Changes in cerebral and somatic oxygenation during stage 1 palliation of hypoplastic left heart syndrome using continuous regional cerebral perfusion

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Objectives: Stage 1 palliation of hypoplastic left heart syndrome requires the interruption of whole-body perfusion. Delayed reflow in the cerebral circulation secondary to prolonged elevation in vascular resistance occurs in neonates after deep hypothermic circulatory arrest. We examined relative changes in cerebral and somatic oxygenation with near-infrared spectroscopy while using a modified perfusion strategy that allowed continuous cerebral perfusion.

Methods: Nine neonates undergoing stage 1 palliation for hypoplastic left heart syndrome had regional tissue oxygenation continuously measured by frontal cerebral and thoraco-lumbar (T10-L2) somatic (renal) reflectance oximetry probes (rSO₂, INVOS; Somanetics, Troy, Mich). Surgery was accomplished using cardiopulmonary bypass with whole-body cooling (18°C-20°C) and regional cerebral perfusion through the innominate artery at flow rates guided by estimated minimum flow requirements and measured rSO₂ during reconstruction of the aortic arch. Data were logged at 1-minute intervals and analyzed using repeated measures analysis of variance.

Results: A total of 3176 minutes of data were analyzed. Prebypass cerebral rSO₂ was 65.4 ± 8.9, and somatic rSO₂ was 58.9 ± 12.4 ($P < .001$, cerebral vs somatic). During regional cerebral perfusion, cerebral rSO₂ was 80.7 ± 8.6, and somatic rSO₂ was 41.4 ± 7.1 ($P < .001$). Postbypass cerebral rSO₂ was 53.2 ± 14.9, and somatic rSO₂ was 76.4 ± 7.7 ($P < .001$). The risk of cerebral desaturation was significantly increased after cardiopulmonary bypass.

Conclusions: Cerebral oxygenation was maintained during regional cerebral perfusion at prebypass levels with deep hypothermia. However, after rewarming and separation from cardiopulmonary bypass, cerebral oxygenation was lower compared with prebypass or somatic values. These results indicate that cerebrovascular resistance is increased after deep hypothermic cardiopulmonary bypass, even with continuous perfusion techniques, placing the cerebral circulation at risk postoperatively.

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Strategies to improve outcome after cardiopulmonary bypass (CPB) and circulatory arrest for complex congenital heart disease have been directed at optimizing metabolic suppression and oxygen delivery with adequate cooling time and pH-stat perfusion strategies,^{1,2} reducing hemodilution^{3,4} and ischemia time,⁵ and preventing endothelial dysfunction and related injury.^{1,6-9} Near-infrared spectroscopy (NIRS) has aided the study of regional perfusion by providing a noninvasive

estimate of tissue oxyhemoglobin saturation.¹⁰⁻¹² Continuous regional cerebral perfusion (RCP) can provide blood flow to the head during aortic arch reconstruction,¹³ and maintenance of cerebral oxygen saturation during RCP has been demonstrated with NIRS.^{12,14} However, delayed abnormalities in regional circulation may occur after deep hypothermic CPB and deep hypothermic circulatory arrest (DHCA) because of prolonged changes in cerebrovascular function.^{15,16} We hypothesized that such alterations in regional autoregulation, in combination with inadequate global oxygen delivery post-CPB secondary to functional and anatomic circulatory limitations,¹⁷ place tissues in selective vascular beds at risk for ischemia.¹⁷⁻¹⁹ We used a commercially available NIRS device to assess changes in oxyhemoglobin saturation in two different regional circulations (cerebral^{10,11,14} and thoraco-lumbar or somatic^{13,20}) before, during, and immediately after stage 1 palliation (S1P) of hypoplastic left heart syndrome, hypothesizing that continuous cerebral perfusion would preserve cerebral oxygenation both during and after CPB.

