Published in final edited form as:

Paediatr Anaesth. 2018 July ; 28(7): 618–624. doi:10.1111/pan.13423.

Correlation between minute carbon dioxide elimination and pulmonary blood flow in single-ventricle patients after stage 1 palliation and 2-ventricle patients with intracardiac shunts: A pilot study

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Summary

Background—Assessment of pulmonary blood flow and cardiac output is critical in the postoperative management of patients with single-ventricle physiology or 2-ventricle physiology with intracardiac shunting. Currently, such hemodynamic data are only obtainable by invasive procedures, such as cardiac catheterization or the use of a pulmonary artery catheter. Ready availability of such information, especially if attainable noninvasively, could be a valuable addition to postoperative management.

Aims—The aim of this study was to assess the correlation between volume of CO₂ elimination obtained by volumetric capnography and pulmonary blood flow in pediatric patients with single-ventricle physiology after stage 1 palliation as well as in patients with other cardiac lesions associated with intracardiac shunting.

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ETHICAL APPROVAL

The Institutional Review Board at Duke University Hospital and Health System approved this prospective study. Parental informed and written consents and patients' assent, when applicable, were obtained. This study was performed according to the Declaration of Helsinki.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Methods—This prospective cohort study included children with congenital or acquired heart disease who underwent cardiac catheterization as part of clinical care. Cardiac output, pulmonary blood flow, and volume of CO₂ elimination were simultaneously collected. Spearman's rank correlation coefficients were used to assess correlation between measurements after controlling for minute ventilation.

Results—Thirty-five patients were enrolled and divided into 3 groups. Group 1 (n = 8) included single-ventricle patients after stage 1 palliation. Group 2 (n = 10) patients had structural heart disease with 2 ventricles and intracardiac shunting. Group 3 (n = 17) had structurally normal hearts. Among Group 1 patients, the correlation coefficients (R^2) between volume of CO₂ elimination and pulmonary blood flow and volume of CO₂ elimination and cardiac output were 0.60 ($P = .02$) 95% CI [0.01–0.79] and 0.29 ($P = .74$) 95% CI [–0.91 – 0.86], respectively. In patients with 2 ventricles associated with intracardiac shunts (Group 2), the correlation coefficients between volume of CO₂ elimination and pulmonary blood flow and volume of CO₂ elimination and cardiac output were 0.86 ($P = .001$) 95% CI [0.53 – 0.97] and 0.73 ($P = .001$) 95% CI [0.29 – 0.95], respectively. Among Group 3 patients, the correlation coefficient between volume of CO₂ elimination and pulmonary blood flow was 0.66 ($P = .038$) 95% CI [0.29 – 0.87].

Conclusion—Volume of CO₂ elimination may be a surrogate marker of pulmonary blood flow in single-ventricle patients and patients with biventricular physiology with intracardiac shunting. Also, among patients with normal cardiac anatomy, volume of CO₂ elimination may be a marker of cardiac output.

Keywords

capnography; cardiac output; congenital heart disease; pulmonary blood flow; volume of CO₂ elimination

1 | BACKGROUND

Cardiorespiratory monitoring is essential in the care of critically ill patients.^{1–3} In the current era, assessment of hemodynamic status in the pediatric cardiac intensive care unit involves the use of arterial lines, intracardiac lines, and occasionally pulmonary arterial catheters.⁴ These invasive lines pose potential risks for infection, intravascular thrombosis, and blood vessel perforation. In an attempt to minimize the risk of such complications, there has been growing interest in the use of noninvasive monitoring, such as near infrared spectroscopy (NIRS) and capnography, to assess hemodynamic status.⁵

Capnography is used to assess respiratory function in critically ill patients.^{6,7} Endtidal carbon dioxide tension (EtCO₂) is the gold standard for determining successful endotracheal intubation⁸ and is used to assess adequacy of chest compressions during cardiopulmonary resuscitation.^{9–12} Additionally, capnography has been used to monitor physiologic dead space ventilation and serves as a guide for optimizing gas exchange during mechanical ventilation.^{13–15}

While capnography has broad applications in respiratory management in the intensive care unit, its utility in patients with heart disease is still under investigation.^{16,17} As exhalation of CO₂ requires perfusion to the alveoli, capnography may be utilized as an indirect

measurement of pulmonary blood flow (Q_P), and in patients with normal cardiac physiology, it may be an indirect assessment of cardiac output (CO_2).

