Correlation of Central Venous and Arterial Blood Gas Measurements in Mechanically Ventilated Trauma Patients

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Hypothesis: Central venous blood gas (VBG) measurements of pH, PCO2, and base excess can be substituted for the same values obtained from an arterial blood gas (ABG) analysis in mechanically ventilated trauma patients, obviating the need for arterial puncture.

Design and Setting: Prospective comparison of 99 sets of VBGs and ABGs at a level 1 academic trauma center.

Patients: A consecutive sample of 25 trauma patients admitted to the intensive care unit who required mechanical ventilation and had both central venous and arterial catheters.

Main Outcome Measures: Pearson correlations and Bland-Altman limits of agreement (LOAs) for pH, PCO2, and base excess values from each set of VBGs and ABGs.

Results: When VBG and ABG values were compared, pH had R = 0.92, P < .001, and 95% LOAs of −0.09 to 0.03; PCO2, R = 0.88, P < .001, and 95% LOAs of −2.2 to 10.9; and base excess, R = 0.96, P < .001, and 95% LOAs of −2.2 to 1.8. A receiver operating characteristic curve showed that a central venous PCO2 of 50 mm Hg had 100% sensitivity and 84% specificity for determining significant hypercarbia (arterial PCO2 > 50 mm Hg).

Conclusions: Central venous and arterial PCO2, pH, and base excess values correlate well, but their LOAs represent clinically significant ranges that could affect management. Although VBGs cannot be substituted for ABGs in mechanically ventilated trauma patients during the initial phases of resuscitation, clinically reliable conclusions can be reached with VBG analysis.

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We conducted a prospective comparison between venous and arterial pH, PCO₂, and BE values in mechanically ventilated trauma patients. Thirty consecutive trauma patients who were admitted to the trauma/neurosurgical intensive care unit during an 8-week period, required intubation, and had both a central venous and an arterial catheter in place were enrolled. When a patient had a routine ABG sample drawn to assist in managing ventilator treatment, a VBG sample was also drawn. The venous values were obtained only for research purposes and were not available to the treating physicians. Because minimal blood was required to perform this study, a waiver of consent was granted from our review board.

The ABG and VBG analyses were run on an immunoradiometric assay point-of-care blood gas analyzer that is operated by the respiratory therapists working in the intensive care unit (Diametrics Medical Inc, St Paul, Minn). The assay is routinely used for analyzing blood gases in patients receiving mechanical ventilation; its accuracy is validated daily. The ABG values were recorded on the patients’ bedside ventilator flow sheets as well as in a secure computer database. Investigators recorded the venous values in a research database. Treating physicians and respiratory therapists were blinded to these results.

The pH, PCO₂, and BE values from both samples were analyzed with the Pearson test of correlation (R) to determine the strength of relationship between the central venous and arterial values. Linear regression analysis was then used to create a graphic representation of this relationship with the formula of the “best fit” line allowing the arterial pH, PCO₂, and BE values to be calculated from the central venous pH, PCO₂, and BE values, respectively (Figure 1). The coefficient of determination (r²) is the proportion of variation in the dependent variable (arterial) explained by a linear regression model using the independent variable (venous).

The Bland-Altman limits of agreement (LOAs) were also determined by plotting the difference between 2 paired values (arterial and venous) against their mean, thus creating a bias plot (Figure 2). The 95% LOAs represent the mean difference between each pair of venous and arterial values ±1.96 SD, and they estimate by how much a venous value is likely to differ from the criterion standard, the arterial value. This is the accepted method for assessing the agreement between 2 tests and represents a clinically relevant measure of comparison.1,2,4,9,10

A power analysis indicated that we would need 25 patients to determine, with statistical significance (type I error, .05; type II error, .20), a difference between a correlation value of R = 0.8 (not correlated well enough) vs R = 0.95 (strongly correlated). We intended to draw 4 samples from each patient, with each sample consisting of an ABG measurement and a VBG measurement. We enrolled 30 patients to account for incomplete data collection, early extubation, and death. Data are presented as mean ± SD unless otherwise indicated.

