Shunt resistance is associated with clinically important outcomes after the Norwood operation

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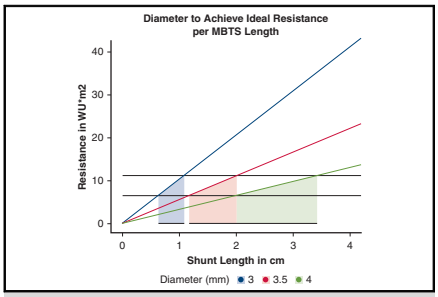
ABSTRACT

Background: In single-ventricle physiology, focus on pulmonary vascular resistance neglects the resistance in the conduit supplying the pulmonary inflow.

Methods: Conduit length and diameter, which can approximate conduit resistance, are available in the public dataset of Single Ventricle Reconstruction (SVR) trial. Conduit resistance was then calculated for SVR trial participants and the relationship with clinically important variables (death or transplant at 1 year, pulmonary artery size at second-stage palliation, pulmonary-to-systemic blood flow ratio, and supplemental oxygen requirement) was explored. To validate this calculated resistance, calculated resistance was compared with catheterization measurements at a single institution (not included in the SVR trial).

Results: In the institutional dataset, calculated and measured resistances had an intraclass correlation of 0.78 for modified Blalock–Taussig shunts (MBTS). Within the SVR trial, transplant-free survivors had a lower MBTS resistance (median, 8.3 Woods Units [WU]. interquartile range [IQR], 6.5-11.1 WU) than patients who died or required transplantation (median, 13.0 WU; IQR, 9.4-16.6 WU, $P = .0001$). When we controlled for left pulmonary artery diameter after the Norwood procedure in the SVR trial, for each unit increase in MBTS resistance, the left pulmonary artery diameter at stage II decreased (-0.006 ± 0.002 cm, $P = .005$). When we controlled for pulmonary vascular resistance, greater MBTS resistance was associated with a decrease in log pulmonary-to-systemic blood flow ratio (-0.04 ± 0.015, $P = .0048$) in the SVR trial. Patients in the SVR trial requiring supplemental oxygen on admission for stage II palliation had greater MBTS resistance (median. 11.1 WU; IQR, 6.6-16.6 WU) than patients not requiring oxygen (median 8.3, WU; IQR, 6.5-11.1 WU, $P = .015$).

Conclusions: Conduit resistance is associated with important clinical outcomes after Norwood; however, further studies are required to guide conduit resistance optimization. (JTCVS Open 2022;9:206-14)



Length required for ideal resistance at each MBTS diameter.

CENTRAL MESSAGE

The resistance across a shunt after a Norwood procedure is associated with transplant-free survival, pulmonary artery size at second stage palliation, QpQs, and supplemental oxygen requirement.

PERSPECTIVE

Shunt resistance is a potentially modifiable variable in patient outcomes after a Norwood procedure. Enhancing understanding of the relationship between shunt resistance and clinically significant outcomes could improve single ventricle palliation results, in addition to helping guide post-operative management by informing potential modes of failure.

See Commentary on page 215.

The development of the pulmonary vascular bed is of paramount importance to the successful palliation of single-ventricle physiology, indicated by low pulmonary vascular resistance (PVR). The resistance through the shunt supplying pulmonary inflow has not been considered clinically in relation to transplant-free survival, yet its importance is likely tantamount to that of the PVR in the early phases of vascular development.

Although the importance of using a shunt to control pulmonary blood flow in a Norwood procedure is well-recognized, published models of survival after the Norwood procedure from the Pediatric Heart Network Single Ventricle Reconstruction (SVR) trial do not include a variable that directly represents shunt resistance. Conduit resistance can be approximated from the length and width, given some

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Abbreviations and Acronyms

CI = confidence interval
ICC = intraclass correlation
IQR = interquartile range
LPA = left pulmonary artery
MBTS = modified Blalock–Taussig shunt
PA = pulmonary artery
PVR = pulmonary vascular resistance
QpQs = pulmonary-to-systemic blood flow ratio
RPA = right pulmonary artery
RVPA = right ventricle to pulmonary artery conduit
SVR = Single Ventricle Reconstruction trial
WU = Woods units

assumptions with respect to viscosity, and measured using the trans-shunt gradient. We present an argument for considering a shunt resistance variable using SVR trial datasets and a dataset using patients from our institution (representing patients who were not included in the SVR trial).

