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IMPROVING THE VALIDITY OF PERIPHERAL VENOUS BLOOD GAS ANALYSIS AS AN ESTIMATE OF ARTERIAL BLOOD GAS BY CORRECTING THE VENOUS VALUES WITH SvO₂

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□ Abstract—Background: Peripheral venous blood gas (pVBG) analysis in replacement of arterial blood gas (ABG) is limited by the unpredictable differences between arterial and venous values, especially for PCO₂ and pH (ΔPCO_2 and ΔpH). Objectives: We hypothesized that, using the theoretical relationship linking SvO2 and blood flow, we could diminish the effect of local circulatory conditions on ΔPCO_2 and ΔpH and thereby increase pVBG validity. Methods: This was a prospective cross-sectional study performed in emergency patients requiring a blood gas analysis in which ABG and pVBG were performed simultaneously. The data of 50 randomly selected patients (model group) were used for developing two equations to correct PvCO₂ and pHv according to the peripheral SvO₂ (SpvO₂) level. The formulas derived were $PvCO_{2cor}$ = $PvCO_2 - 0.30 \times$ $(75 - \text{SpvO}_2)$, and $\text{pHv}_{cor} = \text{pHv} + 0.001 \times (75 - \text{SpvO}_2)$. The validity of the corrected values was then tested on the remaining population (validation group). Results: There were 281 patients included in the study, mainly for dyspnea. ΔPCO_2 and ΔpH were strongly correlated with SpvO₂ (r² = 0.62 and $r^2 = 0.53$, respectively, p < 0.001). Using the data of the model group, we developed equations that we applied on the validation group. We found that the corrected values were more valid than the raw values for detecting a $PaCO_2 > 45 \text{ mm Hg}$ (AUC ROC = 0.96 ± 0.01 vs. 0.89 ± 0.02, p < 0.001), a PaCO₂ < 35 mm Hg (AUC = 0.95 ± 0.02 vs. 0.84 ± 0.03 , p < 0.001), a pHa < 7.35 (AUC = 0.97 ± 0.01 vs. 0.95 ± 0.02 , p < 0.05), or a pHa > 7.45 (AUC = 0.91 ± $0.02 \text{ vs. } 0.81 \pm 0.04, p < 0.001$). Conclusions: The variability of ΔPCO_2 and ΔpH is significantly lowered when the venous values are corrected according to the SpvO₂ value, and pVBG is therefore more accurate and valid for detecting an arterial abnormality. © 2013 Elsevier Inc.

□ Keywords—acid-base; arterial blood gas; carbon dioxide; venous blood gas; bicarbonate; arterial puncture

INTRODUCTION

Arterial blood gas analysis plays an important role in interpreting metabolic and respiratory consequences of severe acute illness in the Emergency Department (ED). It is also an important tool for assessing the respiratory status of severe patients with a history of chronic obstructive pulmonary disease (COPD) admitted to the ED, regardless of the cause of their admission. Arterial puncture, however, is more time-consuming, more painful, and may lead to more complications when compared to venous puncture. Several studies have proposed the use of peripheral venous blood gas in replacement of arterial blood gas in the Emergency Medicine setting (1-14). However, most of these studies have shown that this method seems to be somewhat unreliable, especially for evaluating PCO₂, due to unpredictable discrepancies between arterial and venous values (2,7,9,13-18). Therefore, the substitution of venous blood gas for arterial blood gas could be

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limited for patients in whom assessment of $PaCO_2$ is of great importance, such as those presenting to the ED with acute respiratory failure. Indeed, a method for correction of this discrepancy between arterial and venous PCO_2 would be of great interest, particularly in this category of patients.

Several factors may be at the origin of the variability of arteriovenous differences in PCO₂. First, tissue CO₂ production may be different from one patient to another. However, this factor is unlikely to be important when the venous sample is taken from the forearm, because the CO_2 production inducing change in $PvCO_2$ is then limited to the hand and the forearm tissues. The second factor that could explain this variability is linked to the local blood flow. According to the Fick equation, CO₂ may stagnate in the venous blood stream in the case of low blood flow, thereby increasing the gap between the arterial and the venous values (19). The decrease in forearm blood flow may be due to poor circulatory conditions, but may also be the consequence of tourniquet placement on the arm during venous blood sampling (20). In the case of low forearm blood flow, the local venous saturation of oxygen should decrease because local oxygen extraction increases to maintain local oxygen consumption.

