SURGERY FOR CONGENITAL HEART DISEASE

FACTORS INFLUENCING ARTERIAL OXYGENATION EARLY AFTER BIDIRECTIONAL CAVOPULMONARY SHUNT WITHOUT ADDITIONAL SOURCES OF PULMONARY BLOOD FLOW

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Objectives: Although the arterial oxygen saturation after bidirectional cavopulmonary shunting should theoretically be homogeneous if additional pulmonary flow is obliterated, the arterial oxygen saturation has been found to vary in clinical practice. Knowledge of the preoperative and operative determinants of arterial oxygen saturation early after bidirectional cavopulmonary shunting may lead to a better understanding of this unique physiology.

Methods: Thirty-five patients who underwent bidirectional cavopulmonary shunting with obliteration of additional pulmonary flow were included in this study. The arterial oxygen saturation was determined at the 5 time points over a 48-hour period. Multivariable regression analysis was used to identify the independent predictors of the arterial oxygen saturation.

Results: No significant interval changes occurred in the arterial oxygen saturation during the 48 hours after bidirectional cavopulmonary shunting, which ranged from 61.6% to 95.6%. There was a significant inverse correlation between the postoperative superior vena cava pressure and the arterial oxygen saturation (P = .003). A low arterial oxygen saturation early after bidirectional cavopulmonary shunting was a predictor of mortality or exclusion from univentricular repair within 24 months (P = .012, odds ratio = 1.14). Of 11 factors identified by univariable analysis, multiple regression analysis indicated that age less than 8 months at the time of shunting (P < .0001) and ventricular volume overload (P = .002) predicted a lower arterial oxygen saturation after bidirectional cavopulmonary shunting.

Conclusions: Even without additional sources of pulmonary blood flow, several preoperative factors, including younger age and severe ventricular volume overload, predicted a decrease in the arterial oxygen saturation early after bidirectional cavopulmonary shunting. This, in turn, predicted poor outcome during 2 years of follow-up. (J Thorac Cardiovasc Surg 2000;120:589-95)

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Creation of a bidirectional cavopulmonary shunt (BCPS) has become increasingly popular as an interim step in univentricular repair for several reasons. First, it abolishes ventricular volume overload, which can impair ventricular performance. Second, it offers the opportunity to repair associated lesions (systemic ventricular outflow tract obstruction, atrioventricular valve regurgitation, and pulmonary venous obstruction), which may adversely affect prognosis. Third, the unique circulation created by BCPS makes the patient's physiology simpler, allowing more accurate evaluation

Table I. *Patient demographic and operative characteristics*

Variables	*n*	*Sao_2*	P *value*
Sex			.14
Male	19	83.7 ± 8.9	
Female	16	79.1 ± 8.8	
Age at BCPS			<.0001*
≥8 mo	29	84.5 ± 6.7	
<8 mo	6	67.6 ± 4.8	
Weight at BCPS			.002*
≥7 kg	27	84.1 ± 6.7	
<7 kg	8	73.2 ± 11.1	
Height at BCPS			.18
≥70 cm	25	82.9 ± 7.2	
<70 cm	10	78.3 ± 12.4	
Body surface area at BCPS			.045*
≥0.35 m^2	27	83.2 ± 7.3	
< 0.35 m^2	8	76.0 ± 12.3	
Heterotaxy			>.2
Present	20	80.7 ± 9.2	
Absent	15	82.8 ± 9.0	
Urgency for BCPS			.027*
Elective	23	84.0 ± 7.8	
Urgent	12	77.0 ± 9.8	
Morphology of the atrioventricular valve			.018*
Double-inlet or tricuspid atresia	15	85.7 ± 6.6	
Common-inlet or mitral atresia	20	78.5 ± 9.5	
Associated major aortopulmonary collateral artery			>.2
Present	2	81.5 ± 8.4	
Absent	33	81.6 ± 9.2	
Pulmonary flow from systemic arteries			.009*
Present	25	84.1 ± 8.2	
Absent	10	75.5 ± 8.4	
Atrioventricular valve regurgitation			.14
Moderate or massive	10	78.0 ± 9.3	
Mild or less	25	83.0 ± 8.7	
Ventricular volume overload			.005*
Greater	12	75.6 ± 10.2	
Lesser	23	84.3 ± 7.1	
Previous operation(s)			.100*
Yes	26	83.1 ± 9.3	
No	9	77.3 ± 7.1	
Previous operation(s) through a lateral thoracotomy			.14
Yes	23	83.2 ± 9.2	
No	12	78.4 ± 8.2	
Pulmonary vein obstruction			.050*
Present	5	74.6 ± 6.7	
Absent	30	82.8 ± 8.9	
Use of cardioplegic cardiac arrest			.011*
Yes	18	77.9 ± 9.9	
No	17	85.5 ± 6.2	
Aortic crossclamp time			.008*
≥20 min	15	77.0 ± 10.7	
<20 min	20	85.0 ± 5.8	
Application of modified ultrafiltration			>.2
Yes	15	82.8 ± 10.2	
No	20	80.7 ± 8.2	
Concomitant procedure			.12
Yes	22	79.8 ± 9.3	
No	13	84.7 ± 7.9	
Bilateral SVC anastomosis			>.2
Yes	14	81.8 ± 9.4	
No	21	81.4 ± 9.0	