METHODS

Datasets

Two datasets were used to evaluate the hypothesis that shunt resistance following the Norwood operation was associated with death or transplant. The SVR trial design and methodology were previously reported.[1] Patients were assigned to modified Blalock-Taussig shunts (MBTS) or right ventricle to pulmonary artery conduits (RVPA) cohorts based on the source of pulmonary inflow at the conclusion of the Norwood operation. The cohorts were considered separately, as the measured resistance across each shunt type is calculated differently. The shunt type at the conclusion of the operation was selected instead of the randomized shunt to evaluate the actual shunt resistance on the patient's outcomes.

Patients from Texas Children's Hospital with hypoplastic left heart syndrome who had a Norwood operation from January 2011 through July 2018 were included if they had a cardiac catheterization performed within the usual course of their care in the first interstage period, with measurements needed for calculated and measured shunt resistance. The institutional dataset contained granular catheterization data, which allowed both the measured and calculated resistances to be derived for each patient.

Calculated Resistance

The formula used to calculate resistance in Woods units (WU) was as follows:

$$\text{Calculated Resistance} = \frac{8\mu L}{\pi r^4}$$

Where μ is the viscosity of whole blood in mPa*s, L is the length of the shunt in millimeters, and r is the radius of the shunt in millimeters.[2] For the "Reliability of Measures" section of the manuscript, calculated resistance was multiplied by patient's body surface area to allow calculated and measured resistance to be expressed in WU meters-squared ($WU*m^2$). For the remainder of the manuscript, body surface area was not available in the SVR trial dataset; therefore, calculated resistance is expressed in Woods units. Shunt length and diameter are provided in the SVR trial public use datasets for 402 (73%) patients. Assuming the distributions of patient temperatures and hematocrits over the duration of shunted physiology would not vary significantly between patients, and pulmonary blood flow was constant, viscosity was approximated at 3 relative units, the asymptote of hemoviscosity as tube radius exceeds 1.5 mm, based on the work by Haynes.[3]

Measured Resistance

Due to the limitations in calculating resistance, resistance was measured in the patients from the institutional dataset. Measured resistance was obtained using the hydraulic equivalent of Ohm's law[4]:

$$\text{Measured Resistance} = \frac{\Delta P}{Q_p}$$

Where ΔP is the pressure gradient across the shunt and Q_p is the pulmonary blood flow indexed to body surface area. For patients with MBTS, ΔP is the mean pulmonary artery (PA) pressure or mean pulmonary venous wedge pressure subtracted from the mean ascending aortic pressure, taken under the same conditions. The most proximal mean branch PA pressure was used and the pressures in both branch PA were averaged, where available. If branch PA pressures were not available, mean pulmonary venous wedge pressure was used and averaged across all pulmonary veins where data were available.[5,6] For patients with RVPA, ΔP is the systolic PA pressure or systolic pulmonary venous wedge pressure subtracted from the systolic RV pressure.

Reliability of Measurements

Shunt length was measured in both anteroposterior (right anterior oblique 0, cranial 0) and lateral (left anterior oblique 90, caudal 0) projections or in the next closest projections. Diameters were measured at multiple points along the shunt. The maximum and minimum diameters were recorded. In some patients, the shunt could not be clearly identified in one projection; therefore, all measurements were taken from the anteroposterior projection for patients with MBTS and lateral projection for patients with RVPA. In patients with both sets of measurements, the intraclass correlation (ICC)[7] was calculated to ensure validity of measurements in both projections. Conduit measurements were performed by a single blinded reviewer (Z.A.S.) with a random sample, comprising 15% of the total sample, measured by a second blinded reviewer (A.M.Q.) with ICC performed to confirm reproducibility of measurements.

Resistance in the institutional dataset was calculated using the same formula and assumptions as the patients in the SVR trial. Minimum shunt diameter was used to calculate the shunt radius as the smallest diameter is the primary driver of resistance. Given that patients may develop focal stenoses and these areas are not representative of the total shunt resistance, patients with a percent diameter change of 30% or greater, calculated as

$$\text{Percent Diameter Change} = \frac{(\text{Maximum Shunt Diameter} - \text{Minimum Shunt Diameter})}{\text{Maximum Shunt Diameter}}$$

Were not included (n = 8; MBTS = 3, RVPA = 5). The cut point of 30% was chosen due to the shift in the relationship of calculated resistance to diameter from quasilinear to exponential at this point.

Correlation of Measured and Calculated Conduit Resistance: Institutional Dataset

Using the same cardiac catheterization, the "gold standard" measured shunt resistance was obtained. ICC was calculated to determine the agreement between calculated and measured shunt resistance. Without agreement between measured and calculated RVPA resistance, only patients with MBTS were used in further analysis.

Resistance and Mortality: SVR Trial Dataset

The Wilcoxon rank-sum test was used to compare calculated shunt resistance between patients who died or had a transplant by 1 year. A

Cox proportional hazards model was developed to assess the association of transplant-free survival with shunt resistance. All model assumptions held.