We hypothesized that the gradient between arterial and venous PCO_2 (and pH) is mainly dependent on the local blood flow, which may be evaluated by the peripheral SvO_2 value ($SpvO_2$). Therefore, the goals of our study were:

- to check if the gradient between arterial and venous PCO₂ (and pH) is related to the SpvO₂ values
- to propose a correction of the venous PCO₂ and pH values according to the SpvO₂ level to control for the effect of local circulatory conditions on the variability of venous values
- to test the validity of this calculation in a sample of emergency patients

MATERIALS AND METHODS

Study Design

This was a cross-sectional study performed prospectively in patients presenting to the ED who required blood gas analysis. The patients included in the study were randomly divided into two groups: data from the first group of patients was used to construct a model for correction of PCO_2 and pH values (model group), and data from the second group was used to validate that model (validation group).

This study was approved by our local ethics committee and informed consent was obtained from each individual or relatives before inclusion into the study.

Study Setting and Population

This study was performed in a large urban ED with an annual adult census of 70,000.

All patients fulfilling the following criteria were eligible for inclusion in this study:

- Age > 18 years
- Need for arterial blood gas analysis, as decided by the overseeing attending physician, independently of the study.

Study Protocol, Measurements, and Data Collection

Eligible consenting adults were enrolled in the study by the physician treating the patient. Each patient enrolled had a venous blood sample taken from a peripheral venous catheter placed in the arm to start an infusion. Venous blood sample analysis included blood gas measurement (using a 1-mL blood gas syringe) and other examinations, according to the discretion of the attending physician in charge of the patient. Arterial blood gas measurement was made shortly thereafter by puncture in the radial or femoral artery.

Outcome Measures

The main objective of this work was to test the benefit of the correction of PCO_2 and pH values as a function of $SpvO_2$ to detect the presence of an abnormal arterial blood gas. Accordingly, the capacity of the raw venous PCO_2 and pH blood values to detect the presence of an arterial PCO_2 or pH abnormality was compared with that obtained from the values corrected by the $SpvO_2$.

Data Analysis

Statistical and data analyses were made using the StatView[®] (version 5.0; SAS Institute, Cary, NC) and the MedCalc[®] (version 11.1.0.0; MedCalc Software, Mariakerke, Belgium) software. Descriptive data are presented as means plus or minus SDs. A *p* value of < 0.05 was considered significant.

Relationship between arteriovenous gradients and $SpvO_2$. Delta pH (Δ pH) and delta PCO₂ (Δ PCO₂) were calculated as the difference between venous and arterial samples for the pH and PCO₂ for each patient:

 $\Delta pH = pHv - pHa$ $\Delta PCO_2 = PvCO_2 - PaCO_2.$

The strength of the relationship between the ΔPCO_2 (or ΔpH) and the level of SpvO₂ was evaluated by calculating the determination coefficient (r²).

Construction of the model of correction. The multiple linear regression method was applied to the data of the patients randomly assigned to the model group to determine the relationship between the venous values and the SpvO₂ level, for a given arterial value. The obtained coefficient was then used to calculate the correction of venous values for a fixed SpvO₂ value of 75%, which was assumed to be the normal SpvO₂ value.

Validation of the model. The venous values of PCO₂ and pH obtained by correction of venous raw values by SpvO₂ in the validation group were named, respectively, PvCO_{2cor} and pHv_{cor}. The variability of the Δ PCO₂ with and without correction was compared using an F test. The same comparison was done for the pH. The Bland-Altman plots were then constructed to illustrate those comparisons. The ability of the raw and corrected venous values to detect a physiological abnormality (PaCO₂ > 45 mm Hg, PaCO₂ < 35 mm Hg, pH < 7.35, pH > 7.45) was assessed by constructing and comparing the receiver operating characteristic (ROC) curves. The areas under the ROC curves were compared using the methodology described by Delong et al (21).

Because the physiological difference between arterial and central venous PCO₂ is about 6 mm Hg, we assumed that an acceptable difference between arterial and peripheral venous PCO₂ is < 10 mm Hg. In the same way, we assumed an acceptable difference between arterial and peripheral pH to be < 0.05 pH units. Therefore, the impact of the correction was assessed by determining how often the venous blood gas analysis was acceptably close to arterial results with or without correction. Paired proportions were compared using a McNemar test.