Volume of CO_2 elimination (VCO_2) is the volume of carbon dioxide eliminated per single breath. It is obtained through analysis of the volumetric capnography and is measured in mL/min. VCO_2 is, therefore, dependent on the patient's CO_2 production, pulmonary blood flow, and minute ventilation.¹⁸ At stable minute ventilation and metabolic state, VCO_2 becomes an indirect indicator of Q_P and, by extension, CO in patients with normal cardiac physiology.¹⁹

The correlation between VCO_2 , Q_P , and CO has not been studied in pediatric patients with single-ventricle physiology or in patients with congenital heart disease lesions other than single ventricle. Understanding such correlations may enhance postoperative monitoring and management of critically ill cardiac patients.

2 | MATERIALS AND METHODS

The Institutional Review Board at Duke University Hospital and Health System approved this prospective study of infants and children with cardiac disease undergoing cardiac catheterization under general anesthesia and were intubated as part of routine clinical care. Patients were enrolled between December 2013 and November 2014. Patients less than 18 years from whom assent, when applicable, and parental informed consents were obtained were included. Patients with multiple sources of pulmonary blood flow, including patients with significant collateral blood flow, were excluded due to potential inaccuracy in the assessment of Q_P . Patients with passive pulmonary blood flow (bidirectional Glenn or Fontan physiology) were also excluded due to potential inaccuracies in the assessment pulmonary blood flow resulting from variable contributions from systemic to pulmonary collateral flow.²⁰ Finally, patients on extracorporeal membrane oxygenation (ECMO) at the time of cardiac catheterization, and patients with non-English-speaking parent/guardian were also excluded.

All pediatric cardiac catheterizations (diagnostic and interventional) at our institution were performed under the care of a pediatric cardiac anesthesiologist. All patients underwent general anesthesia, intubated with a cuffed endotracheal tube with goal air leak <10%, and mechanical ventilation during cardiac catheterization as part of standard of care. After intubation, the ventilator settings were adjusted based on arterial blood gases to achieve normal arterial pH and stable minute ventilation. GE Aestiva/5 anesthesia system and 7900 ventilators were used to provide mechanical ventilation (General Electric, Fairfield, CT) and Westmed pediatric dual-limb circuits (Westmed, Tucson, AZ) were used to attach the ventilator to the patient's endotracheal tube. Once stable minute ventilation was established, an NM3 volumetric capnography monitor (Philips, Carlsbad, CA) was connected to the patient's ventilator circuit via a side stream capnostat sensor placed at the proximal end of the endotracheal tube (between the endotracheal tube and the ventilator circuit and proximal to any additional standard anesthesia monitors. NM3 monitor measures VCO_2 per breath and computes VCO_2 eliminated per minute (VCO_2 , in mL/min) by multiplying the area under the receiver operating characteristic curve of the volumetric capnography by the

respiratory rate.²¹ Patients were also connected to standard anesthesia monitoring devices, and cardiac catheterization was then initiated as per routine. All data were collected in real time.

2.1 | Data collection

Ventilator settings required for the establishment of stable minute ventilation were recorded. In addition, capnography data from NM3 monitor, including VCO₂, EtCO₂, tidal volume (Vt), and respiratory rate were recorded every 5 minutes during the entire procedure and at each rest state of hemodynamic assessment. Cardiac catheterization data, including oxygen saturations and pressures in the superior vena cava (SVC), pulmonary artery (PA), and cardiac chambers, were recorded. In patients with single ventricle physiology and with intracardiac shunting, Qp and cardiac output were calculated using the Fick principle. Mixed venous saturation was obtained from the SVC. PA and systemic saturation were assumed to be equal and obtained from the descending aorta. Finally, pulmonary vein saturation was taken from the pulmonary veins. In patients without intracardiac shunting, cardiac index was measured using thermodilution except in cases where there was significant tricuspid or pulmonary valvular regurgitation at which time Fick principle was used (total 2 patients). Mixed venous saturation was obtained directly from the PA, and in situations where the descending aorta saturation was greater than or equal to 96%, the pulmonary vein saturation was assumed to be equal to the descending aorta saturation. Pressures were measured in mmHg, and cardiac output was measured in liters/minute. Since Qp and CO are equal in patients without intracardiac shunting, only CO was reported in these patients.

