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Venoarterial PCO₂ Difference: A Marker of Postoperative Cardiac Output in Children with Congenital Heart Disease

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ABSTRACT

Objective: To determine the relationship between venoarterial carbon dioxide gradient (ΔpCO_2) and central venous oxygen saturation (ScvO₂) in children after cardiac surgery.

Study Design: A cohort study.

Place and Duration of Study: The Paediatric cardiac intensive care unit of the Aga Khan University Hospital, Karachi, from June 2006 to May 2007.

Methodology: All children admitted in the paediatric cardiac intensive care after complete repair of congenital heart defect using cardiopulmonary bypass were included in the study. Simultaneous arterial and central venous blood gas samples were obtained from a catheter placed in the artery (either radial or femoral) and superior vena cava respectively. Linear regression analysis was performed between ScvO₂ and ΔpCO_2 .

Results: Fifty seven children aged from 5 days to 14 years were included and 272-paired simultaneous arterial and central venous samples were analyzed. Mean venous pCO₂ was 47.82±9.03 mmHg and mean arterial pCO₂ was 40.50±9.06 mmHg. One hundred seventy four samples had ScvO₂ > 70% with mean ΔpCO_2 of 5.44±2.55 mmHg and 98 samples had ScvO₂ < 70% with mean ΔpCO_2 of 9.07±3.90 mmHg. With ScvO₂ < 70%, 77 samples had ΔpCO_2 of > 6 mmHg while only 21 samples had ΔpCO_2 of < 6 mmHg ($p < 0.001$). On the contrary with ScvO₂ > 70%, 71 samples had ΔpCO_2 of > 6 mmHg and 103 samples had ΔpCO_2 of < 6 mmHg. Coefficient of correlation (R_2) between 0.340 was ScvO₂ and ΔpCO_2 .

Conclusion: Elevated ΔpCO_2 is practical and can be utilized as a useful adjunct to low ScvO₂ in the assessment of low cardiac output syndrome in children after cardiac surgery.

Key words: Veno-arterial pCO₂ difference. Cardiac output. Central venous oxygen saturation. Paediatric.

INTRODUCTION

Advancement in the field of paediatric cardiology, cardiac surgery and critical care medicine has led to an increased survival of children having congenital heart diseases.¹ Despite this, the postoperative period can be complicated by a predictable fall in cardiac output which has a significant impact on the future wellbeing of these children. The factors responsible for low cardiac output after cardiopulmonary bypass include myocardial ischemia, hypothermia, reperfusion injury, inflammatory mediators and altered vascular reactivity. Early identification and appropriate management of this perfusion abnormality (low cardiac output syndrome = LCOS) is essential for the smooth recovery of these patients. Various invasive and non-invasive techniques/methods have been described and are in use to estimate the cardiac output in this population. Of these,

mixed venous oxygen saturation (SvO₂) is one of the commonly employed measures for assessing low flow states. According to the Fick principle if oxygen consumption and the arterial content of oxygen remain constant, then SvO₂ is proportional to cardiac output and can be used as a reliable indicator for cardiac index.²⁻⁵ SvO₂ is determined by a catheter in the pulmonary artery while the central venous O₂ saturation (ScvO₂) is measured by a catheter in the superior vena cava, which can be used as surrogate of SvO₂ without involving the complication of pulmonary artery catheterization.

Some studies reported high venoarterial difference in pCO₂ (ΔpCO_2) in hypoperfusive states that can be used as a powerful clinical adjunct to ScvO₂ in predicting low flow states.⁶⁻⁹ The purpose of this study was to determine the relationship between these two markers of cardiac index that is ΔpCO_2 and ScvO₂ during the postoperative course of the children who underwent cardiac surgery using cardiopulmonary bypass.

METHODOLOGY

A retrospective chart review of children, who underwent cardiac surgery for correction of their congenital heart

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defects, using cardiopulmonary bypass from June 2005 to May 2006 at the Aga Khan University Hospital (AKUH), Karachi was conducted. Patients with single ventricular physiology and with residual shunt, as determined by echocardiography, were excluded from the study. The study protocol was approved by Ethical Review Committee of AKUH.