Although mortality is likely associated with extremes of high and low resistances, the low range of resistance was not realized in the SVR data. Therefore, the interquartile range (IQR) of resistance for patients who survived to 1 year without transplant was used as the "ideal" range of resistance. The range of lengths that result in an expected shunt resistance in the ideal range was calculated for 3-mm, 3.5-mm, and 4-mm diameter MBTS.

Resistance and Pulmonary Artery Size: SVR Trial Dataset

A linear model assessed the association of MBTS resistance and PA size at second stage palliation, controlling for the size at the Norwood procedure, as assessed by echocardiogram at both time points. All assumptions for the linear model were met.

Resistance and Pulmonary-to-Systemic Blood Flow (QpQs): SVR Trial Dataset

A linear model assessed the association of MBTS resistance and QpQs by cardiac catheterization performed before stage II. Pulmonary blood flow is mediated by both MBTS resistance and PVR; therefore, PVR was included in the model. An interaction term between MBTS resistance and PVR was identified and included in the model. QpQs required log transformation to establish normality and homoscedascity of residuals.

Resistance and Supplemental Oxygen: SVR Trial Dataset

A Wilcoxon rank-sum test was used to compare MBTS resistance between patients known to require oxygen and patients not requiring oxygen on admission for stage II palliation. A logistic regression model assessed the association between supplemental oxygen requirement at stage II palliation and shunt resistance. All model assumptions held.

Effect of Hematocrit on Resistance: Theoretical

Given the assumptions around blood viscosity, the relationship between hematocrit and resistance was explored theoretically. A modified Krieger model[8] was used with assumed plasma viscocity of 1.23 cP, critical hematocrit of 99%, and Krieger exponent of 1.66. Shunt resistance was then calculated for a range of hematocrits from 21% to 51% for 3.0-mm, 3.5-mm, and 4.0-mm MBTS at a fixed length of 1.8 cm (median length). To simplify calculations, it was assumed that each change in hematocrit was not accompanied by a change in cardiac output.

This study was approved by the Baylor College of Medicine institutional review board with waiver of informed consent (H-46306; approved September 2, 2020). Statistical analysis was performed in R, version 3.5.2 (R Foundation for Statistical Computing) with graphics made in the ggplot2 package[9] and tables in the finalfit package.[10]

RESULTS

Correlation of Measured and Calculated Conduit Resistance: Institutional Dataset

In total 12 patients with MBTS and 14 patients with RVPA conduits had cardiac catheterizations performed between the Norwood procedure and second stage palliation. All 12 patients with MBTS had 3.5-mm shunts. Patients with RVPA conduits had either 5 mm (n = 3, 21%) or 6-mm (n = 11, 79%) conduits. The ICC between reviewers was excellent for conduit length (ICC rho = 0.987, $P < .001$), maximum diameter (ICC rho = 0.969, $P = .0001$), and minimum diameter (ICC rho = 0.75, $P = .015$). The ICC between anteroposterior and lateral projections were 0.85 ($P < .0001$) for minimum diameter and 0.97 ($P < .0001$) for maximum diameter. Patients with MBTS had an ICC between calculated and measured conduit resistance of 0.78 (95% confidence interval [CI], 0.42-0.93, $P = .0010$, Figure E1). There was no ICC between calculated and measured resistances across RVPA conduits (ICC, 0.0; 95% CI, 0.11-0.20, $P = .55$, Figure E2).

Resistance and Mortality–SVR Trial Dataset

Patient characteristics of the SVR trial have been published elsewhere,[11] with baseline characteristics of the 234 included patients presented in Table E1. MBTS length was similar for MBTS with 3-mm diameters (median 1.8 cm; IQR, 1.5-2.0), 3.5-mm diameters (median, 2.0 cm; IQR, 1.5-2.9), and 4-mm diameters (median, 1.8 cm; IQR, 1.5-2.3, Kruskal–Wallis $P = .21$). Median MBTS resistance was 9.8 WU (IQR, 6.6-13.8). Patients with MBTS who survived without transplant had a lower calculated shunt resistance (median, 8.3 WU; IQR, 6.5-11.1) than patients who died or required transplantation (median, 13.0 WU; IQR, 9.4-16.6, $P = .0001$). The difference was largely driven by the 3.5-mm subgroup (Table 1). "Cardiovascular," "vascular," or "airway" was listed as cause of death for 35 of 85 (41%) patients. These patients had a greater MBTS resistance (median, 13.6 WU; IQR, 11.1-16.6) compared with patients with all other causes of death (median, 10.5 WU; IQR, 7.74-13.8, $P = .034$). When we controlled for MBTS diameter, greater MBTS resistance was associated with decreased transplant-free survival (hazard ratio, 1.05; 95% CI, 1.01-1.08, $P = .01$). Table 2 shows the range of lengths for each MBTS diameter that would achieve IQR of shunt resistance for patients who survived without transplant (Figure 1).