2.2 | Data analysis

We used standard summary statistics and graphing techniques to explore all study variables. We report medians and ranges for all continuous and counts with percentages for all categorical variables. We compared distribution of study variables using Wilcoxon rank sum, Fisher's exact, and chi-square tests of association where appropriate. To evaluate the correlation between VCO₂ with CO and Qp, we used linear regression and reported R² values with overall F-test p-values. We used STATA12 (College Station, TX) for all statistical analyses and considered a $P < .05$ to represent statistical significance.

3 | RESULTS

Fifty-one consecutive patients were screened during the study period. Sixteen were excluded; 2 patients had pulmonary atresia/stenosis with major aortopulmonary collateral vessels, 7 had bidirectional Glenn or Fontan physiology, and 2 were supported with ECMO at the time of catheterization. Four patients were excluded because of inability to provide consent due to language barrier, and 1 elected not to participate. Of the 35 patients enrolled into the study, 18 had structural heart disease with intracardiac shunts: eight of these had single ventricle with stage 1 palliation (Group 1), and 10 had 2 ventricles with atrial and/or ventricular septal defects (Group 2) (Table S1). Seventeen had structurally normal hearts with no intracardiac shunt (absence of intracardiac shunting was confirmed during cardiac catheterization), 7 of these had valvar heart disease, and 10 had heart transplantation (Group 3) (Figure 1).

Demographics and baseline oxygen saturation for the groups are shown in Table 1. For Group 1, median age was 0.33 years (0.25–0.66), and median oxygen saturation was 81% (70–86); Group 2 had a median age of 0.50 years (0.08–17), and median oxygen saturation was 98% (88–100). Among Group 3 patients, median age was 7 years (1.5–16), and median oxygen saturation was 99% (94–100).

Hemodynamic data obtained during cardiac catheterization are summarized in Table 2. Single-ventricle patients (Group 1) had lower systemic oxygen saturation and mixed venous saturation compared to patients in both Groups 2 and 3. Median cardiac output was 1.06 L/min (0.83–1.54) and cardiac index of 3.68 L/min/m² (3.04–5.29) in Group 1, 1.27 L/min (0.57–9.30) and 3.99 L/min/m² (2.99–5.02) in Group 2, while cardiac output was 4.41 L/min (2.16–7.41) and cardiac index was 3.83 L/min/m² (2.66–8.95) in Group 3. Group 3 patients had no shunts, and, therefore, Qp:Qs was 1; however, in Group 1 median Qp:Qs was 1.11 (0.73–2.07), while median Qp:Qs for Group 2 was 0.99 (0.47–2.0) (Table 2).

Ventilator and volumetric capnography data obtained simultaneously with hemodynamic data are shown in Table 3. Minute ventilation was adjusted to achieve normal physiologic pH and PaCO₂ (35–45 mm Hg) in all patients. Median VCO₂ was 27.1 mL/min (4.6–267.0) (4.57 mL/kg/min) among Group 1 patients, 36.4 mL/min (4.6–267.0) (4.26 mL/kg/min) among Group 2, and 113.5 mL/min (70.8–166) (3.67 mL/kg/min) in patients with structurally normal hearts with no intracardiac shunts (Group 3).

The relationship between VCO₂ and Qp as well as VCO₂ and CO was assessed using linear regression model in groups 1 and 2. Among the single-ventricle patients, VCO₂ and Qp had moderate but statistically significant correlation with R^2 value of 0.60 ($P = .02$) 95% CI [0.01–0.79]. However, the correlation between VCO₂ and CO was weak and not statistically significant with R^2 of 0.29 ($P = .74$) 95% CI [–0.91 – 0.86] (Figure 2A, B). In Group 2, the R^2 for VCO₂ and Qp and VCO₂ and CO were higher at 0.86 ($P < .001$) 95% CI [0.53 – 0.97] and 0.73 ($P < .001$) 95% CI [0.29 – 0.95], respectively (Figure 3A, B). More moderate but significant correlations were found among the patients with no intracardiac shunts (Group 3), with R^2 for VCO₂ and Qp of 0.66 ($P = .038$) 95% CI [0.29–0.87] (Figure 3C).

