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Variability in Resting Oxygen Consumption Estimation in Adult Patients After Fontan Operation

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Abstract

The hemodynamics in patients after Fontan operation are commonly evaluated through cardiac catheterization, where accurate assessment depends on reliable estimation of the resting oxygen consumption (rVO_2). In clinical practice, rVO_2 is frequently estimated using predictive equations rather than measured directly. In this prospective observational study, we compared rVO_2 measured by indirect calorimetry with rVO_2 measured during pre-exercise rest phase on routine cardiopulmonary exercise testing (CPET) or estimated using commonly applied predictive equations. To illustrate the potential consequences of rVO_2 variability on hemodynamic calculations, pulmonary

vascular resistance (PVR) was modeled using a hypothetical patient scenario. Twenty-six clinically stable adult patients after Fontan operation were included (mean age 26.6 ± 5.8 years, 46% female, 73% extracardiac conduit, 42% open fenestration). Substantial variability in rVO_2 was observed depending on the method used. Measured rVO_2 by indirect calorimetry (246.2 ± 45.1 ml/min) differed significantly from that acquired by CPET ($n=22$; 355.5 ± 74.4 ml/min) or calculated using predictive equations ($n=26$ for all; LaFarge: 146.0 ± 31.9 ml/min; Dehmer: 229.3 ± 28.1 ml/min; Bergstra: 264.6 ± 38.8 ml/min; $p < 0.05$ for all comparisons against indirect calorimetry). The Dehmer and Bergstra equations showed the smallest systematic differences and strongest correlations with measured rVO_2 . Modeled PVR varied markedly and inversely with the rVO_2 estimation method used, reflecting its direct mathematical derivation from the rVO_2 values. Given the observed limitations of predictive equations and CPET-derived estimates in this population, direct measurement of rVO_2 by indirect calorimetry should be the preferred method for hemodynamic calculations in adult patients after Fontan operation.

Key words: Fontan operation, oxygen consumption, pulmonary vascular resistance, indirect calorimetry

Statements and Declarations

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Introduction

Accurate hemodynamic assessment is crucial in patients with complex congenital heart disease (CHD) and a functionally single ventricle following Fontan operation. The unique physiology of the Fontan circulation is commonly evaluated through invasive cardiac catheterization, where cardiac index (CI) and pulmonary vascular resistance (PVR) serve as key hemodynamic parameters guiding pharmacological therapy and possible interventions [1,2]. Combination of high PVR index and low CI represents an independent risk factor for Fontan circulatory failure [3].

Calculation of both CI and PVR requires an estimate of resting oxygen consumption (rVO_2). Estimation of rVO_2 is most often derived from predictive equations rather than direct measurement by indirect calorimetry, as a recent study showed that only 15% of congenital cardiac catheterization laboratories use direct rVO_2 measurements [4]. The use of predictive equations can potentially lead to misinterpretation of hemodynamic data and inappropriate therapeutic decisions [5,6]. Since predictive rVO_2 equations are mostly based on young, healthy and lean populations, the disagreement in patients after Fontan operation may be even more profound, as body composition alterations with lower muscle and higher fat mass are common in these patients [7]. However, data on the agreement of these estimates in patients after Fontan operation remain limited. Although a prior study evaluated measured versus calculated rVO_2 in a heterogeneous cohort of adults with CHD that included a subgroup of 24 patients after Fontan operation, no study to our knowledge has specifically focused on a homogeneous cohort of Fontan patients incorporating multiple estimation methods, including values obtained by routine cardiopulmonary exercise testing (CPET) [5]. This study aimed to compare rVO_2 measured by

indirect calorimetry with values measured during pre-exercise rest phase on CPET or calculated by commonly used predictive equations, and to illustrate the consequences of rVO_2 variability on PVR calculation among adults after Fontan operation.