Resistance and Pulmonary Artery Size: SVR Trial Dataset

Mean left pulmonary artery (LPA) diameter was 0.39 ± 0.09 cm at Norwood and 0.44 ± 0.12 cm at stage II palliation. When we controlled for LPA diameter after the Norwood procedure, for each WU increase in MBTS resistance, the LPA diameter at stage II decreased by 0.006 centimeters (Table E2). Figure 2 demonstrates the expected LPA diameter at stage II by MBTS resistance at the median LPA size at Norwood. Median right pulmonary artery (RPA) diameter was 0.40 cm (IQR, 0.34-0.48) at Norwood and 0.46 cm (IQR, 0.40-0.50) at stage II palliation. There was no association between MBTS resistance and RPA diameter at stage II.

Resistance and QpQs: SVR Trial Dataset

QpQs and PVR were available on pre-stage II catheterization in 116 (43%) patients. Median QpQs was 1.1

TABLE 1. Resistance by MBTS diameter and outcome

MBTS diameter	Survived	Death/transplant
3 mm, n; median (IQR)	11; 16.4 (15.4-20.5)	6; 19.5 (16.9-20.5)
3.5 mm, n; median (IQR)	97; 9.4 (7.7-13.8)	65; 11.1 (9.4-16.6)
4 mm, n; median (IQR)	42; 5.8 (4.9-6.5)	13; 6.5 (4.5-8.1)

MBTS, Modified Blalock-Taussig shunt; *IQR*, interquartile range.

(IQR, 0.8-1.5) and median PVR was 2.0 WU (IQR, 1.3-2.3). When we controlled for PVR, greater MBTS resistance was associated with a decrease in log QpQs (–0.04 ± 0.015, $P = .0048$); however, as PVR increases, the effect of MBTS resistance on log QpQs decreases (0.01 ± 0.006, $P = .029$, Table 3). Figure 3 shows the expected QpQs by MBTS resistance at given levels of PVR.

Resistance and Supplemental Oxygen: SVR Trial Dataset

Supplemental oxygen requirement at Norwood discharge and at second stage palliation was known in 132 patients, with 2 (2%) requiring oxygen at discharge but not on admission, 25 (19%) requiring oxygen on admission but did not at discharge, and 14 (11%) requiring supplemental oxygen at both time points. Patients known to require supplemental oxygen on admission for stage II palliation had greater MBTS resistance (median, 11.1 WU; IQR, 6.6-16.6) than patients not known to require supplemental oxygen on admission for stage II palliation (median, 8.3 WU; IQR; 6.5-11.1, $P = .015$). When we controlled for oxygen requirement at Norwood discharge, for each additional WU increase in MBTS resistance, the odds of requiring oxygen at stage II admission increased at least 2% (odds ratio, 1.1; 95% CI, 1.02-1.19, $P = .011$).

Effect of Resistance on Hematocrit: Theoretical

As hematocrit increases, viscosity increases, resulting in an increase in shunt resistance. Viscosity is estimated at 1.83 cP at a hematocrit of 21% and 4.09 cP at a hematocrit of 51%. The difference in resistance between each shunt diameter increases as hematocrit increases (Figure 4).

DISCUSSION

This study demonstrates the importance of shunt resistance following the Norwood procedure using a large sample of patients. Greater calculated shunt resistance was associated with increased mortality or transplant, smaller

TABLE 2. MBTS length by diameter for ideal MBTS resistance

	3-mm diameter	3.5-mm diameter	4-mm diameter
Minimum length, cm	0.63	1.18	2.00
Maximum length, cm	1.08	2.01	3.42

MBTS, Modified Blalock-Taussig shunt.

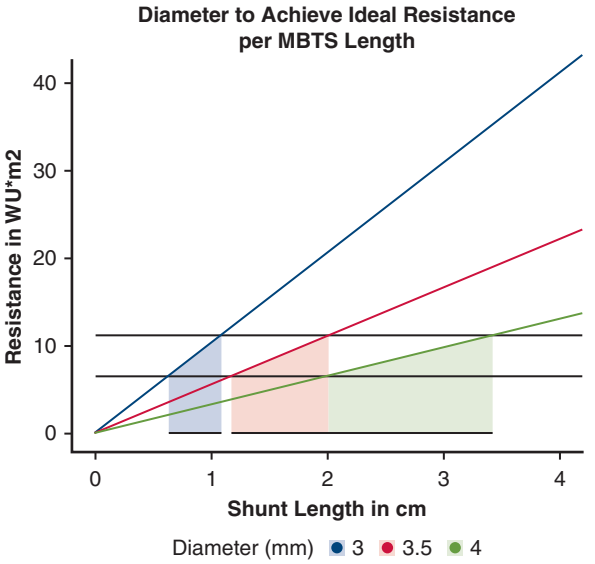

FIGURE 1. Ranges of modified Blalock–Taussig shunt (*MBTS*) length for each diameter to achieve MBTS resistance in the interquartile range of patients who survived to 1 year without death or transplant.