As one of our subjects in Group 2 had very high VCO₂ and CO, we repeated our analyses excluding that subject, the correlation between VCO₂ and Qp remained significant with $R^2 = 0.66$ ($P = .001$). However, with the subject excluded, the correlation between VCO₂ and CO was weak with $R^2 = 0.35$ ($P = .07$).

4 | DISCUSSION

We report the potential use of volumetric capnography as an intraoperative and bedside monitor for the noninvasive assessment of pulmonary blood flow and Qp:Qs in single-ventricle patients and in patients with 2-ventricle physiology with intracardiac shunts. We also report the application of volumetric capnography to assess, and potentially trend, Qp and, therefore, cardiac output among control patients who have normal intracardiac anatomy. Benefits of this mode of monitoring are that it is readily available, noninvasive, and the data are easily obtained at the bedside. The data are obtained on a continuous basis, and,

therefore, such an approach has the potential for early detection of clinical deterioration such as acute changes in $Q_p:Q_s$ ratio or more seriously shunt thrombosis, which leads to acute loss of pulmonary blood flow. It can also be used to assess the acute impact of therapy on patients' hemodynamic status in this high-risk patient population.

Volume of elimination of carbon dioxide is largely dependent on the patient's metabolic rate, minute ventilation, and pulmonary blood flow. The way CO_2 elimination changes determine the relationship between $EtCO_2$ and VCO_2 . Alteration of minute ventilation changes $EtCO_2$ and VCO_2 in opposite directions, while changes in Q_p or metabolism move $EtCO_2$ and VCO_2 in the same direction. However, in steady-state conditions, when metabolism and minute ventilation are maintained at a constant, carbon dioxide elimination and, hence VCO_2 , represents an assessment of Q_p and by extension CO in patients with normal cardiac physiology and anatomy.^{22,23} Among critically ill patients on mechanical ventilation and sedation, there may be very little variation in metabolic rate and minute ventilation at any given time within each individual patient. Therefore, the use of VCO_2 as a tool for assessment of hemodynamic status could be an important addition to our management tools in the cardiac intensive care unit.

Patients with single-ventricle physiology are at risk for increased physiologic dead-space-to-tidal-volume ratio (VD/VT) due to alveolar ventilation-perfusion (V/Q) mismatch.²⁴ Q_p may be decreased as a result of low cardiac output, increased pulmonary vascular resistance or an acute decrease in systemic vascular resistance. As (VD/VT) can affect VCO_2 , studying single ventricle group aside from other congenital cardiac lesions become necessary. In this study, we demonstrate a correlation between VCO_2 and Q_p in single-ventricle patients after stage 1 palliation and in 2-ventricle patients with intracardiac shunts. Also, we showed the correlation between VCO_2 and CO among patients with normal intracardiac anatomy. In the single-ventricle group, we showed a moderate correlation between VCO_2 and pulmonary blood flow, Q_p . The recognition of acute changes in Q_p could be extremely vital in the management of single-ventricle patients after stage 1 palliation, especially in the early postoperative period. Since derangements in PBF can influence perioperative and ICU outcomes for such patients, having an immediately available parameter at the bedside is important.

Among Group 3 patients (2-ventricle physiology with no intracardiac shunts), we found that VCO_2 correlated directly with Q_p . As our patients had steady minute ventilation and stable metabolic state prior to obtaining volumetric capnography, it was not surprising to note that VCO_2 was reflective of Q_p and by extension cardiac output.

More notably, this study demonstrates that, in stable physiologic state, VCO_2 closely correlates with Q_p and CO in patients with small to moderate intracardiac shunting and balanced $Q_p:Q_s$ ratio (group 2 median $Q_p:Q_s$ was 0.99). On the other hand, poor correlation between VCO_2 and cardiac output was noted in patients with significant shunts (ie, single ventricle patients). This could be explained by the fact that in single-ventricle patients, PBF is dependent on the balance between $Q_p:Q_s$ and in parallel with systemic blood flow and might not reflect cardiac output.