Material and methods

This study was part of a prospective single-centre study on adult patients after Fontan operation (“GIN-Fontan Study”, Clinical Trial number NCT06731322). As part of the study protocol patients with known or clinically suspected protein-losing enteropathy were excluded. The study followed the Declaration of Helsinki and was approved by the Slovenian Medical Ethics Committee (number 0120-434/2024-2711-3). All participants provided written informed consent.

As a part of the study protocol all patients underwent indirect calorimetry using the Q-NRG calorimeter (COSMED, Rome, Italy) with a canopy hood, where exhaled gases of spontaneously breathing subjects are collected and diluted within the canopy. Using dilution flow, O_2 and CO_2 concentrations the system can accurately measure rVO_2 (ml/min). Indirect calorimetry measurements were performed in the morning, after overnight fasting, with the medications taken as prescribed. The patients were in a supine position, and great care was taken to avoid air leaks. The test lasted for 15 minutes; the best 5-minute average steady-state interval, automatically identified by the Q-NRG software as the interval with the lowest coefficient of variation for VO_2 and VCO_2 (<10%), was used for rVO_2 measurement.

Cardiopulmonary exercise testing was also performed in the morning on the same day, following the indirect calorimetry. The test was performed on an upright cycle ergometer (Schiller Cardiovit CS 200 Ergo-Spiro), but in a non-fasting state after a light meal. For the purpose of this

study, VO_2 (ml/min) measured by CPET at the end of the initial 3-minute unloaded rest phase before the beginning of exercise was analyzed. This comparison was intended to evaluate whether VO_2 routinely recorded during the pre-exercise rest phase of CPET could serve as an easily accessible and practical surrogate for rVO_2 measurement.

Calculation of rVO_2 was performed using the three most widely applied predictive equations, utilizing the DuBois equation for body surface area (BSA) assessment across all calculations:

LaFarge equation:

$$\text{rVO}_2 \text{ (ml/min)} = 138.1 - (K \times \ln(\text{age})) + (0.378 \times \text{heart rate}) \times \text{BSA}$$

Where $K = 17.04$ for women and $K = 11.49$ for men.

Dehmer equation:

$$\text{rVO}_2 \text{ (ml/min)} = 125 \times \text{BSA}$$

Bergstra equation:

$$\text{rVO}_2 \text{ (ml/min)} = 157.3 \times \text{BSA} + K - (\ln(\text{age}) \times 10.5) + 4.8$$

Where $K = 0$ for women and $K = 10$ for men.

To demonstrate the downstream consequences of rVO_2 variability on hemodynamic calculations, a hypothetical patient scenario was modeled using the standard Fick principle. Cardiac output (CO) and PVR were calculated as follows:

$$CO \text{ (L/min)} = \frac{rVO_2 \text{ (ml/min)}}{10 \times Hb \text{ (g/dL)} \times 1.34 \times (SaO_2 - SMCvO_2)}$$

$$PVR \text{ (WU)} = \frac{mPAP - mAP}{CO}$$

Individual measured or calculated rVO_2 values were used, while the following fixed baseline clinical parameters were assumed in all patients: mean pulmonary artery pressure (mPAP) = 16 mmHg, mean atrial pressure (mAP) = 10 mmHg, hemoglobin (Hb) = 140 g/L, arterial oxygen saturation (SaO_2) = 95%, and mixed venous oxygen saturation (SMCvO₂) = 65%.

Statistical analysis was performed using IBM SPSS Statistics (IBM Corp., Armonk, NY, USA) and GraphPad Prism version 10. Categorical variables are presented as frequencies and percentages, while continuous variables are presented as mean $\pm$ standard deviation (SD) or median (interquartile range [IQR]) based on distribution normality assessed using the Shapiro–Wilk test. Comparison between rVO_2 measured by indirect calorimetry and the three predictive equations was performed by repeated-measure analysis of variance (RM-ANOVA) followed by Bonferroni-adjusted pairwise comparisons. Because CPET measurements were available only in a subset of patients, analyses involving CPET were performed separately in the subgroup with complete CPET data using paired t-test. PVR comparison was performed by paired t-test, however as modeled PVR values were mathematically derived from rVO_2 under fixed assumptions, statistical comparisons of PVR were not interpreted as independent hypothesis tests. Agreement between indirect calorimetry and each alternative method was further assessed by Pearson correlation coefficients, intraclass correlation coefficient (ICC) using a two-way absolute agreement, single-measure ICC (ICC (2,1), and a Bland–Altman plot. Bland–Altman