RESULTS

Overall Patients

Two hundred eighty-one patients were included in the study from September 2009 to January 2010. The mean age was 76 \pm 16 years, and 48% were male. The diagnoses for which blood gas analysis was indicated are summarized in Table 1. The mean SpvO₂ value was 66.1 \pm 23%, with extremes ranging from 14% to 99%.

There was a strong relationship between the SpvO₂ and the Δ PCO₂ and Δ pH (Figure 1). The determination coefficient showed that the level of SpvO₂ could explain 62% and 53% of the variability of the Δ PCO₂ and Δ pH, respectively.

Model Group

Fifty patients were randomly assigned to construct the model. The multiple linear regression confirmed that

Table 1. Diagnoses by Indications for Blood Gas Analysis

Indication	Number
Shortness of breathing	256
Assessment of stable severe COPD	95
Cardiac failure	66
Pneumonia	31
Worsening of CRF	26
Unknown	10
Pulmonary embolism	9
Febrile pulmonary congestion	9
Asthma	7
Lung cancer	3
Other	25
Sepsis	11
Metabolic disorder	7
Alteration of consciousness	5
Smoke inhalation	2

COPD = chronic obstructive pulmonary disease; CRF = chronic respiratory failure.

the PvCO₂ depends on both PaCO₂ (p < 0.0001) and SpvO₂ (p < 0.0001, slope coefficient = 0.30). The same was observed with pHv (p < 0.0001 for both pHa and SpvO₂, slope coefficient for SpvO₂ = 0.001). By assuming that the normal peripheral SpvO₂ is 75%, we therefore obtained the following correction formula:

 $PvCO_{2cor} = PvCO_2 - 0.30 \times (75 - SpvO_2)$ $pHv_{cor} = pHv + 0.001 \times (75 - SpvO_2).$



Figure 1. Graphs showing the influence of the level of peripheral venous oxygen saturation $(SpvO_2)$ on the arteriovenous difference in PCO₂ (top) and the arteriovenous difference in pH (bottom).



Figure 2. Bland & Altman plots of $PaCO_2 vs. PvCO_2$ (top) and $PaCO_2 vs. PvCO_{2cor}$ (bottom).

Validation Group

Among the 231 patients assigned to this group, 64 had a $PaCO_2 > 45 \text{ mm Hg} (27.7\%)$ and 45 had a $PaCO_2 < 35 \text{ mm Hg} (19.5\%)$. An arterial pH of < 7.35 was present in 29 patients (12.6%), and an arterial pH of more than 7.45 was observed in 43 patients (18.7%).

The mean difference between $PvCO_2$ and $PvCO_{2cor}$ was 1.6 \pm 4.7 mm Hg, with extremes ranging from -5 to 13 mm Hg. The mean difference between pHv and pHv_{cor} was -0.008 ± 0.023 , with extremes ranging from -0.02 to 0.06 pH units.

The Bland-Altman plot shown in Figure 2 illustrates the data distribution of pair differences with less variability among the corrected values compared to the venous raw data (p < 0.001 using an F test for both pH and PCO₂).

The corrected values were more valid than the raw venous values for detecting a physiological abnormality (Figure 3). The areas under the ROC curves were significantly greater with correction, showing more accurate detection of ABG abnormalities when using the corrected values of $PvCO_2$ and pHv:



Figure 3. Receiver operating characteristic (ROC) curves showing the reliability of venous values to detect the presence of an arterial $PCO_2 > 45 \text{ mm Hg}$ (top) or an arterial $PCO_2 < 35 \text{ mm Hg}$ (bottom). The solid lines represent the raw values and the dotted lines the corrected values with $SpvO_2$. The area under the ROC curves is significantly greater for each graph.

PaCO₂ > 45 mm Hg (AUC = 0.96 \pm 0.01 vs. 0.89 \pm 0.02, *p* < 0.001); PaCO₂ < 35 mm Hg (AUC = 0.95 \pm 0.02 vs. 0.84 \pm 0.03, *p* < 0.001); pHa < 7.35 (AUC = 0.97 \pm 0.01 vs. 0.95 \pm 0.02, *p* < 0.05); pHa > 7.45 (AUC = 0.91 \pm 0.02 vs. 0.81 \pm 0.04, *p* < 0.001).