Demographic details, primary diagnosis and the values of simultaneous arterial and venous blood parameters were recorded on the data sheet. Arterial blood samples were drawn either from the radial or femoral artery while venous blood samples were obtained from the superior vena cava. These samples were analyzed immediately in an Automatic Blood Gas System (Stat Profile pHox, Nova Biomedical Waltham, MA, USA). Values of ΔpCO_2 were calculated by subtracting venous pCO_2 from arterial pCO_2 . For the purpose of the study; $\Delta pCO_2 > 6$ mmHg and $ScvO_2 < 70\%$ represented hypoperfusion.^{10,11} Data was expressed as mean \pm SD or percentages as appropriate. Chi-square test for categorical variables and student t-test for continuous variables were used for statistical comparison. P-value of < 0.05 was considered statistically significant. Linear regression analysis was applied to measure the degree of correlation between the ΔpCO_2 and $ScvO_2$ by using the Pearson correlation coefficients. The statistical analysis was performed by using SPSS version 14 (SPSS Inc. Chicago, IL, USA).

RESULTS

Fifty seven children underwent cardiac surgery during the study period having a mean age of 14 months ranging from 5 days to 14 years. Table I shows the type of congenital heart defect for which cardiac surgery was required. A total of 272-paired simultaneous arterial and venous samples were collected for blood gas analysis. The mean venous pCO_2 was 47.8 ± 9 mmHg and the mean arterial pCO_2 was 40.5 ± 9 mmHg ($p < 0.001$) and mean ΔpCO_2 was 7 ± 4 mmHg. In 148 (54.4%) out of 272

Table I: List of congenital heart disease in study population.

Congenital heart disease	n
Tetralogy's of Fallot	20
Ventricular Septal Defect (VSD)	10
ASD and VSD	10
Atrial Septal Defect (ASD)	8
Aortic stenosis	3
Total Anomalous pulmonary venous return	3
Transposition of great arteries	2
Atrioventricular canal defect	1

Table 2: Number of samples within each range of ΔpCO_2 in relation to $ScvO_2$.

$ScvO_2$ %	Number of samples having	
	$\Delta pCO_2 > 6$ mmHg	$\Delta pCO_2 < 6$ mmHg
$< 70\%$ (n=98)	77	21
$\geq 70\%$ (n=174)	71	103
Total (n=272)	148	124

samples ΔpCO_2 was elevated (> 6 mmHg). Mean ΔpCO_2 was 9.0 ± 3.9 mmHg when $ScvO_2$ was $< 70\%$ while mean ΔpCO_2 was 5.4 ± 2.55 mmHg when $ScvO_2$ was $> 70\%$. When $ScvO_2$ was $> 70\%$ more than half (59.2%) of the samples had delta pCO_2 of < 6 mmHg and 41% had $\Delta pCO_2 > 6$ mmHg. However, in those patients who had $ScvO_2$ of $< 70\%$, delta pCO_2 of > 6 mmHg was observed in 79% of patients as compared to only 21% of patients with delta pCO_2 of < 6 mmHg ($p < 0.001$). Linear regression analysis of delta pCO_2 versus $ScvO_2$ revealed $R^2=0.340$ (Figure 1).

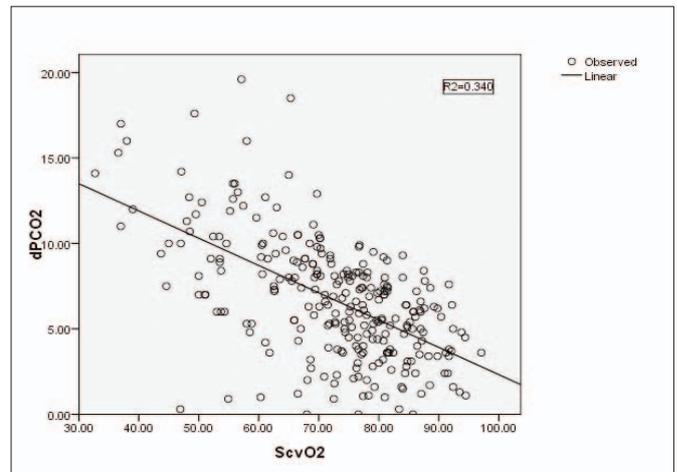


Figure 1: Linear regression analysis: Relation between ΔpCO_2 (dpCO₂) and central venous oxygen saturation $ScvO_2$.

The mean venous pH value was 7.39 ± 0.065 and the mean arterial pH value was 7.43 ± 0.079 . Moreover, forty-four percent (44%) and 28% of samples had pH differences of greater than 0.05 when $ScvO_2$ was $< 70\%$ and $\geq 70\%$ respectively.