Our study has limitations. First, our study included a small number of patients, with stable $Q_p:Q_s$, which might explain the higher correlation between CO and VCO_2 in group 2 when compared to group 3. Second, the cardiac catheterization procedure was performed electively in clinically stable patients who lacked the hemodynamic variability seen in critically ill patients in the ICU. As a result, the magnitude of changes in VCO_2 in response to changes in Q_p and CO could not be well delineated. Third, $Q_p:Q_s$ was close to 1 in all groups which does not always occur in patients with intracardiac shunting. Fourth, all patients had relatively healthy lungs, which is not always the case in the pediatric cardiac intensive care unit. This latter point presents a concern as VCO_2 is affected by alveolar dead space, which may be a confounding factor when assessing changes in Q_p . However, rapid changes in alveolar dead space are not commonly seen in the pediatric cardiac ICU population. Finally, multiple factors, which can impact capnographic measurements, including minute ventilation and CO_2 production, which were kept relatively stable during the study period, are potentially variable in the ICU setting.

5 | CONCLUSIONS

This study explores the use of a noninvasive tool for the assessment of hemodynamic status in patients with complex cardiac physiology. Volume of CO_2 elimination (VCO_2) may be a surrogate marker of pulmonary blood flow, Q_p , in single-ventricle patients after stage 1 palliation and in patients with biventricular physiology with intracardiac shunting. Also, among pediatric patients with normal cardiac anatomy, VCO_2 may be a marker of cardiac output as $Q_p=Q_s$. VCO_2 may serve as a practical, noninvasive tool in the assessment of hemodynamic status in critically ill complex cardiac patients. This new understanding of the correlation between VCO_2 and pulmonary blood flow could allow early detection of hemodynamic changes and the immediate assessment of the impact of interventions among critically ill pediatric cardiac patients. Further studies are needed to establish its utility in the intensive care unit.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

CONFLICT OF INTEREST

Dr. Cheifetz received funding from Philips (advisor) and UptoDate (contributing author). Dr. Hornik received salary support for research from the National Center for Advancing Translational Sciences of the National Institutes of Health (UL1TR001117) and the U.S. government for his work in pediatric and neonatal clinical pharmacology (Government Contract HHSN267200700051C, PI: Benjamin under the Best Pharmaceuticals for Children Act). The remaining authors have disclosed that they do not have any potential conflicts of interest.

REFERENCES

1. Matthay MA. Invasive hemodynamic monitoring in critically ill patients. Clin Chest Med. 1983;4:233–249. [PubMed: 6342922]
2. Botte A, Leclerc F, Riou Y, et al. Evaluation of a noninvasive cardiac output monitor in mechanically ventilated children. Pediatr Crit Care Med. 2006;7:231–236. [PubMed: 16575346]

