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CORRELATION OF VENOUS BLOOD GAS AND PULSE OXIMETRY WITH ARTERIAL BLOOD GAS IN CRITICALLY ILL PATIENTS

Anesthesiology

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ABSTRACT

Introduction: Arterial blood gas (ABG) analysis is the gold standard method and frequently performed intervention to evaluate acid-base status along with adequacy of ventilation and oxygenation among patients with predominantly critical / acute diseases.

Aims And Objectives: The aim of this study is to evaluate the correlation of VBG analysis and pulse oximetry (SpO2) with ABG analysis in critically ill patients.

Materials And Methods: Intensive Care Unit (ICU), Command Hospital (Eastern Command), Kolkata, Adult patients requiring arterial blood gas analysis, JAN 2018 –JUNE 2019, 100 critically ill patients and Age – 18yrs and older, Sex – Either sex.

Conclusion: In this study population of critically ill patients, pH and pCO2 on VBG analysis correlated with pH and pCO2 on ABG analysis. The SpO2 correlated well with pO2 on ABG analysis.

KEYWORDS

Critically ill, Arterial blood gas, Venous blood gas and Pulse oximetry

INTRODUCTION

Arterial blood gas (ABG) analysis is the gold standard method and frequently performed intervention to evaluate acid-base status along with adequacy of ventilation and oxygenation among patients with predominantly critical / acute diseases. Unfortunately, prolonged arterial catheterization or repeated direct blood sampling from the artery carries risk for both the patient and the health care providers.[1] Patients requiring frequent blood gas analysis often have central venous catheters for intravenous drug administration or central venous pressure monitoring, enabling them for repeated central venous blood gas (VBG) sampling and analysis. Venous blood gas analysis needs fewer punctures, and is a comparatively safer method for both the patient and health care providers, and can be an option to ABG assessment (Walkey et al., 2010)[2]. The purpose of this research was to determine the correlation between arterial and venous blood gas measurements along with SpO2 in the critically ill patients to monitor acid-base balance & oxygenation.

Patient safety was widely conceived as the avoidance of unnecessary patient harm or probable harm (European Union Council, 2009 & WHO 2010). In relation to this goal of harm prevention, patient safety can be practically defined as the pursuit of the reduction and mitigation of unsafe acts within the healthcare system, as well as the use of best practices shown to lead to optimal patient outcomes. By their nature, critically ill patients are suffering from many life-threatening problems that require continuous monitoring and management. The objective for management of critically ill patients is the maintenance and optimization of cellular (and organ) health. This goal can be achieved by maintaining oxygenation, perfusion, fluid, electrolytes, acid base balance and others (Adrogué et al., 1998)[3]. Acid base and oxygenation abnormalities are also critical, particularly when these disorders develop rapidly. Additionally, serious defects can be the immediate cause of organ dysfunction. Monitoring of acid base and oxygenation is a vital step in managing critically ill patients. Arterial blood gas (ABG) is a precious tool for assessing a variety of diseases and constitutes the criterion standard for determining the acid-base status of a ventilated and non-ventilated patient.[4]Critical care nurse caring for critically ill patients should be skilful in the continuous bedside assessment and management and she needs to understand the issues related to the blood gas interpretation and skilfully perform blood gas sampling.

Arterial sampling can be done either by direct arterial puncture or by arterial catheter placement. Although the arterial blood gas is considered a standard, it is not without complications including local hematoma, haemorrhage, infection, aneurysm formation, arterial thrombosis or embolization with eventual digit ischemic injury, needle stick injury to healthcare providers.

Moreover, the technique is more challenging technically and more painful. Central venous sample is simpler to acquire, the technique is less difficult, and at a moment when the patient is being sampled for further investigations, there is a benefit of blood being drawn. In addition, central venous sampling is a safer method than arterial sampling and can decrease complications resulting from extended arterial cannula placed in the artery (Lorente et al., 2006)[5], Scheeretal., 2002)[6].

Although the venous sample for the patient is comparatively safer and painless, a question has been raised about reliability and precision. This study was conducted to find out, could the pH, pCO2, pO2 measurements of venous blood gas (VBG) collected from the central or peripheral venous catheter along with SpO2 monitoring be as accurate as the same measurements collected from the artery for acid –base status & oxygenation in critically ill patients, avoiding the need for arterial puncture.[7]

The aim of this study is to evaluate the correlation of VBG analysis and pulse oximetry (SpO2) with ABG analysis in critically ill patients.

