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. 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 (SvO\textsubscript{2}) 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 SvO\textsubscript{2} is proportional to cardiac output and can be used as a reliable indicator for cardiac index. SvO\textsubscript{2} 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 ScvO\textsubscript{2} and \Delta pCO\textsubscript{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 pCO\textsubscript{2} was 47.82±9.03 mmHg and mean arterial pCO\textsubscript{2} was 40.50±9.06 mmHg. One hundred seventy four samples had ScvO\textsubscript{2} > 70% with mean \Delta pCO\textsubscript{2} of 5.44±2.55 mmHg and 98 samples had ScvO\textsubscript{2} < 70% with mean \Delta pCO\textsubscript{2} of 9.07±3.90 mmHg. With ScvO\textsubscript{2} < 70%, 77 samples had \Delta pCO\textsubscript{2} of > 6 mmHg while only 21 samples had \Delta pCO\textsubscript{2} of < 6 mmHg (p < 0.001). On the contrary with ScvO\textsubscript{2} > 70%, 71 samples had \Delta pCO\textsubscript{2} of > 6 mmHg and 103 samples had \Delta pCO\textsubscript{2} of < 6 mmHg. Coefficient of correlation (R\textsuperscript{2}) between 0.340 was ScvO\textsubscript{2} and \Delta pCO\textsubscript{2}.

CONCLUSION: Elevated \Delta pCO\textsubscript{2} is practical and can be utilized as a useful adjunct to low ScvO\textsubscript{2} in the assessment of low cardiac output syndrome in children after cardiac surgery.

Key words: Veno-arterial pCO\textsubscript{2} difference. Cardiac output. Central venous oxygen saturation. Paediatric.
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 $\Delta p\text{CO}_2$ were calculated by subtracting venous $p\text{CO}_2$ from arterial $p\text{CO}_2$. For the purpose of the study; $\Delta p\text{CO}_2 > 6$ 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 $\Delta p\text{CO}_2$ 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 $p\text{CO}_2$ was 47.8±9 mmHg and the mean arterial $p\text{CO}_2$ was 40.5±9 mmHg (p < 0.001) and mean $\Delta p\text{CO}_2$ was 7±4 mmHg. In 148 (54.4%) out of 272 samples $\Delta p\text{CO}_2$ was elevated (> 6 mmHg). Mean $\Delta p\text{CO}_2$ was 9.0±3.9 mmHg when ScvO$_2$ was < 70% while mean $\Delta p\text{CO}_2$ 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 $\Delta p\text{CO}_2$ of < 6 mmHg and 41% had $\Delta p\text{CO}_2 > 6$ mmHg. However, in those patients who had ScvO$_2$ of < 70%, delta $p\text{CO}_2$ of > 6 mmHg was observed in 79% of patients as compared to only 21% of patients with delta $p\text{CO}_2$ of < 6 mmHg (p < 0.001). Linear regression analysis of delta $p\text{CO}_2$ versus ScvO$_2$ revealed R$^2$=0.340 (Figure 1).

![Figure 1: Linear regression analysis: Relation between $\Delta p\text{CO}_2$ (dpCO2) and central venous oxygen saturation ScvO$_2$.](image-url)

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 $\Delta p\text{CO}_2$. 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.\textsuperscript{1,6,7,14-23} It should be clear that $\Delta pCO_2$ does not indicate hypoxia but ischemic hypoxia as proved by Vallet et al.\textsuperscript{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 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 $\Delta pCO_2$ 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.\textsuperscript{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**


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