analysis was performed by plotting the difference between estimated and measured rVO_2 against their mean. A two-sided p value of 0.05 was considered statistically significant.

Results

The study included 26 adult patients after Fontan operation. Baseline clinical characteristics of the included patients are presented in Table 1. Results of CPET are available for 22 patients, the test was not performed in 4 patients (physical disability in 3 patients, tachycardia before testing in 1 patient).

Table 1. Clinical characteristics of included adult patients after Fontan operation.

	Patients after Fontan operation (n=26)
Age (years)	26.6 ± 5.8
Sex (female)	12 (46%)
BMI (kg/m ²)	24.1 (20.2-27.8)
BSA (m ²)	1.8 ± 0.2
Peripheral O ₂ saturation (%)	91.8 ± 3.3
NYHA class	
NYHA class 1	4 (16%)
NYHA class 2	20 (77%)
NYHA class 3	2 (8%)
NYHA class 4	0

Anatomic diagnosis	
Tricuspid atresia	3 (11%)
Mitral atresia	3 (11%)
HLHS	2 (8%)
DORV	5 (19%)
DILV	5 (19%)
TGA/VSD	1 (4%)
Unbalanced AVSD	3 (11%)
Other	4 (15%)
Systemic ventricle	
Left	10 (38%)
Right	14 (54%)
Unknown	2 (8%)
Fontan type	
Intracardiac	7 (27%)
Extracardiac	19 (73%)
Age at Fontan operation (years)	6.4 ± 2.8
Open fenestration	11 (42%)
History of Fontan complications	
Arrhythmias	9 (35%)
Heart failure	4 (15%)
Thromboembolic	2 (8%)
Collaterals	10 (38%)

Pulmonary hypertension	1 (4%)
Protein-losing enteropathy	0
Plastic bronchitis	0
Pacemaker	3 (11%)
Medications	
Aspirin	18 (69%)
Anticoagulant therapy	7 (27%)
Beta-blockers	11 (42%)
MRA	9 (35%)
ACEi/ARBs	18 (69%)
ARNI	1 (4%)
SGLT2i	3 (11%)
Diuretics	2 (8%)
PAH specific therapy	1 (4%)
NT-proBNP (ng/L)	142.3 (85.5-280.6)
Albumin (g/L)	49.2 ± 3.2
Hemoglobin (g/L)	164.5 ± 19.1
pVO₂ (ml/kg/min) (n=22)	22.0 ± 5.3
ppVO₂ (%) (n= 22)	60.3 ± 11.2

131

132 ACEi/ARBs – Angiotensin-Converting Enzyme inhibitors /Angiotensin Receptor Blockers, ARNI – angiotensin receptor/neprilysin inhibitors,

133 AVSD – atrioventricular septal defect, BMI – body mass index, BSA – body surface area, DILV – double inlet left ventricle, DORV – double

134 outlet right ventricle, HLHS – hypoplastic left heart syndrome, MRA - mineralocorticoid receptor antagonists, NYHA – New York Heart

135 Association class, NT-proBNP - N-terminal pro-brain natriuretic peptide, PAH – pulmonary arterial hypertension, pVO₂ - peak oxygen
 136 consumption; ppVO₂ – percent of predicted pVO₂, SGLT2i – Sodium-glucose cotransporter 2 inhibitors, TGA/VSD – transposition of the great
 137 arteries with ventricular septal defect.

