 (1-1.7) mmol/l; PO₂, 23.8 (21-26.6) mmHg, and SO₂, 26.6% (23.7-29.6).

The venous blood gas identified all 52 patients with arterial hypercarbia, defined as arterial PCO₂ > 46 mmHg. The positive predictive value of venous PCO₂ > 46 mmHg for arterial PCO₂ > 46 mmHg was 62%, (52/84). The negative predictive value of venous PCO₂ ≤ 46 mmHg for arterial PCO₂ ≤ 46 mmHg was 100%, (48/48) (Table 2).

The Pearson correlation coefficients between arterial and venous values were found to be 0.934, 0.908, and 0.927 for pH, PCO₂, and HCO₃⁻, respectively. Arterial and venous pH, PCO₂ and HCO₃ values (Figs. 1, 2 and 3) were highly correlated in patients with AECOPD (p < 0.001). On the other hand, there was a poor correlation between arterial PO₂ and venous PO₂ values (r = 0.252), and also between arterial SO₂ and venous SO₂ values (r = 0.296). Linear regression equations were used to estimate arterial blood gas

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**Table 1.** Mean and mean difference in blood gas values between arterial and venous gas sampling.

<table>
<thead>
<tr>
<th></th>
<th>Arterial (mean ± s.d.)</th>
<th>Venous (mean ± s.d.)</th>
<th>Mean difference (mean ± s.d.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.405 ± 0.08</td>
<td>7.37 ± 0.07</td>
<td>0.031 ± 0.029</td>
</tr>
<tr>
<td>PCO₂</td>
<td>44.7 ± 12.4</td>
<td>51.3 ± 12.3</td>
<td>-6.6 ± 5.3</td>
</tr>
<tr>
<td>PO₂</td>
<td>59.4 ± 15.1</td>
<td>35.6 ± 10.2</td>
<td>23.8 ± 15.9</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>27.2 ± 4.7</td>
<td>28.6 ± 4.7</td>
<td>-1.39 ± 1.81</td>
</tr>
<tr>
<td>SO₂</td>
<td>87.6 ± 9.2</td>
<td>60.9 ± 17.1</td>
<td>26.6 ± 16.8</td>
</tr>
</tbody>
</table>

PCO₂, partial pressure of carbon dioxide (mmHg); PO₂, partial pressure of oxygen (mmHg); HCO₃⁻, bicarbonate (mmol/l); SO₂, oxygen saturation (%); s.d., standard deviation.

**Table 2.** Agreement of hypercarbia (PCO₂ > 46 mmHg [ > 6.1 kPa]), diagnosed by arterial (ABG) and venous (VBG) blood gas measurements.

<table>
<thead>
<tr>
<th></th>
<th>Hypercarbia present (ABG &gt; 46 mmHg)</th>
<th>Hypercarbia absent (ABG ≤ 46 mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VBG &gt; 46 mmHg</td>
<td>52</td>
<td>32</td>
</tr>
<tr>
<td>VBG ≤ 46 mmHg</td>
<td>0</td>
<td>48</td>
</tr>
</tbody>
</table>

The venous blood gas identified all 52 patients with arterial hypercarbia. The positive predictive value of venous PCO₂ > 46 mmHg for arterial PCO₂ > 46 mmHg was 62% (52/84). The negative predictive value of venous PCO₂ ≤ 46 mmHg for arterial PCO₂ ≤ 46 mmHg was 100% (48/48).
values of pH, PCO₂ and HCO₃ from their venous blood gas values and their coefficient of determination, which is the proportion of variability explained by the regression equation. These equations are arterial pH = 1.004 × venous pH, (R² = 1.000); arterial PCO₂ = 0.873 × venous PCO₂ (R² = 0.987); and arterial HCO₃ = 0.951 × venous HCO₃ (R² = 0.996) (Constants were 0 in all equations p > 0.05).

**Discussion**

This study showed a significant correlation between arterial and venous pH, PCO₂ and HCO₃ values in AECOPD patients.

Arterial blood gas analysis is the standard method for the clinical evaluation of patients with AECOPD in the emergency department. However, technical difficulties and complications of arterial puncture led clinicians to look for an alternative method.

