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Venoarterial PCO2 difference: a marker of postoperative cardiac output in children with congenital heart disease

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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 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 ∆pCO2.

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 ∆pCO2 of 5.44±2.55 mmHg and 98 samples had ScvO2 < 70% with mean ∆pCO2 of 9.07±3.90 mmHg. With ScvO2 < 70%, 77 samples had ∆pCO2 of > 6 mmHg while only 21 samples had ∆pCO2 of < 6 mmHg (p < 0.001). On the contrary with ScvO2 > 70%, 71 samples had ∆pCO2 of > 6 mmHg and 103 samples had ∆pCO2 of < 6 mmHg. Coefficient of correlation (R2) between ScvO2 and ∆pCO2 was 0.340.

CONCLUSION: Elevated ∆pCO2 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.

ABSTRACT
Objective: To determine the relationship between venoarterial carbon dioxide gradient (∆pCO2) 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 ∆pCO2.

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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 (∆pCO2) 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 ∆pCO2 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
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 $\Delta pCO_2$ 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 ± 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 $\Delta pCO_2$ 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 $\Delta pCO_2$ was 7±4 mmHg. In 148 (54.4%) out of 272 samples $\Delta pCO_2$ was elevated (> 6 mmHg). Mean $\Delta pCO_2$ was 9.0±3.9 mmHg when ScvO$_2$ was < 70% while mean $\Delta pCO_2$ 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 R2=0.340 (Figure 1).

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 .05 when ScvO$_2$ was < 70% and ≥ 70% respectively.

DISCUSSION

In this study, ScvO$_2$ was found to have an inverse relation with $\Delta pCO_2$. 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 $\Delta pCO_2$, superior vena cava blood is employed instead of pulmonary artery blood.

Studies highlighting the significance of $\Delta pCO_2$, for assessment of hypoperfusive states strongly advocate its specificity for this purpose unless pulmonary impairment is present.\(^5\)\(^6\)\(^7\)\(^14\)\(^15\)\(^22\)\(^23\) It should be clear that $\Delta pCO_2$ 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 $\Delta pCO_2$, 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 pCO$_2$ 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 $\Delta pCO_2$ 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 $\Delta pCO_2$. After cardiopulmonary bypass, oxygen utilization increases in almost all cases to match the oxygen debt (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 $\Delta pCO_2$ 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 Adrogué et al. also explained the widening of veno-arterial pCO$_2$ 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 $\Delta pCO_2$ 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 $\Delta pCO_2$ and cardiac output by showing strong relationships between ScvO$_2$, SvO$_2$ and $\Delta pCO_2$ 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 $\Delta pCO_2$ 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.

**REFERENCES**

Central venous-to-arterial carbon-dioxide difference: an additional target for goal-directed therapy in septic shock?* 