left pulmonary arteries, lower QpQs, and increased need for supplemental oxygen (Figure 5). While shunt resistance is not the only variable influencing with these outcomes, the association between each outcome and shunt resistance warrants further research into shunt resistance.

The concepts of resistance modeled in this paper are additive to those published by Migliavacca and colleagues,[12]

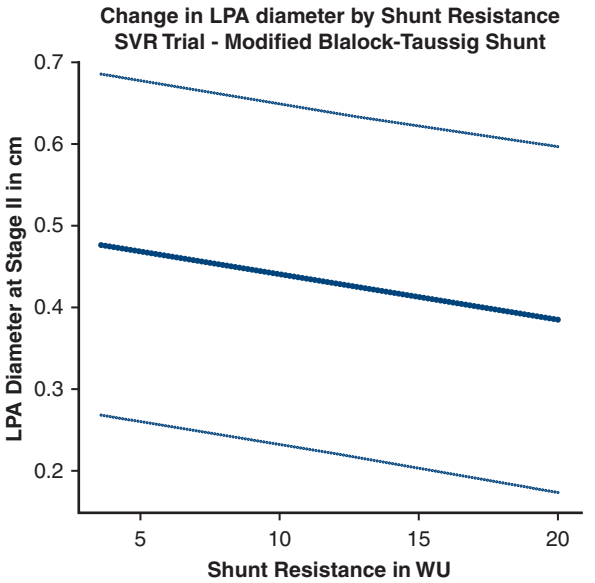

FIGURE 2. Expected left pulmonary artery diameter at second-stage palliation in centimeters by modified Blalock–Taussig shunt resistance for the median left pulmonary artery diameter at Norwood in centimeters. *Dotted lines* represent 95% confidence intervals. *LPA*, Left pulmonary artery; *WU*, Wood units; *SVR*, Single Ventricle Reconstruction.

TABLE 3. Linear regression model for log QpQs

Variable	Estimate ± SE	*P* value
Intercept	0.72 ± 0.182	.0001
MBTS resistance, WU	−0.04 ± 0.015	.0048
Pulmonary vascular resistance, WU	−0.21 ± 0.079	.0082
MBTS × pulmonary vascular resistance interaction	0.01 ± 0.006	.029

QpQs, Pulmonary-to-systemic blood flow ratio; *SE*, standard error, *MBTS*, modified Blalock-Taussig shunt; *WU*, Wood units.

who primarily focused on the effects of the shunt diameter on cardiac index, ratio of pulmonary and systemic blood flow, and oxygen balance using a mathematical model based on 28 patients with MBTS. They concluded that a shunt diameter of 3.5-mm optimized these parameters in the postoperative period for their average patient but would be insufficient during exercise.

While we do not have a point in our dataset where lower shunt resistance was associated with greater mortality, we would hypothesize that this point likely exists as MBTS without resistance would have increased diastolic runoff and coronary steal. Migliavacca and colleagues[12] share this hypothesis noting as modeled shunt diameter increases (resistance decreases) initially systemic oxygen delivery increases, but begins to decrease with continued modeled shunt diameters increases. It is likely the concern for overcirculation has led to surgeons avoiding large-diameter, short shunts that would result in a low shunt resistance. Furthermore, the risk of overcirculation represents an interplay between the cardiac output and systemic vascular resistance in addition to the shunt resistance. A lower shunt resistance would be better tolerated in patients with lower systemic vascular resistance and decrease afterload on a systemic right ventricle. However, high systemic vascular resistance would necessitate greater shunt resistance. Concordance between systemic and shunt resistance would therefore improve survival, with discordance increasing mortality. In addition, if cardiac output is low, greater shunt resistance would be deleterious. Shunt resistance is, in essence, not a parameter that should be considered in isolation, but is still important to consider in optimizing post-Norwood outcomes.