The Sao_2s are averages during the first 48 hours after BCPS. Pulmonary flow from systemic arteries includes surgically created shunt, patent ductus arteriosus, and major aortopulmonary collateral arteries. *BCPS,* Bidirectional cavopulmonary shunt; *Sao_2,* arterial oxygen saturation.
*$P < .1$.

of ventricular performance and pulmonary vascular resistance, which is important for patient selection for univentricular repair. BCPS, therefore, can be considered as a screening test to identify patients who would benefit from univentricular repair. The pulmonary/systemic flow ratio after BCPS theoretically should be approximately 0.5 if additional pulmonary flow is obliterated, because the pulmonary blood flow is equal to the flow to the upper systemic arterial system or the superior vena cava (SVC) flow.[1,2] Arterial blood oxygen saturation (Sa_{O_2}) in the presence of BCPS is the weighted average of the oxygen saturation in the inferior vena cava and in the pulmonary veins and should be relatively constant if the oxygen saturation of the systemic and pulmonary veins is normal.[3] However, the Sa_{O_2} after BCPS often varies in the clinical setting. A low level of Sa_{O_2} can occur early after BCPS, leading to injury of the brain, heart, liver, and kidneys. Identifying the determinants of Sa_{O_2} early after BCPS may increase our understanding of the physiology of this unique circulation, improve the selection of candidates for BCPS, determine the need for additional blood flow, and improve the management of postoperative oxygenation. This study was therefore performed to identify the clinical parameters that affect the Sa_{O_2} early after BCPS.

Patients and methods

Patients. This study included 35 patients with single ventricle physiology undergoing BCPS and concomitant obliteration of additional pulmonary flow between November 1993 and July 1999 at the Keio University Hospital. Patients with pre-existing myocardial dysfunction necessitating inotropic support (n = 4), takedown of a univentricular repair to a BCPS (n = 2), or associated azygos continuation (n = 1) undergoing total cavopulmonary shunt (Kawashima procedure[4]) were excluded from this series. There were 19 male and 16 female patients. The ages at the time of BCPS ranged from 4 months to 22 years (median, 1.2 years). Atrioventricular alignment consisted of common inlet (n = 15, 43%), tricuspid atresia or severe stenosis (n = 8, 23%), double inlet (n = 7, 20%), and mitral atresia or severe stenosis (n = 5, 14%). Twenty-six patients (74%) had undergone previous operative procedure(s), including creation of systemic–pulmonary arterial shunts (22 patients), pulmonary arterial banding (2 patients), pulmonary vein reconstruction (2 patients), and pulmonary arterial augmentation (1 patient) (numbers are not mutually exclusive). All patients were catheterized before BCPS to assess all sources of pulmonary blood flow.

BCPS was indicated for a variety of reasons. In 12 patients, BCPS was performed urgently to treat symptoms. Eight patients underwent BCPS to treat congestive heart failure, which was due to excessive pulmonary blood flow or moderate pulmonary overcirculation in the face of atrioventricular valve regurgitation. In 2 patients, BCPS was performed to treat the outgrowth of the pulmonary blood flow limited by a systemic–pulmonary artery shunt or congenital pulmonary stenosis. Two patients underwent BCPS to treat anoxic spells. The remaining 23 patients were free of symptoms and underwent BCPS on an elective basis as an interim part of staged univentricular repair.