Methods

Patients and Clinical Techniques

Nine patients undergoing stage 1 palliation for hypoplastic left heart syndrome were studied. Institutional review board approval, parental consent for administration of phenoxymethylamine, and clinical physiologic data were obtained for all patients. Hypercapnia was used to control pulmonary blood flow and improve systemic oxygen delivery as necessary before CPB.²¹ High-dose, synthetic, opioid-based anesthesia with supplemental isoflurane was induced in all patients, with opioid infusions maintained into the postoperative period.²² Surgical repair was performed using techniques previously described: relief of arch obstruction, ascending aorta-to-pulmonary artery trunk anastomosis, homograft augmentation of the aortic arch, placement of a systemic-to-pulmonary shunt, and creation of unrestrictive interatrial patency.²³ CPB was initiated through a synthetic shunt anastomosed to the innominate artery using a Stockert SC roller pump (Sorin Biomedical, Irvine, Calif) and a Dideco Lilliput 1 hollow fiber membrane oxygenator (Cobe Cardiovascular, Arvada, Calif). CPB management consisted of target flows of 2 to 4 L · min⁻¹ and hematocrit 25% to 30% with pH-stat blood gas management for uniform cooling (18°C-20°C) at nasopharyngeal and bladder sites (minimum time 30 minutes); continuous cerebral perfusion through the innominate shunt at pump flows of 30 to 70 mL · kg⁻¹ · min⁻¹ during neo-aortic and arch reconstruction; and resumption of full support at target flows of 3 to 4 L · min⁻¹ after somatic and cardiac reperfusion, with a gradual switch to alpha-stat strategy and hemoconcentration during rewarming to a target hematocrit of 35% or greater before separation from CPB. All patients received aprotinin 1.7 × 10⁶ KIU/m² and methylprednisolone 10 mg/kg before CPB, and phenoxymethylamine 0.25 mg/kg was added to the pump prime. Separation from CPB occurred after initiation of infusions of milrinone (0.5 μg · kg⁻¹ · min⁻¹), dopamine (3 μg · kg⁻¹ · min⁻¹), and epinephrine or norepinephrine (0.05-0.15 μg · kg⁻¹ · min⁻¹) or both as needed to achieve an approximate

systemic vascular resistance index of 10 to 12 Wood units and adequate contractility. Modified ultrafiltration was used in all patients immediately after separation from CPB. Post-CPB hemodynamic management included a primary target of SvO₂ of 50% or greater, including mean arterial pressure greater than 45 mm Hg, SaO₂ greater than 75%, and hematocrit greater than 40% as previously described.¹⁷ No alterations from standard treatment were undertaken during the study period except to use rSO₂ data to guide RCP to a target of 75% or greater.

Monitoring and Data Acquisition

In all patients, blood pressure was invasively monitored with an existing umbilical artery catheter, and atrial pressure was monitored with an umbilical vein catheter if present or an atrial line placed at the time of surgery. Arterial oxygen saturation was monitored continuously in the lower extremity (Nellcor N200; Pleasanton, Calif). Systemic venous oxygenation (SvO₂) was monitored continuously during CPB from the venous drainage (Terumo CDI-500; Ann Arbor, Mich) and post-CPB using a 4FR optical catheter (Abbott Oxycath; Abbott Laboratories, Abbott Park, Ill) placed in the superior vena cava before separation from CPB. Airway gas tensions were monitored using Raman spectroscopy (Ohmeda Rascal-2, Louisville, Colo). Data from these sources were captured on a clinical information system (GE/Marquette Solar; Milwaukee, Wis) and trended at 5-minute intervals. NIRS probes were placed on the patient's midline forehead (cerebral) and slightly to the right of midline on the T10-L2 posterior flank (somatic) after entry into the operating room. The probes were monitored by a dual-detector device (Somanetics INVOS 5100A, Troy, Mich) and trended at 1-minute intervals. CPB flows, temperatures, and pressures were captured at 5-minute intervals. Arterial and venous blood gases were obtained at clinically appropriate intervals, with tensions reported at 37°C (Radiometer ABL, Copenhagen, Denmark).