A receiver operating characteristic curve was created to determine the ideal central venous PCO₂ value that could serve as a screening cutoff for significant arterial hypercarbia (PCO₂ > 50 mm Hg). The pH, PCO₂, and BE values from both samples were analyzed with the Pearson test of correlation (R) to determine the strength of relationship between the central venous and arterial values. Linear regression analysis was then used to create a graphic representation of this relationship with the formula of the “best fit” line allowing the arterial pH, PCO₂, and BE values to be calculated from the central venous pH, PCO₂, and BE values, respectively (Figure 1). The coefficient of determination (r²) is the proportion of variation in the dependent variable (arterial) explained by a linear regression model using the independent variable (venous).

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**RESULTS**

Thirty consecutive injured patients who met inclusion criteria were enrolled in this study. Five patients were excluded because of early extubation or death. The remaining 25 patients had 4 sets of ABG and VBG samples collected, with the exception of 1 patient in whom the central line was removed before the fourth blood gas measurement. In total, 99 sets of blood gas values were analyzed.

Our subjects had an average age of 43±18 years, had an Injury Severity Score of 26±7, and required 9±5 days of mechanical ventilation; 18 (72%) were male, all suffered blunt trauma, and 4 (16%) died. The indications for intubation were neurologic in 15 (60%) (traumatic brain injury or altered mental status), respiratory in 6 (24%) (hypoxia or hypoventilation), and hypotension in 4 (16%).

The Table presents a summary of the PCO2, pH, and BE values obtained from 99 sets of VBG and ABG measurements. For PCO2, the mean VBG value was 45.0±6.9 mm Hg compared with an ABG value of 41.0±7.4 mm Hg. The central venous and arterial PCO2 values correlated significantly, with an R value of 0.88 (P<.001; r²=0.78) (Figure 1). The mean difference between each set of central venous and arterial values was 4.36±3.34 mm Hg (Figure 2). The 95% LOAs between central venous and arterial PCO2 were −2.2 to 10.9 mm Hg (VBG−ABG). Consequently, if a central venous PCO2 value were to be obtained, there would be a 95% chance that the arterial value would be between 2.2 mm Hg higher and 10.9 mm Hg lower than the measured central venous value.

For pH, the mean VBG value was 7.35±0.07 compared with an ABG value of 7.39±0.07. The VBG and ABG values correlated significantly (R=0.92; r²=0.84, P<.001) (Figure 1). The bias plot showed a mean difference between the VBG and ABG values of 0.032±0.026 (Figure 2). The LOAs were −0.09 to 0.03 (VBG−ABG). One could estimate that the arterial pH would be between 0.09 higher and 0.03 lower than the measured VBG value.

With respect to BE, the mean VBG value was −0.34±3.72 compared with an ABG value of −0.01±3.88. The 2 values correlated significantly (R=0.96; r²=0.93; P<.001) (Figure 1). The mean difference on bias plot was −0.18±1.03, with LOAs of −2.2 to 1.8 (VBG−ABG) (Figure 2). Hence, the estimated ABG value would have a 95% chance of being between 2.2 mm Hg higher and 1.8 lower than the measured VBG value.

To determine whether ABG and VBG values would differ less as time from the injury increased, the mean differences in the arterial and central venous PCO2, pH, and BE in the first through fourth sets were compared with each other, and they were not found to be significantly different (analysis of variance; all P>.38). In addition, 7 patients were found to have a significant lactic acidosis (ΔΔ ratio >0.75), and their mean PCO2, pH, and BE differences were not significantly different from those of the other 19 patients (unpaired t test, lactic acidosis present vs absent; PCO2: 6.4 vs 4.2 mm Hg, P=.1; pH: −0.03 vs −0.04, P=.2; BE: −0.19 vs 0, P=.6).

Six patients had significant hypercarbia (ABG PCO2>50 mm Hg), and all of them had a central venous PCO2 greater than 50 mm Hg. A receiver operating characteristic curve showed that a central venous PCO2 of 50 mm Hg had a sensitivity of 100% and a specificity of 84% for detecting hypercarbia (area under the curve, 0.95) (Figure 3).