138
 139 Significant differences were observed between reference indirect calorimetry measurements and
 140 all alternative rVO₂ evaluation methods, with the largest absolute mean biases demonstrated by
 141 CPET and the LaFarge equation (Table 2, Figure 1). RM-ANOVA demonstrated a significant
 142 overall effect of measurement method on rVO₂ ($p < 0.001$, partial $\eta^2 = 0.896$). Bonferroni-
 143 adjusted pairwise comparisons revealed statistically significant differences between indirect
 144 calorimetry and all three predictive equations (LaFarge $p < 0.001$, Dehmer $p = 0.010$, Bergstra p
 145 $= 0.001$).

146 **Table 2.** Comparison of resting oxygen consumption (rVO₂) between different estimation
 147 methods and its consequences on calculation of pulmonary vascular resistance (PVR).

	Mean rVO ₂ (ml/min)	rVO ₂ mean difference vs measured (ml/min)	rVO ₂ relative difference vs measured (%)	95% Limits of agreement (ml/min)	Pearson r	ICC (95% CI)	Calculate d PVR (WU)
Indirect calorimetry	246.2 ± 45.1	/	/	/	/		1.4 ± 0.3

LaFarge equation	146.0 ± 31.9*	-100.3 ± 31.9	-40.4 ± 10.4	-162.8 to -37.8	0.71	0.156 (-0.043-0.492)	2.5 ± 0.7*
Dehmer equation	229.3 ± 28.1*	-16.9 ± 24.5	-5.6 ± 8.9	-65.0 to 31.2	0.88	0.720 (0.331-0.880)	1.5 ± 0.2*
Bergstra equation	264.6 ± 38.8*	18.3 ± 20.8	8.4 ± 9.2	-22.5 to 59.1	0.89	0.805 (0.320-0.930)	1.3 ± 0.2*
CPET	355.5 ± 74.4*	101.5 ± 57.1	41.1 ± 26.5	-10.4 to 213.3	0.64	0.240 (-0.096-0.603)	1.0 ± 0.3*

148 Mean absolute rVO_2 values ($\pm$ SD) are shown, mean difference was calculated as estimated rVO_2 minus measured rVO_2 , and relative difference
149 as (estimated rVO_2 – measured rVO_2) / measured $rVO_2 \times 100$. Asterisks denote statistical significance ($p < 0.05$) compared to rVO_2 measured by
150 indirect calorimetry (the reference method). Limits of agreement are derived from Bland–Altman analysis, r -value from Pearson correlation
151 analysis and ICC with 95% CI. Because calculated PVR is a deterministic transform of rVO_2 under fixed hemodynamic assumptions (see
152 Methods), the PVR asterisks reflect the same underlying rVO_2 comparison rather than an independently tested hemodynamic endpoint.