MATERIALS AND METHODS

Study Area: Intensive Care Unit (ICU), Command Hospital (Eastern Command), Kolkata

Study Population: Adult patients requiring arterial blood gas analysis

Study Period: JAN 2018 –JUNE 2019

Target Sample Size: 100 critically ill patients

Sample Design: Age – 18yrs and older, Sex – Either sex

Inclusion Criteria: Critically ill adult patients requiring arterial blood gas analysis admitted in the ICU

Exclusion Criteria:

- Patients with contraindication to arterial or venous blood draw
- Patients not giving consent for the study

Study Design: Prospective, Cohort

Study Technique:

After obtaining clearance from ethical committee of the institute, the study was conducted in the Intensive Care Unit (ICU), Command Hospital (EC), Kolkata. After taking written consent, patients were enrolled in the study applying inclusion and exclusion criteria.

Patients are eligible for the study, if they are aged 18 years or older and they require an ABG by the treating physician. Patients were excluded if they have a contraindication to arterial or venous blood draw. After informed consent, ABG and VBG samples were obtained as temporally close to each other as possible. The ABG was obtained from either an existing arterial line or from the radial, brachial, or femoral artery. The VBG was obtained from either an existing venous catheter or from a new peripheral venous access.

Additional data was gathered including presence of ongoing shock state, body temperature, heart rate, respiratory rate, patient on mechanical ventilation or not, patient on inotropic support or not. For the purposes of this study, we define shock as the use of vasopressors and/or inotropes or systolic blood pressure <90 mm Hg or mean arterial blood pressure <65 mm Hg. The ABG and VBG analyses were performed at the bedside.

Statistical Methods:
The strength of association between arterial pH, venous pH, arterial pCO2, venous pCO2, and arterial pO2 and SpO2 was assessed using paired t test, Pearson x2, and Pearson correlation coefficient.

Table: Distribution of mean in Age in Years, Hb, HR, RR, Temperature, MAP, SPO2, Arterial PO, Arterial PCO2, Arterial pH, Venous PO2, Venous PCO2 and Venous pH: parameters

Descriptive Statistics			
	Mean	**Std. Deviation**	**N**
Age in Years	54.93	14.883	100
Hb	9.813	1.9067	100
HR	100.13	15.207	100
RR	19.31	4.331	100
Temperature	99.191	.8189	100
MAP	94.12	13.508	100
SPO2	97.81	2.600	100
Arterial PO2	132.44	58.124	100
Arterial PCO2	37.852	16.5760	100
Arterial pH	7.48175	.095130	100
Venous PO2	41.103	11.2879	100
Venous PCO2	42.057	16.7024	100
Venous pH	7.44736	.094507	100

DISCUSSION

In this study 34(34.0%) patients were female and 66(66.0%) patients were male. It was found that 62(62.0%) patients were on mechanical ventilation and 45(45.0%) patients were on vasopressor support. In 46(46.0%) of patients central VBG and 54(54.0%) patients peripheral VBG samples were collected.

Blood gas analysis remains an important tool for evaluation of acid–base and ventilation status of critically ill patients. The results of this study add to a growing body of evidence that supports the use of VBG instead of ABG for determination of pH and pCO2 in these patients. Previous studies have examined VBG values specifically in COPD and have showed good correlation with ABG values. [8,9] Many studies have evaluated arterial and venous blood gas values in the setting of DKA.[10– 12,13] Several other studies have also examined the correlation between arterial and venous pH and pO2 in a variety of disease states but in isolated locations such as the ED or medical or surgical ICUs. This has led to possible limitations where the focus is on one disease state or a specific location, reflecting a more acute process (the ED patient) or sub acute or chronic process (the ICU patient). This study is unique in that, it includes a diverse group of critically ill patients in both medical and surgical ICUs. Often, the underlying pathophysiologic state is not known for certain at the time of blood gas analysis, and multiple disease processes may occur simultaneously. By simplifying the enrollment criteria to a critically ill patient in whom the treating physician ordered an ABG, this study captured a wide variety of patients and could not identify as ub group in which the ABG failed to correlate with the VBG and pulse oximetry. It is believed that this study design most realistically replicates real-world clinical practice.

In this study positive correlation was found between SPO2 vs. arterial pO2 and it was statistically significant (p<0.0001). Positive correlation was found between arterial pO2 vs. venous pO2 and it was statistically significant (p=.005). Positive correlation was found between arterial pCO2 with venous pCO2 and it was statistically significant (p<0.0001). Positive correlation was found between arterial pH with venous pH and it was statistically significant (p<0.0001).