Statistical Analysis

Prospective, electronically captured, and manually recorded data were inserted into a common statistical database. In addition to measured variables, derived variables were calculated as follows: organ perfusion pressure (PP) = mean arterial blood pressure – central venous pressure; cerebral Da-rO₂ = SaO₂ – cerebral rSO₂; somatic Da-rO₂ = SaO₂ – somatic rSO₂; DrSO₂ (cerebral-somatic) = cerebral rSO₂ – somatic rSO₂; oxygen content PP index (CaO₂ · PPI) = 1.34 · hemoglobin · PP. Each patient's clinical data were divided into 5 major time periods consisting of 11 intervals, demarcated by preinduction baseline, anesthesia induction, surgical incision, initiation of CPB, initiation of myocardial ischemia with aortic crossclamp, initiation of RCP, resumption of CPB, removal of aortic crossclamp, separation from CPB, completion of modified ultrafiltration, and surgical closure. Within each interval for each patient, data were collapsed into 5 epochs of equal duration to create 55 distinct epochs for subsequent statistical analysis. Data were summarized within intervals and periods, and presented as mean ± SD if continuous and number and percent if discrete, with 95% confidence intervals (CIs) calculated as appropriate. Generalized least squares regression and analysis of variance (ANOVA) techniques were used to account for repeated measurements within patients and time intervals. Data were com-

TABLE 1. Demographic data

Characteristic	
Age (d)	4.9 ± 1.7
Weight (kg)	2.84 ± 0.59
BSA (m ²)	0.179 ± 0.038
Preoperative Hgb (g/dL)	40 ± 5
Preoperative SpO ₂ (%)	89 ± 3
Preoperative mechanical ventilation	6/9 (67%)
Preoperative inotropes	5/9 (55%)
CPB duration (min)	113 ± 12
RCP duration (min)	58 ± 10

Data are presented as mean ± SD or number (%).

BSA, Body surface area; Hgb, hemoglobin; CPB, cardiopulmonary bypass; RCP, regional cerebral perfusion.

pared across time periods or intervals by 2-sided *t* tests on regression coefficients or by 2-way analysis of variance for repeated measures for comparisons of means and by the likelihood-ratio test for risk comparisons, with significance cutoff at *P* < .05 after correction for multiple comparisons by the Bonferroni method. All calculations were performed with a standard statistical package (STATA version 8; Stata Corp, College Station, Tex).

Results

Demographic and baseline clinical data from the 9 patients are summarized in Table 1. All patients underwent S1P and survived to hospital discharge; 3176 minutes of data were available for analysis (353 min/patient, range 304–418 minutes). Hypothermic RCP was accomplished in all patients at a flow rate of $47 \pm 22 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ($75 \pm 35 \text{ mL} \cdot \text{m}^{-2} \cdot \text{min}^{-1}$).

Regional saturation data differed markedly during periods of perfusion and surgical repair. Preincision baseline cerebral rSO₂ was $68\% \pm 4\%$ until CPB, and baseline somatic rSO₂ was $68\% \pm 8\%$, decreasing to $56\% \pm 14\%$ before CPB. During cooling on CPB, cerebral rSO₂ increased to $87\% \pm 8\%$, and somatic rSO₂ increased to $77\% \pm 13\%$. During RCP, cerebral rSO₂ was maintained at $81\% \pm 9\%$, whereas somatic rSO₂ decreased to $41\% \pm 7\%$, recovering to $77\% \pm 10\%$ and $84\% \pm 13\%$, respectively, during rewarming on CPB. After separation from CPB, regional saturation in both measured beds decreased; cerebral rSO₂ decreased progressively to $48\% \pm 7\%$, whereas the somatic rSO₂ decreased but remained above baseline at $78\% \pm 4\%$. A summary of regional saturations at major time periods is contained in Table 2. Cerebral and somatic rSO₂ were different from each other and from baseline values at all time points (*P* < .001). Detailed regional saturation data are shown in Figure 1.

The post-CPB decrease in cerebral rSO₂ was not solely caused by the reduction in SaO₂. Figure 2 shows the SaO₂, regional SaO₂ – rSO₂ difference, and cerebral-somatic rSO₂ difference by time period. These data show that regional cerebral oxygen extraction, as approximated by the

TABLE 2. Regional saturations at major time periods

Time period	Cerebral rSO ₂ (%)	Somatic rSO ₂ (%)
Pre-CPB	65.4 ± 8.9 (63.2–67.6)	58.9 ± 12.4 (57.8–59.9)†
CPB (cooling)	87.2 ± 7.8 (85.0–89.5)*	77.2 ± 12.5 (75.6–78.8)*†
RCP	80.7 ± 8.6 (78.6–83.0)*	41.4 ± 7.1 (39.9–42.8)*†
CPB (warming)	76.8 ± 10.2 (74.6–79.0)*	83.5 ± 12.9 (82.2–84.8)*†
Post-CPB	53.2 ± 14.9 (51.0–55.5)*	76.4 ± 7.7 (75.3–77.6)*†

Regional saturation data summarized within major operative periods. Data are presented as mean ± SD and (95% confidence intervals). All differences are significant at *P* < .001 by repeated measures ANOVA.