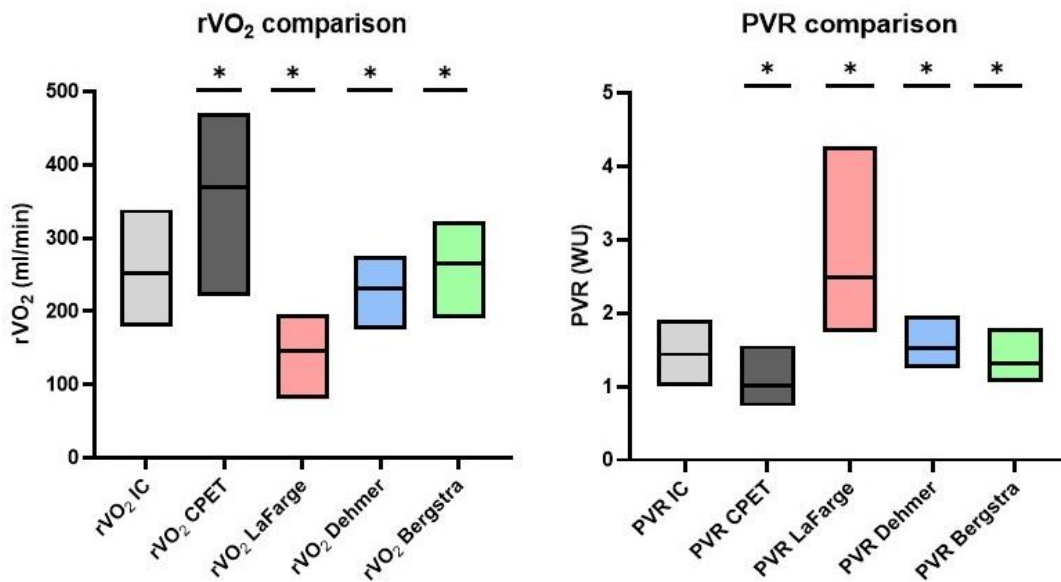


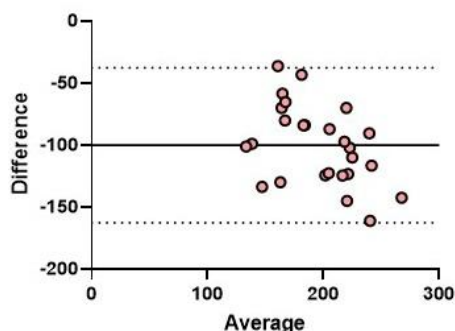
Fig. 1 Comparison of measured and calculated resting oxygen consumption (rVO₂) showing significant differences in obtained rVO₂ and pulmonary vascular resistance (PVR) values. Data are presented as floating bars representing minimum and maximum values, with horizontal lines indicating mean values. Asterisks denote statistical significance ($p<0.05$) for comparison between rVO₂ or PVR value by indirect calorimetry (IC) and by other methods. PVR values shown are a mathematical transform of the corresponding rVO₂ values under fixed hemodynamic assumptions, not an independently tested hemodynamic outcome

Pearson correlation coefficients demonstrated a significant positive association across all modalities; the strength of this association was strong for the Dehmer ($r=0.88$, $p<0.001$) and Bergstra ($r=0.89$, $p<0.001$) equations and moderate for LaFarge equation ($r=0.71$, $p<0.001$) and CPET ($r=0.64$, $p=0.001$). However, absolute agreement using ICC was deemed good for Bergstra equation (ICC 0.805, 95% CI 0.320-0.930), moderate for Dehmer equation (ICC 0.720,

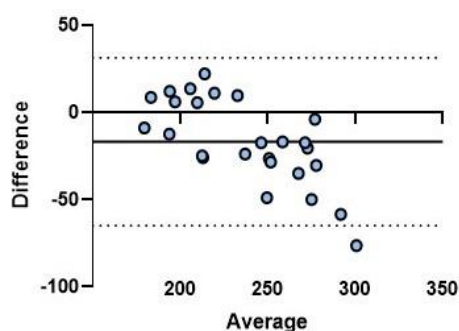
167 95% CI 0.331-0.880) and poor for LaFarge equation (ICC 0.156, 95% CI -0.043-0.492) and
 168 CPET derived $\dot{V}O_2$ (ICC 0.240, 95% CI -0.096-0.603).

169 Bland–Altman analysis indicated a relatively small bias for the Dehmer (mean bias -16.9 ml/min,
 170 limits of agreement -65.0 to 31.2 ml/min) and Bergstra equation (mean bias 18.3 ml/min, limits
 171 of agreement -22.5 to 59.1 ml/min), whereas substantial systematic differences were noted for
 172 CPET (mean bias 101.5 ml/min, limits of agreement -10.4 to 213.3 ml/min) and the LaFarge
 173 equation (mean bias -100.3 ml/min, limits of agreement -162.8 to -37.8 ml/min). However, at
 174 individual level the variability remained substantial in all tests indicating possible relevant errors
 175 (Figure 2).

