Original Article

This article is accompanied by an invited commentary by Prof. Muralidhar Kanchi

The effects of different ventilator modes on cerebral tissue oxygen saturation in patients with bidirectional superior cavopulmonary connection

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ABSTRACT

Aims and Objectives: We used near-infrared spectroscopy to document changes in cerebral tissue oxygen saturation ($SctO_2$) in response to ventilation mode alterations after bidirectional Glenn (BDG; superior cavopulmonary connection) procedure. We also determined whether spontaneous ventilation have a beneficial effect on hemodynamic status, lactate and $SctO_2$ when compared with other ventilation modes. **Materials and Methods:** 20 consecutive patients undergoing BDG were included. We measured $SctO_2$ during three ventilator modes (intermittent positive-pressure ventilation [IPPV]; synchronized intermittent mandatory ventilation [SIMV]; and continuous positive airway pressure + pressure support ventilation [CPAP + PSV]). We, also, measured mean airway pressure (AWP), arterial blood gases, lactate and systolic arterial pressures (SAP). **Results:** There was no change in $SctO_2$ in IPPV and SIMV modes; the $SctO_2$ measured during CPAP + PSV and after extubation increased significantly (60.5 ± 11, 61 ± 10, 65 ± 10, 66 ± 11 respectively) ($P < 0.05$). The differences in the SAP measured during IPPV and SIMV modes was insignificant; the SAP increased significantly during CPAP + PSV mode and after extubation compared with IPPV and SIMV (109 ± 11, 110 ± 12, 95 ± 17, 99 ± 13 mmHg, respectively) ($P < 0.05$). Mean AWP did not change during IPPV and SIMV modes, mean AWP decreased significantly during CPAP + PSV mode (14 ± 4, 14 ± 3, 10 ± 1 mmHg, respectively) ($P < 0.01$). **Conclusions:** The $SctO_2$ was higher during CPAP + PSV ventilation and after extubation compared to IPPV and SIMV modes of ventilation. The mean AWP was lower during CPAP + PSV ventilation compared to IPPV and SIMV modes of ventilation.

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INTRODUCTION

Bidirectional Glenn (BDG) procedure commits venous return from the head and upper body directly into the pulmonary circulation. In this physiology, an increase in superior vena cava (SVC) and pulmonary artery pressures secondary to high pulmonary vascular resistance (PVR) results in decreased cerebral and systemic oxygenation.[1] Recently, several reports have addressed the significance of early spontaneous ventilation and early extubation after BDG and total-cavopulmonary shunt (TCPC).[2-5] These studies reported decreased mean pulmonary artery pressure and enhanced hemodynamic performance in patients with BDG or TCPC after early extubation.[3-5] Although these studies showed that reducing the duration of artificial ventilation by early extubation could minimize potential detrimental effects of positive pressure ventilation,[3-5] it is important to know the effect of different ventilation modes used during the weaning period after BDG and TCPC on organ perfusion. Walsh *et al*. noted that ventilation with airway pressure (AWP) release ventilation improves pulmonary blood flow compared

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with pressure control ventilation in children after tetralogy of Fallot repair and cavopulmonary shunt operations.[6] Walsh *et al*. hypothesize that shorter the duration of the intrathoracic pressure application, better the pulmonary blood flow.[6] The FORE-SIGHT Absolute Cerebral Oximeter is an optical technique that continuously and noninvasively measures cerebral tissue oxygen saturation ($SctO_2$) at the microvasculature level in the frontal lobes.[7] Monitoring $SctO_2$ provide real-time changes in regional tissue oxygenation in cardiac surgery during total circulatory arrest, venous cannula obstruction or sudden global hypoxemia and an absolute value of 50% is considered an indication of potential hypoxic injury and warrants intervention.[7,8] It's utility to assess the adequacy of cardiac output and systemic oxygenation may be valuable in Glenn patients in changing the ventilation modes during perioperative period. Primary purpose of this study was to evaluate the influence of different ventilation modes: intermittent positive-pressure ventilation [IPPV]; synchronized intermittent ventilation [SIMV]; and continuous positive airway pressure + pressure support ventilation [CPAP + PSV] on $SctO_2$ in patients after BDG, by using near-infrared spectroscopy. The secondary aim was to determine whether spontaneous ventilation mode have any beneficial effect on hemodynamic status and lactate levels when compared with other ventilation modes.