DISCUSSION

In this study, $ScvO_2$ was found to have an inverse relation with ΔpCO_2 . Razi *et al.* and McBride *et al.* reported similar findings in their studies.^{12,13} Razi *et al.* did not mention the source of venous blood while McBride *et al.* utilized both the pulmonary artery and superior vena cava to obtain venous blood samples.^{12, 13} By Fick's law, it is SvO_2 rather than $ScvO_2$ that is proportional to the cardiac output provided the arterial oxygen content and oxygen consumption remain constant. The studies by Rocha *et al.* and Waller *et al.* also suggest the same.^{2,3} However, in the paediatric population it is difficult to obtain mixed venous blood through the pulmonary artery. Therefore, many studies have been carried out to uncover the relation between SvO_2 and $ScvO_2$. These have reported that the oxygen saturation in the superior vena cava (Central venous saturation ($ScvO_2$)) and approximate pulmonary artery saturation (mix venous saturation (SvO_2)) is close enough to be used as a surrogate of SvO_2 .^{1,14-16} As a

consequence paediatricians assess cardiac output usually on the basis of central venous oxygen saturation. Likewise for calculating $\Delta p\text{CO}_2$, superior vena cava blood is employed instead of pulmonary artery blood.

Studies highlighting the significance of $\Delta p\text{CO}_2$, for assessment of hypoperfusive states strongly advocate its specificity for this purpose unless pulmonary impairment is present.^{6,7,14-23} It should be clear that $\Delta p\text{CO}_2$ does not indicate hypoxia but ischemic hypoxia as proved by Vallet *et al.*²² Increase in CO_2 production either by non-ischemic hypoxia (anaerobic metabolism) or aerobically during early stages of septic shock (because of high flow) alone cannot cause venous hypercarbia as it can easily be cleared by high venous flow. Thus only low flow states can increase in $\Delta p\text{CO}_2$, regardless of the cause of the circulatory failure, provided normal gas exchange occurs at the pulmonary membrane. The causes of venous hypercarbia in low flow states are multiple. Reduced pulmonary flow leads to increased ventilation to perfusion ratio causing widening of the veno-arterial $p\text{CO}_2$ gradient. Increased production of CO_2 is because of buffering of acids produced during anaerobic metabolism. Others are decarboxylation of metabolic intermediates, and aerobic production of CO_2 . The last is minimal during low flow states.

Confounding factors may play a role while considering low ScvO_2 as an indicator of low cardiac output because it depends upon other variables as well. Haemoglobin concentration, partial pressure of oxygen in the arterial tree and oxygen consumption can all affect ScvO_2 . Keeping haemoglobin concentration constant in a patient is not that difficult and the arterial partial pressure of oxygen depends on inspired oxygen concentration and pulmonary exchange which is a prerequisite for elevation of $\Delta p\text{CO}_2$ as well. Therefore, oxygen consumption is the only factor that can potentially confound the relation of ScvO_2 and cardiac output and hence the relation with $\Delta p\text{CO}_2$. After cardiopulmonary bypass, oxygen utilization increases in almost all cases to match the oxygen debt (oxygen uptake by myoglobin mainly to re-establish oxygen stores) without producing CO_2 . This increase in oxygen consumption without affecting CO_2 production can influence the relation of ScvO_2 with cardiac output.

In addition we have also observed that 44% of our patients had a pH difference of greater than 0.05 when ScvO_2 was < 70% while only 28% patient with > 70% saturation had significant pH difference. Thus $\Delta p\text{CO}_2$ and pH gradient increased with decreasing ScvO_2 . Therefore, both can be used as adjunct to indicate cardiac output besides ScvO_2 . Other investigators like Zhang *et al.* and Adroque *et al.* also explained the widening of veno-arterial $p\text{CO}_2$ and pH differences on

behalf of decreasing cardiac output, provided that alveolar ventilation should be normal.^{8,11} Being retrospective in nature, this study lacks randomization in patient's selection. Oxygen consumption was not measured during the postoperative care of the patients which may have potentially affected ScvO_2 , its relationship with cardiac output and therefore, the final results of this study. Another limitation of the study worth mentioning is that it did not directly measure the predictive value of $\Delta p\text{CO}_2$ in relation to cardiac output. No clinical or outcome parameters were used to prove the presence or absence of a low cardiac output state in the patient population. The study indirectly proves the relationship between $\Delta p\text{CO}_2$ and cardiac output by showing strong relationships between ScvO_2 , SvO_2 and $\Delta p\text{CO}_2$ as mixed venous saturation have previously been shown to correlate to a state of cardiac output.

CONCLUSION

Prompt identification and management of LCOS is essential to the critical care of children with heart disease and may improve the outcome. Elevated $\Delta p\text{CO}_2$ is a practical marker and can be utilized as a useful adjunct to low ScvO_2 in the assessment of LCOS in children after cardiac surgery. Further studies are needed to extend this correlation in the low-systemic flow states.

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