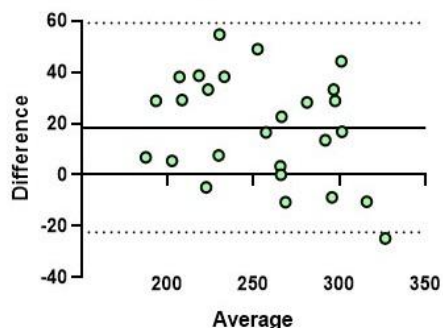
Difference vs. average: Bland-Altman of LaFarge



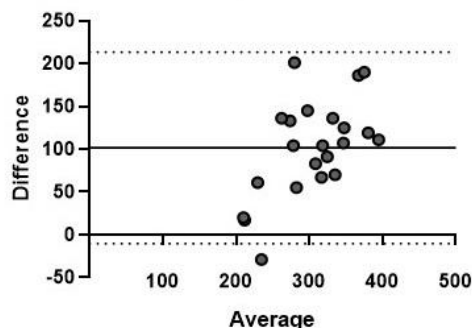
Difference vs. average: Bland-Altman of Dehmer



Difference vs. average: Bland-Altman of Bergstra



Difference vs. average: Bland-Altman of CPET



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Fig. 2 Bland-Altman plots showing agreement between resting oxygen consumption (rVO_2) measured by indirect calorimetry (IC) and rVO_2 measured by routine cardiopulmonary exercise testing (CPET, grey dots) or estimated rVO_2 using LaFarge (red dots), Dehmer (blue dots) and Bergstra (green dots) equation. Dots represent individual patients (n=22 for CPET, n=26 for all equations). Solid line represents mean bias, while dotted lines represent the 95% limits of agreement. All values are expressed in ml/min

Downstream modeled PVR values differed substantially depending on the rVO_2 estimation method (Table 2, Figure 1), illustrating the potential numerical consequences of using different rVO_2 estimates in Fick-based calculations.

Discussion

The results of our study demonstrate substantial variability in rVO_2 values depending on the method used in adult patients after Fontan operation. Measured rVO_2 by indirect calorimetry differs significantly from that measured during pre-exercise rest phase on routine CPET or calculated using commonly used predictive equations. Among the evaluated methods, the Dehmer and Bergstra equations showed the closest association and agreement with measured rVO_2 . The observed differences are not only methodological but may affect the interpretation of hemodynamic data.

Previous large studies in patients without CHD have similarly reported considerable variability and disagreement with calculation of rVO_2 . A large study in patients with heart failure showed that measured rVO_2 obtained using a metabolic cart was significantly different to calculated

values with LaFarge, Dehmer or Bergstra equations, with the largest difference observed with the Bergstra equation [8]. Furthermore, Narang et al. measured rVO_2 by Douglas bag technique in a large cohort of adult patients and also showed significant differences in comparison to the three equations with the largest difference using the LaFarge equation [6]. This study also included a group of patients with congenital heart disease (46 (9%) patients).

Studies in pediatric patients with CHD also demonstrated significant differences in measured and calculated rVO_2 values using the LaFarge and Krovetz-Goldbloom equations [9]. To our knowledge, only one previous study compared measured and calculated rVO_2 specifically in adults with CHD [5]. This study included a heterogeneous cohort of 84 patients with CHD, including 24 patients after Fontan operation. Estimation of rVO_2 with equations in the whole CHD cohort was associated with significant differences compared to measured values and led to clinically important changes in PVR and CI [5].