Operative procedures. The operations were performed by one of two surgeons (R.A. or T.K.). All procedures were performed through a midline sternotomy and used cardiopulmonary bypass (average time, 109 ± 41 minutes). Aortic crossclamping and cardioplegic cardiac arrest were used for any intracardiac procedures (18 patients; average time, 38 ± 19 minutes). The bypass technique included aortic and bicaval (or tricaval, if necessary) cannulation and moderate hypothermia (lowest rectal temperature, 28°C). Conventional ultrafiltration was used during the entire period of cardiopulmonary bypass, and modified ultrafiltration was used in 15 patients who underwent surgery after 1996. Twenty-five patients (71%) had pulmonary flow through the systemic arteries before BCPS, including surgically created systemic–pulmonary arterial shunts in 22 patients, patent ductus arteriosus in 10 patients, and major aortopulmonary collateral arteries in 2 patients (numbers are not mutually exclusive). Thirty-two patients (91%) had pulmonary flow through the ventricles and pulmonary valves (22 patients had both).

All previously created systemic–pulmonary artery shunts were taken down. The patent ductus arteriosus and major aortopulmonary collateral arteries were also obliterated before the initiation of cardiopulmonary bypass. In 30 patients who had pulmonary blood flow through the pulmonary valve, this source was also removed either by ligation of the pulmonary trunk or by suture closure of the pulmonary valve. Therefore, the BCPS provided the only source of pulmonary blood flow at the completion of the operation. All azygos and hemiazygos veins were suture ligated. The unilateral (n = 21) or bilateral (n = 14) SVC was transected 1 cm distal (cephalic) to the cavoatrial junction. The proximal (cardiac) end was primarily suture closed. A longitudinal incision was made in the superior aspect of the pulmonary artery, and the cephalic end of the transected SVC was anastomosed in an end-to-side fashion with 6-0 continuous polypropylene sutures. Temporary atrial pacing was performed as necessary. Concomitant procedures other than the takedown or obliteration of additional sources of pulmonary blood flow were performed in 22 patients (63%). These procedures included pulmonary artery augmentation in 12 patients, atrioventricular valvuloplasty in 7 patients, repair of anomalous pulmonary venous connections or atrial septectomy for pulmonary congestion in 5 patients, and the Damus-Kaye-Stansel procedure in 1 patient (numbers are not mutually exclusive). Of the 10 patients with moderate or massive atrioventricular valve regurgitation, 7 patients underwent reparative procedures. No reparative procedures on the atrioventricular valve were performed in the remaining 3 patients who had excessive pulmonary blood flow.

Anesthesia. All patients underwent the same anesthesia protocol. After oral intubation, general anesthesia was maintained with fentanyl and pancuronium bromide. A ventilator (model E100A, Newport Medical Inc, Newport Beach, Calif)

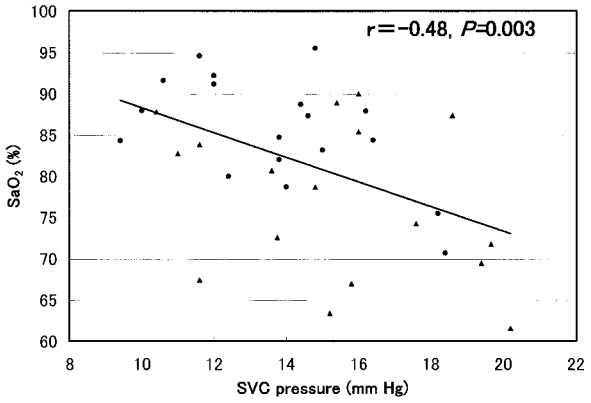

Fig 1. Correlation between the average arterial oxygen saturation *(Sao_2)* and average superior vena cava *(SVC)* pressure during the first 48 postoperative hours. *Circles* represent patients who underwent successful univentricular repair within 24 months after bidirectional cavopulmonary shunt; *triangles* represent patients who did not.

was used in both the operating room and the intensive care unit. The ventilator settings were very similar between the patients during the first 12 hours after surgery, including an inspired oxygen fraction of 1.0, an end-inspiratory pressure of 20 to 24 mm Hg, an end-expiratory pressure of 3 to 5 mm Hg, an inspiratory/expiratory phase ratio of 1:2, and a ventilatory rate of 20 breaths/min. After cardiopulmonary bypass, all patients received intravenous nitroglycerin (2 to 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$), but weaning from cardiopulmonary bypass was always attempted without inotropic support. Only 10 patients (29%) required inotropic support. The patients were weaned from the ventilator, and supplemental oxygen therapy was continued for at least 3 days.