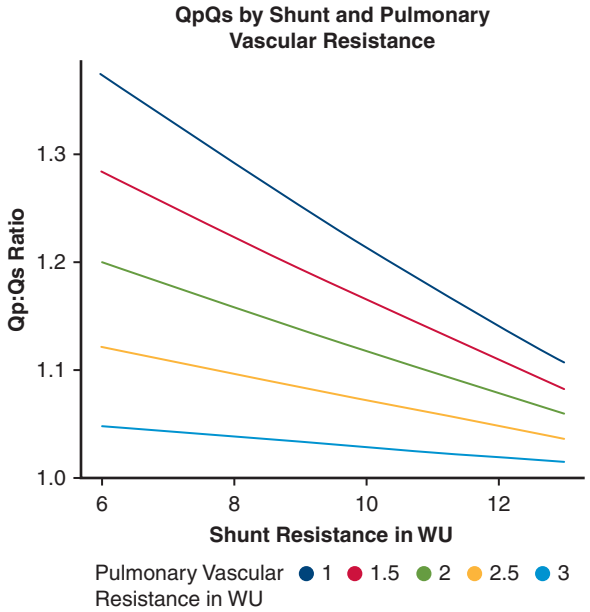

FIGURE 3. Expected QpQs by modified Blalock–Taussig shunt resistance for given levels of pulmonary vascular resistance as measured by cardiac catheterization before second-stage palliation. QpQs was calculated on a log scale with y-axis transformed to standard units for easier interpretation. *QpQs*, Pulmonary-to-systemic blood flow ratio; *WU*, Wood units.

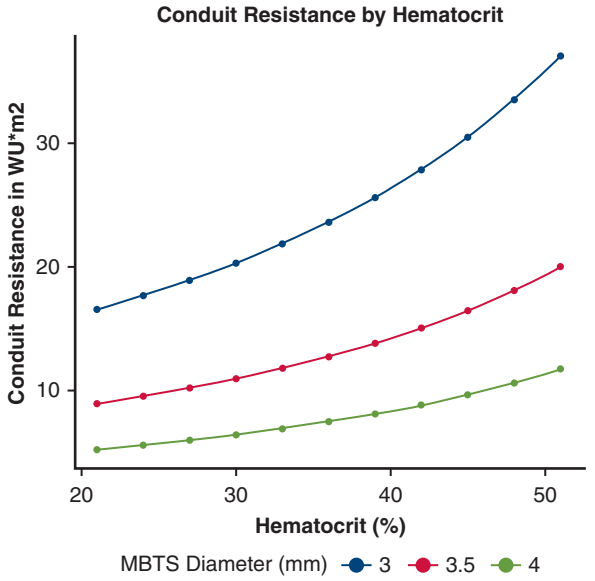

FIGURE 4. Theoretical shunt resistance by hematocrit at a fixed conduit length for 3.0-mm (*red*), 3.5-mm (*blue*), and 4.0-mm (*gold*) diameter shunts. Resistance increases as hematocrit increases, but at varying rates by shunt diameter. *MBTS*, Modified Blalock-Taussig shunt; *WU*m*^{2}, Woods Units meters-squared.

The application of shunt resistance lies in the modifiable variables of shunt length and diameter. The shunt length is effectively fixed by vascular orientation and operative approach, with minimal adjustment available based on shunt origin and insertion sites. These data suggest approximate ranges of lengths in which each diameter is "ideal"; however, these should be interpreted with caution, as they are based on the IQR of MBTS resistance in the subgroup of MBTS survivors in the SVR trial dataset. Ideal MBTS resistance likely changes during the first interstage period. Instead of using these ranges as dogma, they should prepare clinicians for potential problems in the first interstage. A conduit that is longer than the suggested range for a