MATERIALS AND METHODS

Between July 2010 and October 2011, 20 consecutive children undergoing BDG were included in the study. The protocol was approved by the Institutional Review Board and informed consent was obtained from the parents. Before anesthesia, 1 mg/kg intravenous (IV) dexamethasone was administered to all patients. Midazolam 0.05 mg/kg IV was used for premedication in the operating room. In addition to standard monitoring (noninvasive blood pressure, pulse-oximetry and electrocardiograph, capnography and nasopharyngeal temperature) invasive arterial, central venous pressure (CVP) and SVC pressure monitoring was performed. Anesthesia was induced with thiopental (2-4 mg/kg), fentanyl (4-6 μg/kg) and rocuronium (0.6 mg/kg) and patients were intubated with cuffed endotracheal tubes. The lungs were ventilated intermittently with 50% O_2/air mixture with a tidal volume of approximately 8 ml/kg and the peak airway pressure (AWP) was kept lower than 25 cmH_2O; the respiratory rate was adjusted to ensure a partial pressure of carbon dioxide ($PaCO_2$) of 40-50 mmHg. Anesthesia was maintained with fentanyl infusion (2-4 μg/kg/h) and 0.5% to 0.8% isoflurane. Anesthesia depth was monitored with index of consciousness (IOC) (Morpheus Medical, Barcelona, Spain)[9] and the IOC values were maintained between 40 and 60.

All operations were performed using standard cardiopulmonary bypass (CPB). After median sternotomy, the patients were anticoagulated with heparin 2 mg/kg and CPB was initiated using standard aortic, right atrium and left innominate vein cannulation. Full-flow CPB without cardioplegia was performed under conditions of moderate hemodilution, maintaining the hematocrit level between 32% and 35% and hypothermia of 34-35°C. All systemic-to-pulmonary artery shunts and native main pulmonary arteries were divided at the time of BDG. Phentolamine mesylate was routinely administered to the pump circuit at initiation of CPB and during rewarming. Continuous ultrafiltration was performed throughout CPB and modified ultrafiltration was performed after CPB for 10-15 min. The transpulmonary pressure gradient (TPG) was measured after modified ultrafiltration. In patients with BDG, the TPG is the difference between mean pulmonary arterial pressure (mPAP) and right atrial pressure as the two atria are connected via an existing or iatrogenic large atrial septal defect. After construction of BDG, SVC is committed to pulmonary artery and the central venous catheter monitors the mPAP.

Regional oxygen saturation monitoring

Regional oxygen saturation was monitored with FORE-SIGHT Cerebral Oximeter (CAS Medical Systems, Branford, CT, USA), which shows dual-site $SctO_2$ monitoring with pediatric disposable sensor. The FORE-SIGHT cerebral oximeter continuously measures $SctO_2$ without the need for baseline calibration and absolute values are updated every 2 s. The sensors are positioned bilaterally on the patients' lower forehead and covered by an opaque plastic patch in order to prevent the effect of ambient light on the measurements. Cerebral oximetry measures $SctO_2$ at the microvasculature level, which consists of about 70% venous and 30% arterial blood; therefore, $SctO_2$ value is roughly 10% above jugular venous saturation under most clinical condition. The data collection started before induction of anesthesia and ended in the intensive care unit (ICU) after extubation; the data were extracted in excel spreadsheet. We aimed to maintain the bilateral $SctO_2$ level at higher than 50% during the study and its values were recorded at the specified time points.

