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. 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. Venoarterial pCO₂ difference (ΔpCO₂) 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 ΔpCO₂.

METHODOLOGY

A retrospective chart review of children, who underwent cardiac surgery for correction of their congenital heart disease.
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 ∆pCO2 were calculated by subtracting venous pCO2 from arterial pCO2. For the purpose of the study; ∆pCO2 > 6 mmHg and ScvO2 < 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 ∆pCO2 and ScvO2 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 pCO2 was 47.8±9 mmHg and the mean arterial pCO2 was 40.5±9 mmHg (p < 0.001) and mean ∆pCO2 was 7±4 mmHg. In 148 (54.4%) out of 272 samples ∆pCO2 was elevated (> 6 mmHg). Mean ∆pCO2 was 9.0±3.9 mmHg when ScvO2 was < 70% while mean ∆pCO2 was 5.4±2.55 mmHg when ScvO2 was > 70%. When ScvO2 was > 70% more than half (59.2%) of the samples had delta pCO2 of < 6 mmHg and 41% had ∆pCO2 > 6 mmHg. However, in those patients who had ScvO2 of < 70%, delta pCO2 of > 6 mmHg was observed in 79% of patients as compared to only 21% of patients with delta pCO2 of < 6 mmHg (p < 0.001). Linear regression analysis of delta pCO2 versus ScvO2 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 ScvO2 was < 70% and ≥ 70% respectively.

DISCUSSION

In this study, ScvO2 was found to have an inverse relation with ∆pCO2. 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 SvO2 rather than ScvO2 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 SvO2 and ScvO2. These have reported that the oxygen saturation in the superior vena cava (Central venous saturation (ScvO2)) and approximate pulmonary artery saturation (mix venous saturation (SvO2)) is close enough to be used as a surrogate of SvO2.1,14-16 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. It should be clear that $\Delta pCO_2$ does not indicate hypoxia but ischemic hypoxia as proved by Vallet et al. 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 ScvO2 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 ScvO2. 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 ScvO2 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 ScvO2 with cardiac output.

In addition we have also observed that 44% of our patients had a pH difference of greater than 0.05 when ScvO2 was < 70% while only 28% patient with > 70% saturation had significant pH difference. Thus $\Delta pCO_2$ and pH gradient increased with decreasing ScvO2. Therefore, both can be used as adjunct to indicate cardiac output besides ScvO2. 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.

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 ScvO2, 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 ScvO2, SvO2 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 ScvO2 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**