*Differences compared with pre-CPB baseline.

†Differences between sites.

SaO₂ – rSO₂ difference, was increased in the post-CPB period relative to somatic beds. The decrease in cerebral rSO₂ occurred despite increases in hematocrit, CO₂ tension, and mean arterial pressure after separation from CPB, all of which would be expected to favor increases in cerebral blood flow and oxygen delivery. Other potential determinants of cerebral oxygenation such as temperature, pump flow rate, isoflurane concentration, and the CaO₂ PPI, an index of regional oxygen availability, also were maintained in the period after separation from CPB. Complete hemodynamic data are detailed in Table 3.

The relationship between cerebral rSO₂ and potential determinants (mean arterial blood pressure, central venous pressure, SaO₂, Paco₂, temperature, hematocrit, and isoflurane concentration) were evaluated in a multiple linear least squares regression model, comparing all data before and after CPB. The model significantly related the independent variables to cerebral rSO₂ (*r*² = 0.79), and several parameters were significantly different pre- and post-CPB. The complete model parameters are shown in Table 4.

The risk of regional desaturation was assessed during each period as the proportion of time with rSO₂ less than 50%. The overall risks of such cerebral and somatic desaturation were 11% and 23%, respectively. Somatic rSO₂ was threatened after incision (risk 42%, CI 36%–48%, *P* < .001 compared with baseline) and improved during CPB. During RCP, the somatic rSO₂ was below the threshold 89% of the time (CI 84%–92%, *P* < .001 compared with baseline), but the risk of somatic desaturation was essentially zero after separation from CPB. In contrast, the risk of cerebral desaturation was minimal until separation from CPB, when it was 21% (CI 16%–28%, *P* < .001 compared with baseline), and continued to increase after withdrawal of volatile anesthesia to 74% (CI 60%–85%, *P* < .001 compared with baseline). These findings are shown in Figure 3.

Discussion

The major findings of this study were that cerebral rSO₂ decreased post-CPB despite continuous RCP, and that assessment of 2-site regional oxygenation using NIRS tech-

Figure 1. Cerebral and somatic rSO_2 during repair. Repair was divided into 5 major periods consisting of 11 intervals as described. Data are shown as mean and SD for each epoch. *CPB*, Cardiopulmonary bypass; *RCP*, regional cerebral perfusion. *Dotted line* marks preinduction baseline values.

nology provided information about oxygen delivery distinct from global measures. The dual-path length device that was used measures average hemoglobin saturation in a slice of tissue approximately 1.5 to 2.5 cm deep, tracks changes in jugular venous saturation closely, and detects changes in tissue oxygenation in a variety of tissues.^{13,24,25} Although no formal calculations of regional blood flow were made in this study, cerebral rSO_2 has been shown to closely track changes in jugular venous saturation ($r^2 = 0.91$), and thus changes in rSO_2 can estimate regional flow-metabolism relationships.²⁶ We attempted to place somatic probes to capture the renal circulation (which normally has a low extraction ratio but is under intense sympathetic control) as a contrast with the cerebral circulation (which normally exhibits intense perfusion-metabolism coupling). This was performed to serve as a within-patient control for factors such as changes in venous volume and to help differentiate changes in global systemic flow from changes in the distribution of regional flows as potential causes of changes in cerebral rSO_2 .

Interventions to increase cerebral rSO_2 may reduce neurologic morbidity in children undergoing CPB.^{27,28} Although absolute thresholds of rSO_2 for neurologic injury are not known in human neonates, evidence from adult humans²⁹ and neonatal piglets³⁰ indicates that aerobic metabolism is impaired when cerebral rSO_2 decreases below 44% to 47%. Critically low values for cerebral rSO_2 have been reported preoperatively in neonates with a variety of congenital lesions³¹ and were observed with substantial frequency in our patients in the post-CPB period.