ICU management

In ICU, the patients were connected to Servo 300 ventilator (Siemens-Elema, Solna, Sweden) and the head-ends of the beds raised to 45°. All patients received time-cycled (I: E ratio 1:2) IPPV with a tidal volume of 8 ml/kg at a fractional inspired oxygen concentration of ≤ 0.40 and a positive end-expiratory pressure (PEEP) of 5 cmH_2O was applied during ventilation. The respiratory rate was adjusted to ensure a $PaCO_2$ of 40-50 mmHg. The peak AWP was kept lower than 25 cmH_2O. The weaning from ventilation began by ventilating the patients with the SIMV volume control mode at 20 SIMV rate with PEEP 5 cmH_2O and pressure-support (PS) 5 cmH_2O. If the patient remained clinically stable, ventilator support was changed first to SIMV rate 8-10, PEEP 5 cmH_2O and PS 6-8 cmH_2O, then to CPAP of 5 cmH_2O and PS 6-8 cmH_2O when patients were ready for extubation. In each ventilation mode, the patients were observed for at least 2 h. As per our institute protocol arterial oxygen saturation, as measured by pulse oximetry, was maintained between 75% and 90%, and the arterial pH of at least 7.32, permitting moderate hypercapnia with a $PaCO_2$ of 40-50 mmHg. Weaning was delayed if the patients developed an increase or decrease in heart rate (HR) of more than 30 beats per minute above pre-weaning values, a decrease in systolic arterial pressures (SAP) of more than 30 mmHg, and pH below 7.32. Other reasons to defer the weaning process included development of tachypnea (respiratory rate > 50 per min or the appearance of a new arrhythmia after arrival in the ICU. Morphine (10-30 μg/kg/h) was used to provide analgesia and sedation. Hematocrit was maintained at 35% to 40% during the study. The SAP, diastolic (DAP), and mean arterial pressure (MAP), temperature, HR, peripheral oxygen saturation (SpO_2), end-tidal CO_2, IOC and $SctO_2$ values, FiO_2, arterial blood gases analysis (pO_2, pCO_2, pH, and lactate), hematocrit and inotrope score [dopamine + dobutamine + (adrenaline × 100) + (milrinone × 10] were recorded at the following times - before induction (baseline); after induction; during and after CPB; at the termination of modified ultrafiltration and every 2 h in the ICU until 2nd h after extubation. In addition, $SctO_2$ values < 50% occurring at any time and persisting for at least 10 min was also recorded. Statistical analyses were performed using SPSS™ for Windows software (Version 15.0, Statistical Package for the Social Sciences, Cary, NC). The Kolmogorov-Smirnov test was used to verify the normality of the data distribution. Descriptive statistics (means, standard deviations, frequencies) were calculated on all variables. The variations were analyzed by paired *t*-test and Wilcoxon paired-signed rank test as applicable. Nominal data between groups were compared with the Chi-square test. $P < 0.05$ was accepted as statistically significant.

RESULTS

20 consecutive patients undergoing BDG were included in the study. The ages of patients ranged between 3 and 24 months. The cardiac pathologies were hypoplastic left heart syndrome ($n = 1$), tricuspid atresia ($n = 7$), heterotaxy syndrome ($n = 1$), tetralogy of Fallot/complete atrioventricular (AV) canal defect (unbalanced ventricle) ($n = 1$), double-inlet (left or right) ventricle ($n = 6$), pulmonary atresia with intact ventricular septum ($n = 1$) and unbalanced common AV canal ($n = 3$). Pre-operatively, mPAP and ejection fraction were 15 ± 2.7 mmHg and 64 ± 14%. Demographics and operative characteristics of the patients are shown in Table 1.

Average $SctO_2$ values significantly increased after anesthesia induction and during CPB compared to the baseline value (64 ± 10% and 65 ± 7% vs. 59.8 ± 10%) ($P < 0.05$). The average $SctO_2$ values did not change after CPB and after modified ultrafiltration when compared to the baseline value (60 ± 7% and 61 ± 6% vs. 59.8 ± 10%). The baseline average value was under 50 in 2 patients (32% and 48%). In these 2 patients, $SctO_2$ values increased to 48% and 52% after CPB. Average $SctO_2$ values during IPPV mode and SIMV mode in ICU were similar (60.5 ± 11% and 61 ± 10%, respectively). Average $SctO_2$ values significantly increased during CPAP + PS and after extubation compared with IPPV (65 ± 10% and 66 ± 11%, respectively) ($P < 0.05$) [Figure 1]. Average $SctO_2$ values did not change after extubation when compared to CPAP + PS values. In ICU, duration of IPPV, SIMV and CPAP were 2 ± 1 h, 12 ± 10, and 2 ± 0.7 h, respectively. 422-paired measurements (left and right cerebral $SctO_2$) from 20 patients were recorded. 58-paired measurements (13.7%) from 7 patients showed temporary decrease of more than

Table 1: Demographics and operative characteristics

Age (months)	11.2 ± 8
Weight (kg)	8.3 ± 3.5
Male/female (no)	13/7
Duration of CPB (min)	83 ± 19
Duration of MHF (min)	12 ± 5
TPG (mmHg)	7.4 ± 2.5
mPAP (mmHg)	12.9 ± 3.4
Atrium pressure (mmHg)	5.6 ± 2.1

CPB: Cardiopulmonary Bypass, MHF: Modified hemofiltration, TPG: Transpulmonary gradient, mPAP: Mean pulmonary arterial pressure, CVP: Central venous pressure

50% of $SctO_2$. The distribution of the paired cerebral oximetry values below 50% during various ventilation modes were - IPPV 26 (45%), SIMV 19 (30%), CPAP 7 (12%) and after extubation 6 (10%).