Table 2 reports sensitivity and specificity of raw and SpvO₂-corrected venous values to detect an arterial hyperor hypocarbia. Using uncorrected data, only 28% of arterial blood samples could have been potentially avoided in case of suspected hypercarbia; whereas about 43% could have been avoided using the SpvO₂-corrected values (Table 3). Percentage of arterial sampling that could be avoided for all diagnosis is summarized in Table 3.

Detection		Threshold Value	Sensitivity	Specificity	AUC ROC
PaCO ₂ > 45 mm Hg	Raw	>51	78	81	0.89
	Corrected	>50	91*	89	0.96***
$PaCO_2 < 35 \text{ mm Hg}$	Raw Corrected	≤41 ≤39	64 91**	84 87	0.84 0.95***

Table 2. Sensitivity, Specificity, and Threshold Values of Raw and SpvO₂-corrected Venous Values for Detecting an Arterial PCO₂ > 45 mm Hg or a PCO₂ < 35 mm Hg

AUC = area under the receiver operating characteristic (ROC) curve.

*** *p* < 0.001

Before correction, 71.4% of the patients had an acceptable difference between venous and arterial PCO₂ (< 10 mm Hg), whereas this percentage reached 91.8% after correction with the SpvO₂ model (p < 0.001). The correction for pH was less clinically relevant because the acceptable difference was already 84% with uncorrected data and rose to 90% after correction (p < 0.05).

DISCUSSION

Arterial blood gas analysis plays an important role in the evaluation of patients presenting to the ED for shortness of breath. However, it is a time-consuming, painful, and potentially unsafe procedure. Venous blood gases have been shown to be well related to arterial values, but the arteriovenous difference is highly variable and unpredictable, which makes the substitution not feasible (13,15-18). We have shown that the variability in the arteriovenous difference is, in part, explained by the local blood flow, which may be indirectly evaluated by the SpvO₂ value. When correcting the raw values of the venous blood gas according to the SpvO₂ value, the variability of arteriovenous difference of pH and PCO₂ decreases, making the cut-off screening of venous values more valid in detecting an arterial abnormality. This correction led to a dramatic improvement of the reliability of PvCO₂ as a surrogate of PaCO₂ because the proportion of patients with a clinically acceptable arteriovenous difference of PCO_2 rose from 71% to almost 92%.

Razi and Moosavi evaluated the validity of venous blood gases in patients admitted for exacerbation of COPD (14). They observed that the arteriovenous difference in PCO₂ was greater for the patients with a SpvO₂ of < 70%, but they did not explain this finding. Kelly et al. evaluated the arteriovenous PCO₂ discrepancy in COPD patients and they found a poor agreement between these values, with a bias of 6 mm Hg and 95% confidence interval of -14 to +26 mm Hg (16). We found similar values in uncorrected venous data, whereas this 95% confidence interval decreased to -5 to +13 mm Hg when venous values were corrected with the level of SpvO₂.

We found an important variability of the SpvO₂ values, ranging from 14% to 99%, which accounted for a correction of raw PCO_2 values from -18 to +7 mm Hg. This correction allowed a significant improvement of the validity of the cut-off screening of venous values, which could have theoretically induced a 50% decrease of arterial puncture requirements. The SpvO₂ value is theoretically dependant on two main factors: the local arterial oxygen transport and the oxygen needs of the upstream tissues. Because the oxygen needs of the forearm tissues are not likely to change a lot, the heterogeneity in SpvO₂ that we observed is probably the consequence of a great variability in local oxygen transport. The forearm blood flow may be affected by several factors during peripheral venous sample using a tourniquet. The effect of venous occlusion induced by the tourniquet on the arterial forearm blood flow has been

Table 3. Blood Venous Threshold Values to Exclude an Abnormality of Arterial Blood Gas (Threshold Value for a NPV of 100%) and Percentage of Arterial Blood Samples that Might be Avoided Using Such a Venous Threshold Value

	Blood Venous Threshold Value		Theoretical Percentage of Avoided Arterial Blood Samples		
ABG Anomaly Suspected	Raw	Corrected	Raw	Corrected	p-Value*
$PaCO_2 > 45 \text{ mm Hg}$	>42 mm Hg		28%	43%	<0.001
$PaCO_2 < 35 \text{ mm Hg}$	≤54 mm Hg	≤46 mm Hg	26%	46%	<0.001
pH < 7.35	≤7.36	≤7.36	36%	42%	<0.05
pH > 7.45	>7.33	>7.36	22%	36%	<0.001

NPV = negative predictive value; ABG = arterial blood gas.