The mean measured rVO_2 in their subgroup of patients after Fontan operation was lower than in our cohort (121 ± 23 vs 246.2 ± 45.1 ml/min). The observed difference may be due to different patient characteristics, which are not specifically disclosed, and methodological differences. Their measurements were performed during cardiac catheterization with mild sedation, that is known to lower both oxygen consumption and metabolic rate [5]. They also used a different method of expired gas analysis, using the mouth-piece system. Furthermore, their results showed no difference in measured and calculated rVO_2 with Dehmer equation, while the LaFarge equation underestimated rVO_2 and Bergstra equation overestimated rVO_2 in patients after Fontan operation. In our cohort of clinically stable patients after Fontan operation, the Dehmer and Bergstra equations showed the closest agreement with measured rVO_2 , whereas the LaFarge

equation underestimated and CPET data overestimated rVO_2 . Despite the strong association of Dehmer and Bergstra equations with measured rVO_2 , the observed limits of agreement indicate that individual error may be clinically relevant despite acceptable associations. The lowest bias was observed with Dehmer equation, especially with lower rVO_2 values, which may partly explain why no significant difference between measured and calculated rVO_2 was observed with this equation in the previous study.

We also evaluated measured VO_2 during the pre-exercise rest phase of routine CPET as a potential surrogate of rVO_2 , but we obtained significantly higher values compared to indirect calorimetry. This difference can be methodological as patients were in a different nutritional state and body position. Fasting is related to lower rVO_2 values and the non-fasting state of patients at the time of CPET could explain the higher rVO_2 [10]. The influence of body position is variable, with some studies showing that energy expenditure and rVO_2 are higher in sitting than in supine position [11–14]. Also, in our study rVO_2 in CPET was measured after 3 minutes of unloaded rest phase before the actual exercise part of the test, which was probably not enough to exclude significant rise in rVO_2 due to stress anticipation, muscle activation, respiratory pattern changes and anxiety associated with CPET. In our cohort, values of a VO_2 obtained during the pre-exercise rest phase on CPET were systematically higher than rVO_2 measured by indirect calorimetry and therefore did not provide a reliable surrogate for standardized resting VO_2 measurement.

The analysis of PVR in the hypothetical patient scenario intended to illustrate the isolated consequences of rVO_2 variability on derived PVR and demonstrated substantial differences, highlighting the magnitude of potential error. This does not reflect patient-specific

hemodynamics due to the lack of actual patient catheterization data. Despite this, direct measurement of rVO_2 is rarely performed in routine clinical practice, due to the need for specialized equipment, technical expertise, and the additional time required to obtain accurate measurements [4].

The main limitation of our study is the single centre design with a modest sample size of stable patients after Fontan operation. Therefore, our findings cannot be extrapolated to patients with Fontan failure or complications, such as protein-losing enteropathy. The size limits the precision of agreement estimates and precludes additional subgroup analyses. Furthermore, the study addresses methodological variability in rVO_2 rather than direct clinical outcomes, especially as invasive hemodynamic data of patients was not available since we do not perform routine cardiac catheterizations in stable patients after Fontan operation. Beyond its hemodynamic implications, rVO_2 misestimation may also have consequences for resting energy expenditure (REE) based nutritional assessment and prescription, which is clinically relevant given the well-recognized nutritional vulnerability of patients after Fontan operation. Although rVO_2 is not directly equivalent to REE, both are derived from indirect calorimetry and could be affected by similar sources of error. Calculation and analysis of PVR was based on a hypothetical case example. Additional invasive assessment would have allowed evaluation of inter-patient variability; however our approach also illustrated the potential consequences of differing rVO_2 on PVR.

Conclusion

Our results highlight the variability with rVO_2 estimation in comparison to direct rVO_2 measurement with indirect calorimetry in adult patients after Fontan operation. Direct measurement of rVO_2 should be pursued for the most accurate hemodynamic evaluation, as it

provides a more reliable estimate than predictive equations or routinely obtained pre-exercise rest phase CPET values. Clinicians taking care of adult patients after Fontan operation should be aware of the observed differences between measured and calculated rVO_2 and its possible consequences on hemodynamic assessment with PVR calculation. When indirect calorimetry is unavailable, the Dehmer or Bergstra equations demonstrated the smallest differences in our cohort and may represent preferable alternatives for rVO_2 estimation.

Declarations

Ethics approval: The study was approved by Slovenian Medical Ethics Committee and was performed in accordance with the ethical standards of Declaration of Helsinki.

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