Data acquisition and analysis. Medical and surgical records were reviewed retrospectively. The clinical characteristics of the patients are summarized in Table I. Preoperative echocardiograms and preoperative cardiac catheterization findings were available in 35 patients (100%) and discharge echocardiograms in 31 patients (89%). Preoperative pulmonary arterial pressure at catheterization was measured by passage of the catheter across the pulmonary valve or systemic–pulmonary arterial shunt. Atrioventricular valve regurgitation was graded as none, trivial, mild, moderate, or massive on the basis of qualitative assessment of the regurgitant jet by color Doppler flow mapping and angiography. Ventricular wall motion was graded as normal or mildly, moderately, or severely depressed on the basis of qualitative evaluation by 2-dimensional echocardiography and angiography. Greater ventricular volume overload was defined as moderate or massive atrioventricular valve regurgitation or a cardiothoracic ratio on the chest radiograph of 0.6 or greater.

Perioperative Sa_{O_2} was evaluated by co-oximetry 20 minutes, 2 hours, 12 hours, 24 hours, and 48 hours after the discontinuation of bypass. At the same time points, the arterial blood pH, oxygen tension (Pa_{O_2}), and carbon dioxide tension (Pa_{CO_2}) were determined, and the SVC pressure and hemoglobin concentration were recorded.

Follow-up was completed in all the patients after 2.6 ± 2.1 years (range, 0.1-5.9 years). Follow-up echocardiograms were available in 30 patients (86%), and follow-up cardiac catheterizations were available in 26 patients (74%). All patients surviving beyond 6 months after BCPS were examined with both echocardiograms and cardiac catheterization.

The statistical package SPSS for Windows version 9.0.1 (SPSS Inc, Chicago, Ill) was used for all statistical calculations. The interval changes in Sa_{O_2}, SVC pressure, atrioventricular valve regurgitation, and ventricular wall motion were compared by analysis of variance (ANOVA) with repeated measures or by the Wilcoxon rank test, where appropriate. Putative factors that may have influenced the average values of the Sa_{O_2} at the 5 time points during the 48 hours after BCPS were analyzed univariably (Table I). Relationships of the average Sa_{O_2} and normally distributed continuous variables, including preoperative hemoglobin concentration, preoperative Sa_{O_2}, preoperative pulmonary vascular resistance, preoperative pulmonary arterial pressure, preoperative pulmonary arterial cross-sectional index,[5] cardiopulmonary bypass time, and fluid balance index during surgery (fluid balance [milliliters] divided by body surface area [square meters]) were examined by correlation analysis. Because age at BCPS, weight at BCPS, height at BCPS, body surface area at BCPS, and aortic cross-clamp time were not normally distributed even after the logarithmic transformation, these variables were dichotomized by means of break points after the linearity of the relationship between each variable and the average Sa_{O_2} had been examined. Differences in the average Sa_{O_2} with and without categoric and dichotomized variables were compared by means of the Student *t* test. A stepwise forward selection procedure was then used to develop a multiple regression model predictive of the average Sa_{O_2}. Variables that were significant at the .10 level on the basis of univariable analysis were considered for inclusion in the model. A P value < .05 was required for variables to remain in the model. Logistic regression was used to determine the contribution of the Sa_{O_2} or the SVC pressure to the prediction of mortality or exclusion from completion of the univentricular repair. All continuous values are expressed as the mean ± 1 standard deviation.