* The *p*-value estimates the difference of percentages using a McNemar test.

^{*} p < 0.05

^{**} *p* < 0.01

extensively studied for a long time (20,22). Arterial blood flow is maintained as long as the venous pressure remains sufficiently low and, at one point, the local arterial blood flow starts to decrease. This takes <1 min to occur, which is less than the time necessary for sampling venous blood once the cuff has been placed. Other factors, such as the stress-related release of mediators induced by the clinical condition, could affect the local vasomotricity and be at the origin of a change in local blood flow (23). At last, patients in shock state with low cardiac output have a drop in all peripheral blood flows, including the upper limbs, which decrease the SpvO₂ values (24,25).

A Danish team reported a mathematical method to converse venous values in arterial ones (26). This method calculates arterial values using mathematical models to simulate the transport of venous blood back through the tissues until simulated arterial oxygenation matches that measured by pulse oxymetry. The PaCO₂ is then simulated by assuming that the respiratory quotient is equal to 0.82, which allows calculating the CO₂ load during the tissue crossing. The reported data seem to be very interesting because the bias between arterial and simulated values was close to zero and the method was very accurate (27,28). However, the authors did not provide the mathematical model for making the calculation, which limits the use of such a tool in the ED.

Limitations of the Study

This study has some limitations regarding the interpretation of SpvO₂. First, we did not take into account the time required to take the venous blood sample, nor did we note the duration of tourniquet placement. It would have been interesting to evaluate the relationship between the SpvO₂ value and these parameters. Moreover, a low $SpvO_2$ value could be the consequence of a systemic low blood flow, but we did not take into account the hemodynamic parameters of the patients and thus, we were unable to assess the importance of this parameter. The assessment of cardiac output would have been very helpful for the interpretation of the results, but this monitoring is not easily feasible in a routine emergency practice. Moreover, the presence of hepatic failure is another parameter that could influence the SpvO₂, but we did not take into account this parameter. Finally, a low SpvO₂ could also be the consequence of a low SaO₂, especially in patients admitted for acute respiratory failure. Initially, we took into consideration this parameter, and we first corrected the venous values with the difference between SaO₂ and SpvO₂, instead of SpvO₂ alone. However, the calculations were much more complicated and the results were only slightly improved. Therefore, we deliberately decided not to take into account the SaO₂ value, to simplify the calculations.

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CONCLUSION

In conclusion, the arterial forearm blood flow is probably highly variable from one patient to the other, which is at the origin of a wide discrepancy of the arteriovenous differences in PCO₂ and pH. The venous oxygen saturation value is a simple and convenient tool to assess this discrepancy. The correction of raw venous blood gas values according to the SpvO₂ value allows a great improvement of venous screening test to detect arterial blood gas abnormalities. We suggest that this correction should be performed when physicians extrapolate arterial blood gas from venous measurements, but other studies are required to confirm these results.

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ARTICLE SUMMARY

1. Why is this topic important?

Extrapolating arterial blood gas from a venous sampling may reduce the need for arterial puncture, which is potentially time-saving and limits the risk inherent in this procedure. However, previous studies have shown that this extrapolation is somewhat imprecise due to unpredictable changes in veno-arterial difference values.

2. What does this study attempt to show?

Correcting the venous values with simple equations including the value of peripheral oxygen venous saturation (SpvO₂) allows taking into account the effect of arterial blood flow on veno-arterial differences and increases the validity of this estimation.

3. What are the key findings?

This study confirmed, in 281 emergency patients, that there is a strong relationship between the SpvO₂ and the veno-arterial difference values in PCO₂ and pH. The formulas derived were PvCO_{2cor} = PvCO₂ - 0.30 × (75 -SpvO₂), and pHv_{cor} = pHv + 0.001 × (75 - SpvO₂). Applying this correction significantly improves the capacity of venous blood gas analysis to detect the presence of an arterial abnormality regarding PCO₂ or pH.

4. How is patient care impacted?

The simple correction of blood gas venous values with the $SpvO_2$ allows improving the validity of this procedure and further limits the number of arterial punctures required for blood gas analysis.