Many other studies that have evaluated the correlation between pulse oximetry and arterial blood gas values, have compared SpO2 and SaO2 (saturation of oxygen in hemoglobin as measured from an arterial puncture sample).48-50 we chose to compare SpO2 and PaO2, evaluating the common clinical dictum that when SpO2 is ≥90, PaO2 is ≥60. We also chose to use this comparison because we believe the PaO2 is more clinically relevant.

In this study, SpO2 correlated with PaO2 as predicted by the standard oxygen–hemoglobin dissociation curve. We would still caution that this assumption is only accurate when the typical oxygen– hemoglobin dissociation is present. Clinical factors that may alter this include arterial pH, PaCO2, and temperature. A left shift in the oxygen–hemoglobin dissociation curve causes hemoglobin to have a higher affinity for oxygen and more reluctance to unload oxygen in the capillary beds. This could lead to situations where the actual PaO2 is lower than predicted from the pulse oximetry value.

In patients not on Mechanical Ventilation, it was found that positive correlation was found between arterial pO2 vs. SpO2 and it was statistically significant (p=.003). Positive correlation was found between arterial pO2 vs. venous pO2 and it was statistically significant (p=.003). Positive correlation was found between arterial pCO2 vs. venous pCO2 and it was statistically significant (p<0.0001). Positive correlation was found between arterial pH vs. venous pH and it was statistically significant (p<0.0001).

In patients on Mechanical Ventilation, it was found that Positive correlation was found between Arterial pO2 vs. SpO2 and it was statistically significant (p=0.0001). Positive correlation was found between arterial pO2 vs. venous pO2 and it was not statistically significant (p=.078). Positive correlation was found between arterial pCO2 vs. venous pCO2 and it was statistically significant (p<0.0001). Positive correlation was found between arterial pH vs. venous pH and it was statistically significant (p<0.0001).

Of course pH, pCO2, and temperature can also shift the oxygen dissociation curve to the right, which would lead to a situation where the PaO2 is higher than that predicted by the pulse oximetry value. Another factor that has been described that can cause falsely lower SpO2 values is high venous pressure states. In this scenario, venous pulsations can theoretically be interpreted by the pulse oximeter as arterial flow, which would lead to a markedly low pulse oximeter reading.51 It does not appear that we had any patients in our cohort where this occurred as there were no instances where the SpO2 was markedly low with normal or near normal PaO2 levels.

Anemia has been evaluated and shown to affect pulse oximetry readings. However, the significance of this is not fully known.[14,15] It is likely that this is not relevant except at extremes.[15,16]

Dyshemoglobinemia can cause discrepancies between pulse oximetry and PaO2. With significant carbon monoxide poisoning or methemoglobinemia, pulse oximetry readings would be higher than expected or in a normal range; despite a true low PaO2 value.[15] We did not have any patients in our cohort with a known working diagnosis of carbon monoxide poisoning or methemoglobinemia.

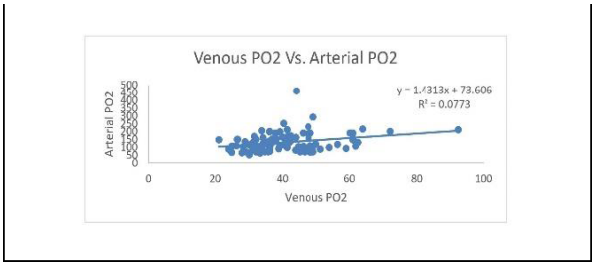
It is important to note that the method of using venous blood gas values and pulse oximetry as a surrogate for arterial blood gas values will not identify potentially clinically relevant hyperoxia. Hemoglobin is fully saturated, in other words, SpO2 equals 100% when the PaO2 is approximately 120 mm Hg.[15] This means, any higher PaO2 level will go unnoticed by pulse oximetry. More recently, hyperoxia has been associated with increased mortality, particularly in post cardiac arrest patients, although this has been challenged in other studies.[17] If using the combination of VBG and pulse oximetry without a known PaO2, the possibility of hyperoxia would still need to be considered.