The SpO_2, end-tidal CO_2, pCO_2, pH, hematocrit, blood lactate, and body temperatures were not different in IPPV, SIMV, CPAP + PSV modes and after extubation. The difference in SAP, DAP and MAP did not differ statistically during IPPV and SIMV modes; the blood pressures significantly increased during CPAP + PSV mode and after extubation [Table 2]. The mean AWP was statistically not different during IPPV and SIMV modes, but there was statistically significant decrease in the mean AWP during CPAP + PSV mode compared to IPPV mode ($P < 0.01$) [Table 2]. The mPA*P* values were not different during IPPV and SIMV modes, but decreased significantly during CPAP + PSV mode and after extubation ($P < 0.01$) [Table 2]. Inotropic scores were not different during IPPV and SIMV modes, but decreased during CPAP + PSV mode and after extubation ($P < 0.05$) [Table 2]. Mean extubation time was 18.8 ± 11.6 h, length of ICU stay was 4.2 ± 2.5 days and length of hospital stay was 12 ± 11 days. There was one post-operative death from pneumonia in a patient with unbalanced complete AV canal defect/tetralogy of Fallot.

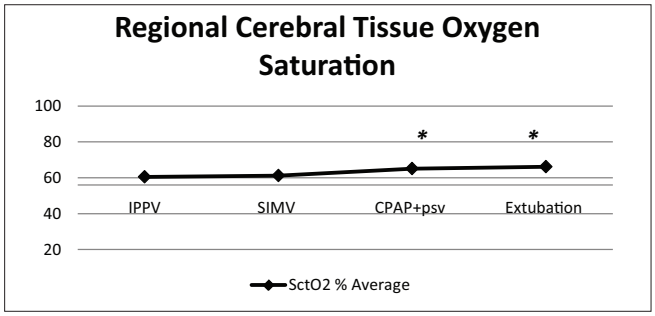

Figure 1: Changes in regional cerebral tissue oxygen saturation in intensive care unit *$P < 0.05$

DISCUSSION

Our study shows that $SctO_2$ and hemodynamic parameters improve with the elimination of positive pressure ventilation. The increase in the average $SctO_2$ and the enhanced hemodynamic performance with the CPAP + PSV mode were similar to the values noted after extubation. It has long been known that the generation of negative intrathoracic pressure with spontaneous ventilation improves pulmonary blood flow compared to positive pressure ventilation, which is known to impair venous return and cardiac output. It is generally accepted that pulmonary perfusion and cardiac output are optimized with negative inspiratory pressure generated during spontaneous ventilation.[9] The improvement in pulmonary blood flow and therefore cardiac output is expected to improve cerebral oxygenation. After BDG with interruption of the main pulmonary artery, the pulmonary circulation is non-pulsatile and passive. The pulmonary flow is influenced adversely by high PVR. Positive intrathoracic pressures lead to reduction in both pulmonary flow and systemic ventricle preload.[10-12] Hypoventilation improves systemic oxygenation in patients after BDG.[13] Hypoventilation-induced hypercarbia decreases cerebral vascular resistance, which results in increased cerebral blood flow, and consequently an increase in SVC and pulmonary blood flow.[13] The improvement in cerebral

Table 2: Summary of clinical variables

	Mean ± SD				*P*		
	IPPV	SIMV	CPAP+PSV	Extubation	IPPV-SIMV	IPPV-CPAP+PSV	IPPV-extubation
SpO_2	78 ± 10	77 ± 9	75 ± 8	77 ± 8	0.246	0.123	0.544
End-tidal CO_2	36 ± 5	38 ± 5	39 ± 5	-	0.189	0.147	-
$PaCO_2$	46 ± 4	48 ± 5	45 ± 5	46 ± 7	0.563	0.068	0.104
Hematocrit	37 ± 3	35 ± 2	36 ± 2	36 ± 3	0.069	0.093	0.066
SAP	95 ± 18	99 ± 14	109 ± 11	110 ± 12	0.703	0.038*	0.042*
DAP	50 ± 10	55 ± 9	60 ± 11	61 ± 10	0.057	0.009**	0.002**
MAP	67 ± 11	71 ± 11	79 ± 10	79 ± 10	0.138	0.007**	0.005**
Lactate	1.9 ± 1	2.2 ± 0.9	1.8 ± 0.8	2.0 ± 0.6	0.126	0.712	0.173
Mean airway pressure	14 ± 4	14 ± 3	10 ± 1	-	0.077	0.001**	-
Temperature	36 ± 0.6	36 ± 0.5	36 ± 0.6	36 ± 0.6	0.139	0.843	0.250
pH	7.3 ± 0	7.3 ± 0	7.3 ± 0	7.3 ± 0	0.117	0.063	0.067
CVP	15 ± 2	14 ± 3	13 ± 3	12 ± 2	0.078	0.003**	0.001**
Inotropic scores	9.9 ± 4	8.7 ± 5	7.3 ± 5	7.5 ± 6	0.147	0.050*	0.049*