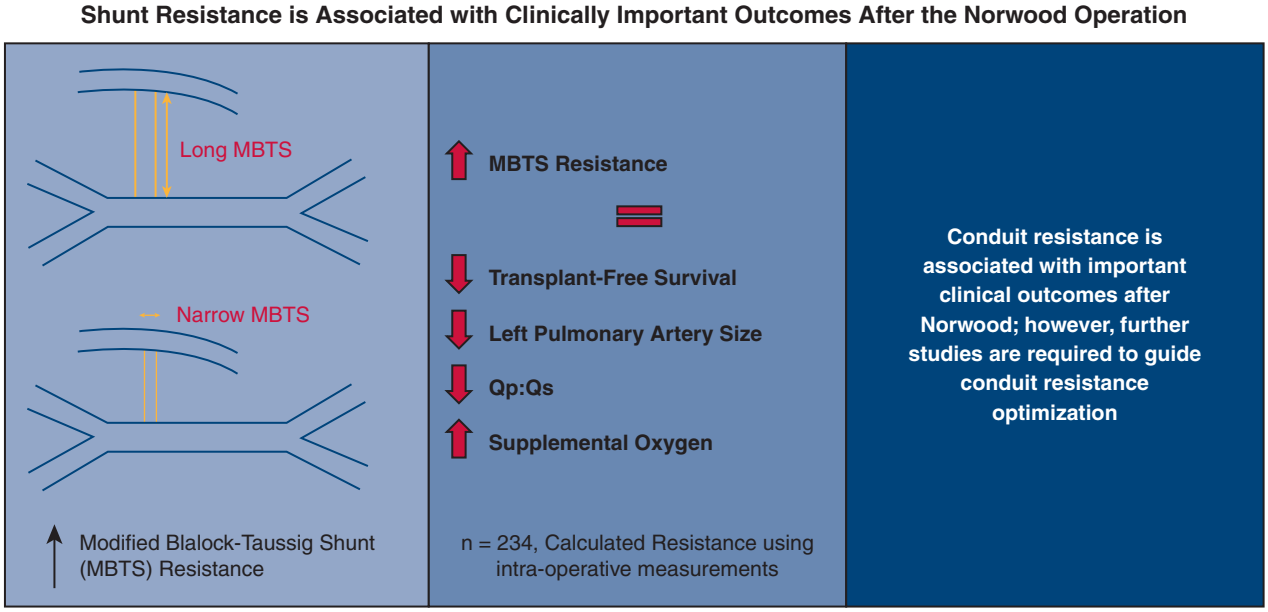

FIGURE 5. Description of the methods, results, and implications of the study. The study comprised 2 datasets, an institutional dataset to validate the use of calculated shunt resistance and the Single Ventricle Reconstruction trial, which evaluated the association between calculated shunt resistance and transplant-free survival, pulmonary artery growth, QpQs, and need for supplemental oxygen. *QpQs*, Pulmonary-to-systemic blood flow ratio; *MBTS*, modified Blalock-Taussig shunt.

diameter is more likely to be undercirculated, whereas a shorter conduit is more likely to be overcirculated. Additional studies, including where low MBTS resistance becomes hazardous, should be accumulated before directly guiding practice.

An additional modifiable variable is the hematocrit. While shunt resistance was relatively insensitive for 3.5 mm and 4.0 mm shunts in the usual range of hematocrits (35%-45%), 3.0-mm shunts observed a more rapid increase in shunt resistances as hematocrit increased within this range. This relationship should be further explored in vitro to confirm these theoretical findings and develop a better understanding of the hematocrit to shunt resistance relationship.

An unexpected finding in this study was the association between MBTS resistance and the growth of the LPA but not the RPA. This likely has multiple explanations, including compression from the neoaorta acting as an additional resistor where the LPA courses posteriorly. Furthermore, it has been suggested that MBTS is associated with risk of LPA stenosis when inserted onto the RPA following the Norwood, potentially due to the flow dynamics associated with the angle of insertion.[13-15]

Limitations

The assumptions for whole blood viscosity were necessary as other parameters, such as longitudinal hematocrit and temperature, were not available in the SVR Trial dataset. The assumptions of equivalent distributions of hematocrit and temperatures are likely true; however, the temperature used in the model[3] was 38 °C, unlikely to be the measure of central tendency for any interstage patient. There were not enough data in either the SVR trial or institutional datasets to identify association of MBTS origin and the resistance across the shunt.

The confirmatory model of measured shunt resistance was limited by patient volume and outcomes. Interstage cardiac catheterizations have become the exception, at our institution, limiting the number of available patients. Furthermore, the correlation of measured and calculated shunt resistance was necessary as there were not enough events in the Institutional dataset to directly associate measured shunt resistance with patient outcomes. The use of minimum shunt diameter does not precisely calculate the resistance across the shunt, but was used as an approximate to simplify our model. This minimum diameter was measured on catheterization images rather than labeled shunt diameter, as was provided in the SVR trial dataset. The shunt lengths were also measured via catheterization, as length was not documented in the operative note. Finally, the hydraulic equivalent of Ohm's law is likely oversimplified for the dynamics of a conduit arising from the right ventricle but provides a reasonable approximation for a shunt connecting 2 arteries, leading to close approximation

for MBTS but not RVPA. The analysis from the portions of the analysis using SVR trial data were unable to account for patient body surface area due to missing data; however, controlling for body surface area would be an important measure to include in further research. The outcomes of PA size, QpQs, and supplemental oxygen requirement at second stage palliation are dependent on patient survival to second stage palliation and may be subjected to survivorship bias.