In Hb<8, Positive correlation was found between arterial pO2 vs. SpO2 and it was statistically significant (p=.046). Positive correlation was found between arterial pO2 vs. venous pO2 and it was statistically significant (p=.001). Positive correlation was found between arterial pCO2 vs. venous pCO2 and it was statistically significant (p<0.0001). Positive correlation was found between arterial pH vs. venous pH and

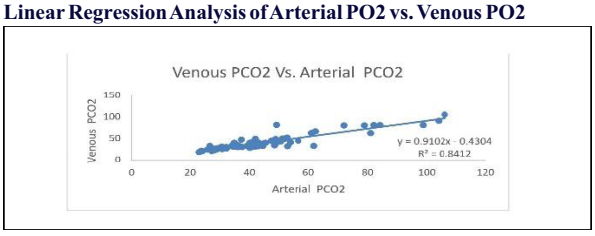
it was statistically significant (p<0.0001).

In Hb>8, Positive correlation was found between arterial pO2 vs. SpO2 and it was statistically significant (p<0.0001). Positive correlation was found between arterial pO2 vs. venous pO2 and it was statistically significant (p=.013). Positive correlation was found between arterial pCO2 vs. venous pCO2 and it was statistically significant (p<0.0001). Positive correlation was found between arterial pH vs. venous pH and it was statistically significant (p<0.0001).

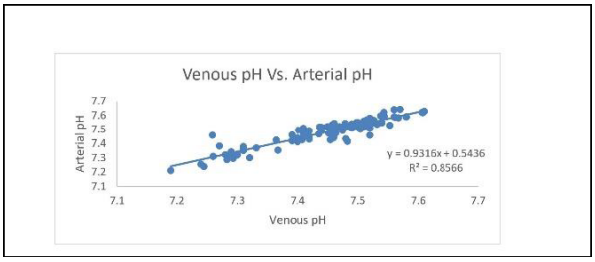
Linear regression analysis is suggestive of a linear relationship between arterial PO2 and Venous PO2 with Coefficient of determination that is R2=0.07and arterial PO2 is dependent on venous PO2 linearly as Y= 1.4313 X + 73.06. Similarly, linear regression analysis is suggestive of a linear relationship between Arterial PCO2and Venous PCO2 with Coefficient of determination that is R2 =0.8412 and arterial PCO2 is dependent on venous PCO2 linearly as Y=0.9102 X – 0.4304 and also a linear relationship between arterial pH and Venous pH with Coefficient of determination that is R2 =0.85 and arterial pH is dependent on venous pH linearly as Y= 0.9316 X +0.5436.



Linear Regression Analysis of Arterial PO2 vs. Venous PO2



Linear Regression Analysis of Arterial PCO2 vs. Venous PCO2



Linear Regression Analysis of Arterial pH vs. Venous pH

We had hypothesized that the poor perfusion associated with shock states could lead to a weaker correlation between VBGs and ABGs. However, the correlation was just as strong in the rest of the patients as in this cohort. Other studies comparing arterial and venous blood gas analysis in shock states have been mixed.[17,18] There is a large body of research evaluating arterial to venous CO2 difference in shock as a marker for inadequate tissue perfusion. An arbitrary number of greater than 6 mm Hg difference between arterial and venous CO2 measurements has often been designated as elevated and potentially a marker of ongoing tissue ischemia.[18] In cases where there is significant discrepancy, it is likely that the more clinically relevant CO2 and pH comes from the venous system rather than the arterial. This becomes even more apparent when evaluating arterial and venous blood gas analysis during cardiopulmonary resuscitation where much wider discrepancies are seen, sometimes with normal pH or only mild arterial acidemia.[18] In terms of pulmonary gas exchange and acid–base status, we do not feel these identified differences in CO2 and pH are clinically relevant.

Another important finding in the subgroups addresses the accuracy of central versus peripheral VBGs compared to ABGs. The best way to answer this question would be to obtain a peripheral VBG, a central VBG, and an ABG in the same patient at the same time as was performed in a prior study. Their findings showed very good correlation between all 3 groups. In our study, only 1 VBG was analyzed per patient, either peripheral or central. However, when the peripheral VBGs were compared to ABGs and the central VBGs were compared to ABGs, there was no statistical difference in the correlation. This result seems to be consistent with others' findings.

CONCLUSION

In this population of critically ill patients, pH and pCO2 on VBG analysis correlated with pH and pCO2 on ABG analysis. The SpO2 correlated well with pO2 on ABG analysis. When compared to ABG, the combination of VBG analysis plus SpO2 provided accurate information on which to make bedside clinical decisions regarding acid–base, ventilation, and oxygenation status for critically ill patients in the ICU.

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