*$P < 0.05$, **$P < 0.01$. IPPV: Intermittent positive-pressure ventilation, SIMV: Synchronized intermittent mandatory ventilation, SAP: Systolic arterial pressure, DAP: Diastolic arterial pressure, MAP: Means arterial pressure, CPAP: Continuous positive airway pressure, CVP: Central venous pressure, PSV: Pressure support ventilation, SD: Standard deviation

oxygen saturation and hemodynamic parameters during CPAP + PSV can be explained by increased functional residual capacity, moderate hypoventilation and the decreased mean AWP (mean 10±1), which remained below pulmonary artery pressure. It is important to note that the use of CPAP with PSV was as beneficial as spontaneous ventilation alone in increasing cerebral oxygenation. This provides some freedom to the clinicians in deciding when to remove the endotracheal tube in situations where the clinical status of the patients is uncertain. However, any respiratory complication such as atelectasis, airway compromise, pneumothorax or pleural effusions, and other complications like hemodynamic instability outweigh the potential benefits of early extubation.[10] A suitable ventilation mode with minimum effect on pulmonary blood flow is necessary in situations where prolonged mechanical ventilatory support is required. The finding of our study is particularly important for cases in which it is difficult to make a decision to extubate, keeping the patients in CPAP + PSV mode until the hemodynamic compromise ceases may be highly beneficial.

Many studies have shown beneficial effects of early extubation after creation of BDG or after a Fontan operation.[2-5] Lofland reported early extubation in 50 consecutive patients undergoing either BDG or completion Fontan.[3] In their study, after resumption of spontaneous respiration and extubation, mPAP decreased and cardiac index increased. In our study, we observed increases in cerebral oxygen saturation and arterial pressure and decrease in mPAP during CPAP + PS and after extubation. Kurihara *et al*. retrospectively compared early extubated patients (<3 h) with late extubated ones (≥3 h) after BDG and TCPC; 54% patients were extubated early.[5] The early extubated patients had lower pre-operative mPAP, mean PVR and mean CVP whereas the late extubated patients (>3 h) had poor pre- and post-operative cardiac functions and higher PVR. Decreased cardiac functions, high PVR and the need for large doses of inotropes and vasodilators are the factors that can delay extubation.[5] In patients having hemorrhagic diathesis, marginally raised mPAP and PVR, right ventricular dominant single ventricle physiology, AV valve insufficiency and/or requiring long aortic cross-clamp time because of additional intracardiac repair, early extubation is usually not feasible. For this group of patients, longer respiratory support is required due to borderline hemodynamics. These patients are likely to be benefited with ventilatory modes that will have minimum negative effect on the hemodynamics.

In patients with BDG, high intrathoracic pressure and high pulmonary artery pressure will impair pulmonary blood flow and cerebral venous drainage. Monitoring $SctO_2$ in these patients may provide real-time information about decreases in cerebral venous drainage. In general, $SctO_2$ is a sensitive index of cerebral hypoperfusion, hypoxia and/or cerebral ischemia, which is one of the main causes of brain injury during and after surgical procedures.[7,8]

In our study, 7 patients had right and/or left $SctO_2$ levels below 50% for a period of 48 ± 15 min. However, during this period the arterial pressure, HR, blood gas measurements, and hematocrit levels were not different from the periods where $SctO_2$ levels were above 50%. There were two other unexpected events. In two patients, post-operative $SctO_2$ levels consistently remained slightly higher than SpO2 values (mean 80). In the second event, a patient was extubated 5 h after admission to the ICU; the patient's SpO2 values remained between 85% and 87% and all other clinical parameters were completely normal, however, average $SctO_2$ values stayed between 43% and 48% respectively. These situations suggest that variations in cerebral oxygen saturation trends should be considered in such situations and be assessed in association with the clinical parameters.

Our study has several limitations, the number of patients included in the study is small; the durations of ventilation is different for each patient; the SIMV mode was initiated between 2nd and 14th h after surgery and CPAP + PSV mode was applied at the end of the 14th h following surgery. However, this variation in duration of ventilation and point of application of weaning mode is unlikely to change the observed $SctO_2$, hemodynamic and respiratory parameters seen during CPAP + PSV and after extubation. The time passed after surgery may have contributed to the hemodynamic improvement.

CONCLUSION

We have shown that CPAP + PSV mode improves cerebral oxygen saturation and hemodynamic parameters and the improvement is similar to the one seen after extubation. The CPAP + PSV mode may be used for prolonged mechanical ventilation after BDG procedure in cases where early extubation is not feasible.

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