**COMMENT**

Arterial blood gas analysis is the current criterion standard for determining acid-base status and making ventilator adjustments with respect to PCO2 and pH. The arterial BE can be interpreted to guide resuscitation and provide...
prognostic information. Arterial sampling carries a small risk of complications associated with arterial puncture and catheter placement as well as the possibility of needlestick injuries to health care providers. Intubated trauma patients often have central venous catheters, and it was our goal to determine whether central venous PCO₂, pH, and BE values could be substituted for ABG analysis, obviating the need for arterial sampling.

The ABG and VBG values correlated satisfactorily (R = 0.88) for PCO₂, but their 95% LOAs were too wide to allow substitution (−2.2 to 10.9). For example, if a central venous PCO₂ value of 40 mm Hg were to be obtained, the arterial value might be as low as 29 mm Hg or as high as 42 mm Hg. However, significant hypercarbia (arterial PCO₂ > 50 mm Hg) can be ruled out with a central venous PCO₂ of 50 mm Hg or less, as all 6 patients with an arterial PCO₂ greater than 50 mm Hg also had a central venous value greater than 50 mm Hg. Consequently, the central venous PCO₂ could be used as a monitor of alveolar ventilation in injured patients who are being weaned from mechanical ventilation.

With respect to pH, the ABG and VBG values also correlated satisfactorily (R = 0.92) and had LOAs that were too wide to allow substitution (−0.09 to 0.03). For example, if a VBG value of 7.20 were obtained, the estimated arterial value could be as low as 7.17 or as high as 7.29. Nonetheless, in general, one can be 95% confident that the arterial value will not be more than 0.03 lower than the central venous value, which would allow patients with a significant acidosis to be reliably identified.

Regarding BE, the ABG and VBG values correlated well (R = 0.96), but their 95% LOAs were, again, too wide to allow substitution (−2.2 to 1.8). For instance, if a VBG value of −2.0 were obtained, the ABG value could be as low as −3.8 or as high as 0.2.

Before the development of modern blood gas analyzers, the venous PCO₂ was calculated by using the measured venous pH and total CO₂ content and the Henderson-Hasselbalch equation. Using this method, Brooks and Wynn discovered that “arterialized” venous values for PCO₂ and pH were not significantly different from arterial values. However, a statistical analysis was not part of this study, and the level of agreement between the arterialized and arterial values was not determined. Blood was considered arterialized if the skin was warm (>35°C) and the patient was lying in bed. These conditions minimized the arteriovenous differences in measured plasma pH and total CO₂.

Venous blood can also be arterialized by the capillary technique that involves a finger prick of a well-perfused digit. Gambino demonstrated that measured pH, PCO₂, and total CO₂ of samples of capillary and brachial artery blood were not significantly different. Heparinized venous blood has also been shown to correlate satisfactorily with heparinized arterial blood pH, PCO₂, bicarbonate, and BE values, with correlation coefficients ranging from 0.85 to 0.93. Venous and capillary blood gas values for pH and PCO₂ have also been shown to correlate satisfactorily (r = 0.92 and 0.80, respectively) in the pediatric population. However, statistical correlation is not a surrogate for clinical substitution. Kelly et al compared peripheral venous and arterial PCO₂ in emergency department patients with acute respiratory conditions and found that, while the 2 values correlated with each other, the venous values were, on average, 5.8 mm Hg higher than the arterial values. The 95% LOAs between the 2 values were −8.8 to 20.5 mm Hg (venous–arterial), and it was concluded that these differences were too large to allow peripheral venous PCO₂ to serve as a substitute for arterial PCO₂. The authors did find that a screening venous PCO₂ of 45 mm Hg had 100% sensitivity and 57.1% specificity for the detection of hypercarbia (arterial PCO₂ > 50 mm Hg).

Similarly, our data demonstrate that central venous and arterial PCO₂, pH, and BE values correlate satisfactorily to well, but their 95% LOAs represent clinically significant ranges that could affect management decisions in critically ill patients during the initial phases of resuscitation from injury. However, we conclude that these data support the utility and reliability of VBGs in mechanically ventilated patients in the recovery phase from injury, where the combination of a normal central venous pH and a PCO₂ of 50 mm Hg or less may be used to rule out a significant respiratory acidosis.

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REFERENCES