Results

Arterial oxygenation. The values for the Sa_{O_2}, Pa_{O_2}, Pa_{CO_2}, arterial pH, SVC pressure, and hemoglobin concentration are summarized in Table II. No significant interval changes in the Sa_{O_2} were noted during the first 48 postoperative hours (ANOVA with repeated measures), even though the inspired oxygen fraction decreased from 1.00 ± 0.00 at 20 minutes to 0.62 ± 0.20 at 48 hours. The Sa_{O_2} ranged from 61.6% to 95.6%

Table II. *Repeated measures of arterial oxygenation and related variables*

	Preop	*20 min*	*2 h*	*12 h*	*24 h*	*48 h*	*Follow-up*
Sa_{O_2} (%)	81.7 ± 6.7	81.3 ± 9.8	80.7 ± 10.9	82.9 ± 9.1	81.8 ± 9.1	81.9 ± 8.6	82.9 ± 6.4
Pa_{O_2} (mm Hg)	45.7 ± 6.1	45.3 ± 12.5	43.4 ± 13.3	45.9 ± 14.6	45.6 ± 9.0	45.9 ± 11.8	52.1 ± 3.6
Pa_{CO_2} (mm Hg)	38.6 ± 4.8	31.0 ± 6.4	27.4 ± 5.1	28.2 ± 5.9	33.2 ± 6.3	33.4 ± 5.9	36.2 ± 5.3
Arterial pH	7.37 ± 0.04	7.46 ± 0.07	7.52 ± 0.08	7.51 ± 0.06	7.45 ± 0.05	7.46 ± 0.04	7.39 ± 0.07
PA/SVC pressure (mm Hg)	16.8 ± 9.6	14.7 ± 3.7	15.9 ± 3.9	14.1 ± 3.1	14.2 ± 3.6	13.3 ± 3.3	10.0 ± 3.3*
Hemoglobin concentration (g/dL)	16.8 ± 2.0	12.4 ± 1.8	12.2 ± 2.0	13.5 ± 1.6	14.0 ± 1.3	14.9 ± 1.3	16.4 ± 1.5†

Repeated measures of arterial oxygen saturation *(Sa_{O_2})*, arterial oxygen tension *(Pa_{O_2})*, arterial carbon dioxide tension *(Pa_{CO_2})*, arterial pH, pulmonary artery or superior vena cava pressure *(PA/SVC)*, and hemoglobin concentration. The PA/SVC pressure is the SVC pressure at all time points except the preoperative time point, where the mean pulmonary arterial pressure is used instead. Measurements were compared with repeated-measures analysis of variance.

*Significantly different ($P < .001$) values when compared with those during the first 48 hours after operation.

†Significantly different ($P < .02$) values when compared with those during the first 48 hours after operation.

(average for the 5 time points, 81.6% ± 9.0%). Similarly, the Pa_{O_2} showed little change during the 48 hours (average, 45.1 ± 11.0 mm Hg). The Pa_{CO_2} (average, 30.6 ± 4.0 mm Hg), arterial pH (average, 7.48 ± 0.04), and hemoglobin concentration (average, 13.4 ± 1.2 g/dL) were all held in a narrow range among the patients during the first 48 hours after BCPS. The SVC pressure ranged from 9 mm Hg to 20 mm Hg (average for the 5 time points, 14.5 ± 2.9 mm Hg). There was a significant inverse correlation between the postoperative SVC pressure and the Sa_{O_2} ($r = –0.48$, $P = .003$, Fig 1).

Univariable analysis revealed that age less than 8 months, weight less than 7 kg, body surface area less than 0.35 m^2, urgent surgery, the presence of common-inlet or mitral atresia, the absence of pulmonary flow from systemic arteries before BCPS, greater ventricular volume overload, no previous operation(s), and pulmonary vein obstruction were identified as preoperative risk factors for a lower average value of the Sa_{O_2} at the 5 time points during the 48 hours after BCPS (Table I). The use of cardioplegic cardiac arrest and an aortic crossclamp time of longer than 20 minutes were identified as operative risk factors for a lower Sa_{O_2}. Sex, height, heterotaxy, the presence of an associated major aortopulmonary collateral artery, atrioventricular valve regurgitation, history of a previous operation through a lateral thoracotomy, the use of modified ultrafiltration, the use of concomitant procedures, and bilateral SVC anastomosis did not influence the postoperative Sa_{O_2}. None of the continuous variables were identified as significant determinants. Of 11 factors identified by univariable analysis, the final model of multiple regression analysis identified age less than 8 months (non-standardized coefficient = –15.6, standard error = 2.5, $P < .0001$) and greater ventricular volume overload (non-standardized coefficient = –6.8, standard error = 2.0, $P = .002$) as predictors of a decreased Sa_{O_2} after BCPS (the intercept = 86.6%, standard error = 2.5). The regression coefficient of the model including these 2 risk factors was 0.80 ($R^2 = 0.64$).