CONCLUSIONS

Shunt resistance is an important concept during the Norwood operation due to its association with transplant-free survival, development of the PAs, QpQs, and supplemental oxygen requirement. Additional research should focus on optimal resistance through each shunt to maintain a balanced circulation and promote development of the pulmonary vascular bed throughout the entire first interstage.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Key Words: congenital, Norwood operation, shunt resistance, modified Blalock–Taussig shunt, pulmonary arteries

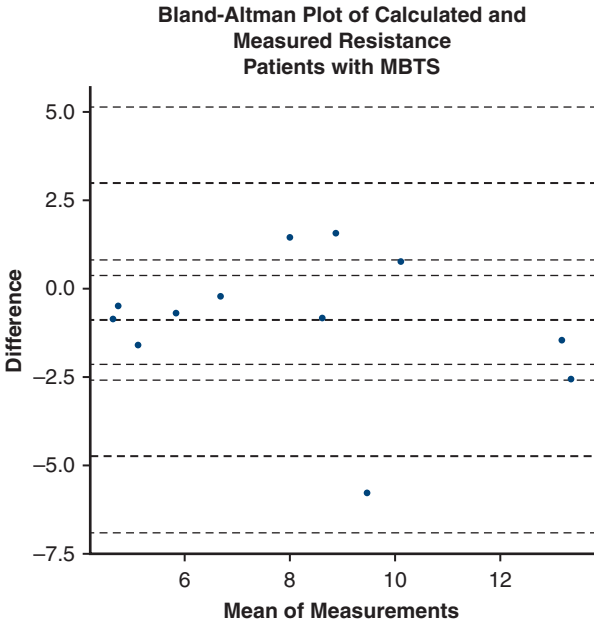

FIGURE E1. Bland–Altman plot showing strong intraclass correlation of calculated and measured shunt resistance in patients with MBTS. *MBTS*, Modified Blalock–Taussig shunt.

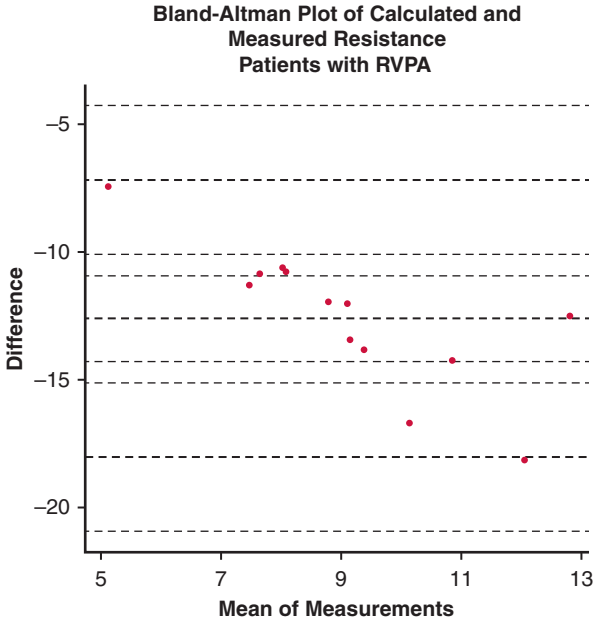

FIGURE E2. Bland–Altman plot showing no intraclass correlation of calculated and measured conduit resistance in patients with RVPA. *RVPA*, Right ventricle to pulmonary artery conduit.

TABLE E1. Baseline patient characteristics

Variable	MBTS (n = 234)
Norwood age, d, median (IQR)	6 (4-8)
Male sex, n/known (%)	149/234 (64%)
Birthweight, g, median (IQR)	3183 (2800-3500)
Gestational age, wk, median (IQR)	38 (38-39)
Hispanic ethnicity, n/known (%)	36/226 (16%)
Intubated pre-Norwood, n/known (%)	102/233 (44%)
Aortic atresia, n/known (%)	142/234 (61%)
Obstructed pulmonary venous return, n/known (%)	11/234 (5%)

MBTS, Modified Blalock–Taussig shunt; *IQR*, interquartile range.

TABLE E2. Linear regression models estimating pulmonary artery size at stage II

Model	Variable	Point estimate ± SE	*P* value
LPA diameter at stage II, cm	Intercept	0.25 ± 0.05	<.0001
	LPA diameter at Norwood, cm	0.61 ± 0.11	<.0001
	MBTS resistance, WU*m^2	–0.006 ± 0.002	.005
RPA diameter at stage II, cm	Intercept	0.32 ± 0.04	<.0001
	RPA Diameter Norwood, cm	0.31 ± 0.09	.0005
	MBTS resistance, WU*m^2	0.00002 ± 0.002	.99

SE, Standard error, *LPA*, left pulmonary artery; *MBTS*, modified Blalock-Taussig Shunt; *WU*m^2*, Woods Units meters-squared; *RPA*, right pulmonary artery.