Gambino (1961) compared “arterialized” capillary blood with arterial samples from the brachial artery and found no significant difference in pH and CO₂ content in 13 patients, undergoing routine pulmonary function tests. Zahn and Weil (1966) examined the relationship between arterial and central venous pH (cvpH) and concluded that cvpH was a reliable indicator of arterial pH with a correlation coefficient of 0.978. Adrogue et al. (1989) measured the pH and PCO₂ in blood drawn simultaneously from the arterial and central venous circulations and concluded that both arte-
rial and central venous blood samples are needed to assess acid-base status in patients with critical hemodynamic compromise.

Many studies have shown good correlation between arterial and peripheral venous blood gas samples, but authors have differed with respect to whether venous gas analysis can replace arterial gas analysis. In one study that showed high correlation, Gennis et al. (1985) were hesitant to endorse venous sampling alone because of the range of differences between venous and arterial values. In another study, involving patients with diabetic ketoacidosis, Brandenburg and Dire (1998) documented similar differences but came to the opposite conclusion, recommending the use of venous blood gas analysis. McGillivray et al. (1999) agreed that the venous gas value provided an acceptable means for assessing the severity of illness and response to treatment in well-perfused patients. Kelly et al. (2004) examined the relationship between arterial and venous HCO$_3^-$ in the emergency department patients with respiratory or metabolic illness and concluded that venous HCO$_3^-$ estimation may be an acceptable substitute for arterial measurement.

None of these studies, however, has focused on patients with AECOPD. In the light of current literature, our study is the first to investigate the correlation of ABG and VBG values in patients with AECOPD. Although our patients’ characteristics are different from those cited above, our correlation results on pH and HCO$_3^-$ values are in agreement with their results. Although, Chu et al.’s study (2003) is similar to our study, there are some important differences. Chu et al. (2003) treated their patients by mechanical ventilation. In contrast, our patients were ventilating spontaneously during ABG and VBG sampling in the emergency department. Chu et al. (2003) studied in a group of patients with acute respiratory failure. However, our patient group had only AECOPD.

In our study, we found a poor correlation between arterial and venous blood gases for PO$_2$ and SO$_2$ values. Yildizdas et al. (2004) reported similar correlation results for pH, PCO$_2$, and PO$_2$.

Most patients need a venous puncture for various laboratory studies and a VBG sample can be obtained at that time in the emergency department. If a patient’s ventilatory and acid-base status can be determined from a venous sample and the management can be guided with a similar accuracy as arterial sampling, this would be preferable because of the ease of blood sample collection.

For the assessment of respiratory function, PCO$_2$, pH and other markers of oxygenation are all measured and the severity of illness as well as the appropriate therapy are determined. According to our results and the formulations made, for the justification of performing a ventilatory (invasive or noninvasive) treatment in patients with AECOPD, venous blood gas can be a reliable guide. However, as we obtained both ABG and VBG, we used both of them together with the clinical findings in our patients.

In this context, the results of our study helped us to make several formulations. These formulations may be useful for estimating arterial acid-base status in patients with AECOPD from VBG values. However, we do not recommend to estimate arterial PO$_2$ and SO$_2$ from VBG values since there was a poor correlation.

There were some limitations of our study that should be considered when interpreting the results. Our study was performed in a single center and the patient population included some patients with metabolic disease (such as diabetes mellitus, renal disease etc.) besides COPD. The study was undertaken in our hospital settled at an altitude of 1,020 meters. At high altitudes, the partial pressure of oxygen decreases as a function of decreasing total barometric pressure. However, Graham and Houston (1978) found that patients with moderate COPD (but without cor pulmonale or carbon dioxide retention) tolerated 2,000 meters well, complaining only of mild fatigue. On the other hand, Karrer et al. (1990) found no significant difference between the COPD patients and the control group in terms of the decrease in PO$_2$ ve PCO$_2$ values at an altitude of 1,500 meters. They concluded that patients of all degrees of COPD can safely tolerate a difference in altitude from 534 to 1,500 meters.
In the future, similar studies should be performed on different patient populations in multiple centers. In this way, various formulations for estimating the ABG from VBG values can be established in different respiratory and metabolic diseases.

References