Postoperative recovery and follow-up. BCPS and obliteration of additional pulmonary blood flow were performed in all of the patients. Nitric oxide inhalation was required in 4 patients (11%) because of severe hypoxemia or a severely elevated SVC pressure. Four (11%) early deaths occurred. Two patients died of severe hypoxemia ($Sa_{O_2} < 70\%$). Nitric oxide inhalation therapy did not improve their hypoxia. One of these 2 patients was a 5-month-old infant with tricuspid atresia and an unrestrictive bulboventricular foramen who underwent BCPS and a Damus-Kaye-Stansel anastomosis; the other was a 7-month-old patient with asplenia syndrome and massive common atrioventricular valve regurgitation who underwent BCPS and a successful atrioventricular valvuloplasty confirmed by postoperative echocardiography. One patient died of sudden-onset bradycardia after a stable postoperative recovery. A 6-month-old patient with asplenia syndrome had respiratory distress and died after an uneventful postoperative recovery. Echocardiography performed at the time of discharge revealed that atrioventricular valve regurgitation had improved significantly ($P = .014$); moderate or massive regurgitation preoperatively noted in 10 patients improved to mild or trivial degrees in all but 2 patients. One patient had residual massive regurgitation after tricuspid valvuloplasty with artificial chordae and rent closure. In the other patient with moderate regurgitation, the degree of regurgitation remained the same after BCPS. Ventricular wall motion was similar before and after BCPS in all of the patients.

There were 5 late deaths (14%), all occurring between 2 and 4 months after BCPS. One patient who died was a 22-year-old patient with asplenia syndrome

in whom pulmonary artery thrombosis developed, causing severe hypoxemia. Other causes of late death included septicemia (n = 2), sudden death (n = 1), and myocarditis (n = 1).

At the time of follow-up cardiac catheterization (6.6 ± 2.8 months after BCPS), additional sources of pulmonary blood flow were all completely eliminated. Pulmonary arteriovenous fistulas were not identified, although major collateral blood vessels between the SVC and atrium were identified in 2 patients. None of the patients had clinically significant pulmonary venous obstruction (pressure gradient > 5 mm Hg). The SVC pressure ranged from 4 to 17 mm Hg (average, 10.0 ± 3.3 mm Hg), which was significantly lower than during the first 48 hours after BCPS ($P < .001$, ANOVA with repeated measures). The $Sa\overline{O}_2$ with the patients breathing room air ranged from 73% to 92% (average, 83.8% ± 4.5%). This value correlated with the $Sa\overline{O}_2$ during the first 48 hours after BCPS ($P = .032$) and did not differ from the values during the first 48 hours. The hemoglobin concentration increased to 16.4 g/dL when compared with those during the first 48 hours after the operation.

Of the 35 patients, 20 underwent completion of univentricular repair by means of a total cavopulmonary connection at a mean of 10.6 ± 3.9 months (range, 1.5-19.1 months) after BCPS, although 2 patients required takedown of the shunt because of severe hypoxemia or an elevated SVC pressure (>25 mm Hg). All patients survived the univentricular repair and are in New York Heart Association functional class II or I. Six patients could not undergo univentricular repair within 24 months after BCPS. Four of the patients had an indexed pulmonary vascular resistance of more than 2 Wood units. One patient had residual massive atrioventricular valve regurgitation after unsuccessful valvuloplasty. One patient had a cerebrovascular accident after BCPS. Logistic regression analysis revealed that a lower $Sa\overline{O}_2$ early after BCPS was a significant predictor of mortality or exclusion from univentricular repair within 24 months after BCPS ($P = .012$; odds ratio, 1.14; 95% confidence interval, 1.02-1.26). The SVC pressure early after BCPS was not identified as a predictor of outcome.