3. Nusmeier A, van der Hoeven JG, Lemson J. Cardiac output monitoring in pediatric patients. *Expert Rev Med Devices*. 2010;7:503–517. [PubMed: 20583887]
4. Vender JS, Franklin M. Hemodynamic assessment of the critically ill patient. *Int Anesthesiol Clin*. 2004;42:31–58. [PubMed: 14716196]
5. Al-Subu AM, Rehder KJ, Cheifetz IM, et al. Non invasive monitoring in mechanically ventilated pediatric patients. *Expert Rev Respir Med*. 2014;8:693–702. [PubMed: 25119483]
6. Deitch K, Miner J, Chudnofsky CR, et al. Does end tidal CO₂ monitoring during emergency department procedural sedation and analgesia with propofol decrease the incidence of hypoxic events? A randomized, controlled trial. *Ann Emerg Med*. 2010;55:258–264. [PubMed: 19783324]
7. Erasmus PD. The use of end-tidal carbon dioxide monitoring to confirm endotracheal tube placement in adult and paediatric intensive care units in Australia and New Zealand. *Anaesth Intensive Care*. 2004;32:672–675. [PubMed: 15535493]
8. McSwain SD, Hamel DS, Smith PB, et al. End-tidal and arterial carbon dioxide measurements correlate across all levels of physiologic dead space. *Respir Care*. 2010;55:288–293. [PubMed: 20196877]
9. Eckstein M, Hatch L, Malleck J, et al. End-tidal CO₂ as a predictor of survival in out-of-hospital cardiac arrest. *Prehosp Disaster Med*. 2011;26:148–150. [PubMed: 22107764]
10. Lewis LM, Stothert J, Standeven J, et al. Correlation of end-tidal CO₂ to cerebral perfusion during CPR. *Ann Emerg Med*. 1992;21:1131–1134. [PubMed: 1514728]
11. von Planta M, Weil MH, Gazmuri RJ. Noninvasive assessment of hemodynamics during resuscitation using capnography. *Schweiz Med Wochenschr*. 1989;119:371–375. [PubMed: 2497515]
12. Bhende MS. End-tidal carbon dioxide monitoring in pediatrics - clinical applications. *J Postgrad Med*. 2001;47:215–218. [PubMed: 11832630]
13. Cannon ML, Cornell J, Tripp-Hamel DS, et al. Tidal volumes for ventilated infants should be determined with a pneumotachometer placed at the endotracheal tube. *Am J Respir Crit Care Med*. 2000;162:2109–2112. [PubMed: 11112123]
14. Langan M. Continuous end-tidal carbon dioxide monitoring in pediatric intensive care units. *J Crit Care*. 2009;24:227–230. [PubMed: 19327292]
15. Hubble CL, Gentile MA, Tripp DS, et al. Deadspace to tidal volume ratio predicts successful extubation in infants and children. *Crit Care Med*. 2000;28:2034–2040. [PubMed: 10890660]
16. Feinstein JA, Benson DW, Dubin AM, et al. Hypoplastic left heart syndrome: current considerations and expectations. *J Am Coll Cardiol*. 2012;59:S1–S42. [PubMed: 22192720]
17. Cote CJ, Wax DF, Jennings MA, et al. Endtidal carbon dioxide monitoring in children with congenital heart disease during sedation for cardiac catheterization by nonanesthesiologists. *Pediatr Anesth*. 2007;17:661–666.
18. Young A, Marik PE, Sibole S, et al. Changes in end-tidal carbon dioxide and volumetric carbon dioxide as predictors of volume responsiveness in hemodynamically unstable patients. *J Cardiothorac Vasc Anesth*. 2013;27:681–684. [PubMed: 23182383]
19. Suarez-Sipmann F, Bohm SH, Tusman G. Volumetric capnography: the time has come. *Curr Opin Crit Care*. 2014;20:333–339. [PubMed: 24785676]
20. Tusman G, Groisman I, Maidana GA, et al. The sensitivity and specificity of pulmonary carbon dioxide elimination for noninvasive assessment of fluid responsiveness. *Anesth Analg*. 2016;122:1404–1411. [PubMed: 26505574]
21. Glatz AC, Rome JJ, Small AJ, et al. Systemic-to-pulmonary collateral flow, as measured by cardiac magnetic resonance imaging, is associated with acute post-Fontan clinical outcomes. *Circ Cardiovasc Imaging*. 2012;5:218–225. [PubMed: 22228054]
22. Smallwood CD, Walsh BK, Bechard LJ, et al. Carbon dioxide elimination and oxygen consumption in mechanically ventilated children. *Respir Care*. 2015;60:718–723. [PubMed: 25550526]
23. Mosing M, Kutter AP, Iff S, et al. The effects of cardiac output and pulmonary arterial hypertension on volumetric capnography derived-variables during normoxia and hypoxia. *J Clin Monit Comput*. 2015;29:187–196. [PubMed: 24908108]

24. Shakti D, McElhinney DB, Gauvreau K, et al. Pulmonary deadspace and postoperative outcomes in neonates undergoing stage 1 palliation operation for single ventricle heart disease. *Pediatr Crit Care Med*. 2014;15:728–734. [PubMed: 25072474]

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What is already known

- Assessment of pulmonary blood flow and cardiac output is critical in the postoperative management of patients with single-ventricle physiology or 2-ventricle physiology with intracardiac shunting.
- Capnography is a standard monitoring tool in mechanically ventilated pediatric patients.
- Endtidal CO₂ (EtCO₂) monitoring has been used during cardiopulmonary resuscitation as a marker of effectiveness of resuscitation and cardiac output.

What this article adds

- Volume of CO₂ elimination (VCO₂) may be a surrogate marker of Q_p in patients with single ventricle physiology.
- VCO₂ may be a surrogate marker of cardiac output in patients with normal cardiac anatomy.

FIGURE 1.
Consort diagram of the study

FIGURE 2.
Correlation between VCO₂ and Qp A, and VCO₂ and Cardiac output B, in Group 1

FIGURE 3.