Discussion

Our results demonstrate that physiologic rather than anatomic factors help to decrease the $Sa\overline{O}_2$ after BCPS. Young infancy at the time of BCPS was an independent predictor of a low $Sa\overline{O}_2$ after BCPS, which is in keeping with previous reports.[6] The SVC flow accounts for 49% of the cardiac output in newborn infants, reaches a maximum contribution of 55% at 2.5 years of age, and then gradually decreases to the adult value of 35% by 6.6 years of age.[2] Since pulmonary flow is almost identical to SVC flow after BCPS because there is no additional source of pulmonary blood flow, the pulmonary/systemic flow ratio should be age-dependent. Although an age of 6.6 years or older is a potential risk factor for a lower $Sa\overline{O}_2$ after BCPS, our series included only 4 patients who were 6.6 years of age or older. This may explain why older age was not identified as a risk factor. In fact, Gross and associates[7] explored determinants of increased cyanosis late after BCPS with an older patient population in which older age was an independent determinant of a systemic $Sa\overline{O}_2$ of less than 75% at follow-up catheterization. Young infants should have a lower SVC flow than older patients, which may explain the lower $Sa\overline{O}_2$ in this subgroup. However, there is another possible explanation. Young infants may have a higher pulmonary vascular resistance, which has been reported to correlate with postoperative hypoxemia ($Sa\overline{O}_2 < 75\%$) early after BCPS.[8] In our study, an elevated SVC pressure correlated with a low $Sa\overline{O}_2$. This can be explained by an elevated SVC or pulmonary arterial pressure serving as a driving force to open collaterals of pulmonary arteriovenous or systemic upper-lower venous channels. In our patients, myocardial function and atrioventricular valve function were not impaired, and pulmonary venous obstruction was either absent or successfully repaired. Pulmonary vascular resistance is one of the most important determinants of SVC pressure and, therefore, $Sa\overline{O}_2$. However, preoperative cardiac catheterization with the Fick method often fails to evaluate pulmonary vascular resistance accurately before cavopulmonary shunt.[9,10] This may explain why the preoperative pulmonary vascular resistance is not an independent risk factor for a low $Sa\overline{O}_2$.

A greater preoperative ventricular volume overload was also an independent predictor of a low $Sa\overline{O}_2$. With a greater preoperative ventricular volume load, ventricular volume-mass mismatch should be greater early after BCPS. Ventricular volume-mass mismatch is a potential cause of impaired ventricular performance. This is the primary reason for performing a staged operation for univentricular repair. Although the impact of ventricular volume-mass mismatch on ventricular performance has not been studied much in BCPS, it should occur in BCPS as well. Lower arterial oxygenation may be explained by lower systemic venous oxygenation caused by poorer ventricular performance.

Our observation has several limitations. First, it is necessary for definition of pathophysiology in BCPS to determine oxygen saturation in both the upper and lower systemic venous blood and cardiac output, which

was not done in our retrospective study. It has been our policy not to place indwelling catheters in the vessels that drain into the systemic ventricle to prevent systemic thromboembolic complications. Continuous or repeated monitoring of the cardiac output after surgery in small patients has several limitations, including inaccuracy. Second, the ventilatory status can affect the cerebral blood flow and, therefore, pulmonary flow after BCPS.[11] However, all of the patients in the present study were hyperventilated, and the interpatient variability in the $PaCO_2$ was low (average $PaCO_2$, 30.6 ± 4.0 mm Hg). Third, this study has ignored several anomalies that cannot be evaluated. Specifically, a mismatch between pulmonary perfusion and ventilation can often develop, causing suboptimal oxygenation after total cavopulmonary connection.[12] In addition, high collateral flows can be encountered at the time of primary univentricular repair even if primary sources to the pulmonary artery are removed.[13] BCPS can also cause perfusion-ventilation mismatch and collateral flows. In this study, the SaO_2 was predicted by 2 risk factors with an R^2 value of 0.64. The remaining could be explained in part by the presence of these anomalies in pulmonary physiology.

Despite the limitations discussed above, our observations have several clinically relevant implications. First, there has previously been no consensus regarding whether or not additional pulmonary blood flow should be added or preserved at BCPS.[6-8,14-19] In our study, decreased arterial oxygenation early after discontinuation of cardiopulmonary bypass in BCPS often persisted for the subsequent 48 hours and was a predictor of increased 2-year morbidity and mortality. This finding suggests that use of a revised procedure with the addition or preservation (reopening) of additional sources of pulmonary blood flow may be beneficial if the patient has decreased SaO_2 and has a low SVC pressure early after BCPS. Second, if a patient with none of these risk factors has an unexpectedly low SaO_2, the cause should be extensively explored.

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