Correlation between VCO₂ and Qp A, VCO₂ and Cardiac output B, in Group 2, and correlation between VCO₂ and Cardiac output C, in Group 3

TABLE 1

Demographic and variables for the study cohort

Parameter	Group 1: single ventricle (n = 8)	Group 2: 2 ventricles (n = 10)	Group3: no shunts (n = 17)
Age (y) ^a	0.33 (0.25–0.67)	0.49 (0.08–17)	7 (1.5–16)
Male ^b	6 (75%)	6 (60%)	11 (64%)
Weight (kg) ^a	6 (3.70–7.40)	4.7 (2.77–96.5)	33.3 (11.8–74)
BSA (m ²) ^a	0.3 (0.21–0.36)	0.26 (0.18–2.08)	1.06 (0.48–1.88)

^aMedian (range).

^bPercentage.

TABLE 2

Hemodynamic data during catheterization procedure

Parameter	Group 1: single ventricle median (range)	Group 2: 2 ventricles median (range)	Group3: no shunts median (range)	Overall median	95% CI	P value
HR	127 (98–144)	125 (70–158)	79 (42–139)	109	(81.5–123)	<.001
SvO ₂ (%)	57 (50–60)	69 (58–84)	73 (65–85)	71	(65–73)	<.001
PA saturation (%)	79 (70–88)	71 (55–88)	73 (65–84)	73	(71–78)	.013
SpO ₂ (%)	81 (70–86)	98 (88–100)	99 (94–100)	98	(95.3–99)	<.001
PA MAP (mm Hg)	20 (11–48)	25 (12–75)	15 (54–90)	17	(15–23)	.04
MAP (mm Hg)	55 (45–62)	57 (44–75)	68 (54–90)	61	(58–66)	<.001
CO (L/min)	1.06 (0.83–1.54)	1.27 (0.57–9.30)	4.41 (2.16–7.41)	2.8	(1.5–4.2)	<.001
CI (L/kg/m ²)	3.68 (3.04–5.29)	3.99 (2.99–5.02)	3.83 (2.66–8.95)	3.8	(3.4–4.4)	.064
Qp (L/min)	1.23 (0.83–2.33)	1.33 (0.48–2.04)	4.41 (2.16–7.41)	2.4	(0.9–1.8)	.504
Qp index (L/kg/m ²)	4.13 (3.67–6.72)	3.77 (2.39–7.48)	3.83 (2.66–8.95)	3.99	(3.7–4.3)	.885
Qp:Qs	1.11 (0.73–2.07) (0.98–1.24) ^a	0.99 (0.47–2.00) (0.81–1.18) ^a	1	1	(0.9–1.2)	.514

CI: Cardiac index, CO: Cardiac output, HR: Heart rate, IQR: Interquartile range, MAP: Mean arterial pressure, PA: Pulmonary artery, Qp: Pulmonary blood flow, Qp:Qs: Pulmonary to systemic blood flow ratio, SpO₂: Arterial oxygen saturation, SvO₂: Mixed venous oxygen saturation.

^aIQR.

TABLE 3

Capnography variables during the catheterization procedure

Parameter	Group 1: single ventricle median (range)	Group 2: 2 ventricles median (range)	Group3: no shunts median (range)	Overall median	95% CI	P value
RR	20 (15–28)	20 (7–36)	12 (7–18)	18	(13–20)	<.001
Vt (mL/kg)	11.2 (9.5–12.8)	11.3 (7.9–15.7)	9.8 (8.5–16.7)	10.3	(9.6–11.0)	.073
MV (L/min)	0.21 (0.17–0.28)	0.24 (0.11–0.49)	0.13 (0.07–0.28)	0.17	(0.14–0.21)	<.001
EtCO ₂	37 (35–46)	42 (34–51)	38 (34–45)	38	(36.4–39.7)	.416
VCO ₂ (mL/min)	27.1 (14.5–45.4)	36.4 (4.6–267.0)	113.5 (70.8–166.0)	71.4	(36.4–107)	<.001
VCO ₂ (mL/kg/min)	4.57 (2.0–8.2)	4.26 (1.7–7.7)	3.67 (2.1–9.1)	96.4	(88.0–114.3)	.318

EtCO₂: Endtidal carbon dioxide, IQR: Interquartile range, MV: Minute ventilation, RR: respiratory rate, VCO₂: Volume of CO₂ elimination, Vt: Tidal volume.