The pre-CPB rSO_2 data revealed a normal cerebral arterial-regional saturation difference³¹ but an increased somatic arterial-regional difference, typical of a “stressed” patient with increased resistance in vascular beds subject to regional sympathetic control, with resultant redistribution of

systemic perfusion from splanchnic to cerebral circulations. The baseline hemodynamic data in these infants revealed high SAO_2 and high pulmonary and systemic flow ratios despite preoperative management with hypercapnia and low FIO_2 ; thus, compromised splanchnic and somatic flow were expected.

Before CPB, SvO_2 was slightly lower than cerebral rSO_2 , reflecting the contribution of relatively desaturated somatic blood to SvO_2 . After CPB, the converse was true; SvO_2 was higher than cerebral rSO_2 , reflecting the contribution of more well-saturated somatic blood to SvO_2 . These findings emphasize the differences between regional and global measures of oxygen supply and demand relationships.^{18,32}

The observed divergence of cerebral from somatic rSO_2 post-CPB was expected,³³ although the decrease in cerebral rSO_2 from the pre-CPB baseline indicates that cerebral oxygenation may be vulnerable despite palliation of the circulation through S1P. The reasons for the reduction in cerebral rSO_2 from baseline include (1) reduced global oxygen delivery, (2) reduced somatic and splanchnic resistance, (3) increased cerebral resistance, and (4) changes in the venous blood compartment.

Although global oxygen delivery (SvO_2) was not routinely assessed preoperatively, SvO_2 was maintained after separation from CPB until closure. Similarly, the $CaO_2 \cdot PPI$, a measure of regional oxygen availability, was maintained post-CPB. Thus the decrease in cerebral rSO_2 post-CPB was not likely explained by a reduction in global oxygen delivery. However, because previous data have described the occurrence of decreasing SvO_2 in the first 6 to 12 hours postoperatively,³⁴ a reduction in global oxygen delivery may increase the risk of cerebral desaturation in the later postoperative period.

A reduction in splanchnic and somatic resistance by reactive hyperemia, phenoxybenzamine, milrinone, and

Figure 2. SaO_2 , cerebral-somatic DrSO_2 , cerebral Da-rO_2 , and somatic Da-rO_2 are shown during repair, presented as mean and SD for each epoch. Dotted lines mark baseline measures for each parameter. CPB, Cardiopulmonary bypass; RCP, regional cerebral perfusion.

isoflurane likely contributed to the low risk of somatic desaturation in the immediate post-CPB period. Alpha-adrenergic mechanisms of vasoconstriction are more active in splanchnic, muscle, and skin beds than in organs with

intense autoregulation such as brain and heart.³⁵⁻³⁷ Therefore, alpha-adrenergic blockade would be expected to reduce the sympathetically mediated increase in somatic resistance that occurs in the patient post-CPB with reduced

TABLE 3. Complete hemodynamic data

Parameter	Baseline	Induction	Incision	CPB	AoX
Duration (min)	19 ± 7	21 ± 6	74 ± 12	42 ± 9	4 ± 2
rSO ₂ cerebral (%)	68 ± 4	64 ± 10	65 ± 9	88 ± 8	87 ± 8
rSO ₂ somatic (%)	68 ± 8	62 ± 6	56 ± 14	78 ± 13	69 ± 19
SaO ₂ (%)	90 ± 3	90 ± 4	91 ± 5	99 ± 2	99 ± 2
SvO ₂ (%)	@	@	63 ± 14 @	93 ± 7	92 ± 4
Temperature (centigrade)	37.2 ± 0.2	37.1 ± 0.3	35.3 ± 1.6	21.5 ± 4.2	18.9 ± 1.4
Hematocrit (%)	41 ± 5	41 ± 6	41 ± 5	25 ± 2	26 ± 3
MABP (mm Hg)	48 ± 4	46 ± 6	42 ± 7	32 ± 9	18 ± 5
CVP (mm Hg)	9 ± 4	9 ± 4	9 ± 4	3 ± 3	4 ± 2
PP (mm Hg)	40 ± 5	38 ± 5	34 ± 5	30 ± 8	15 ± 4
Fio ₂	0.22 ± 0.01	0.22 ± 0.01	0.24 ± 0.05	0.43 ± 0.06	0.47 ± 0.08
Paco ₂ (torr)	44 ± 7	51 ± 8	50 ± 10	43 ± 11	47 ± 13
Petco ₂ (torr)	45 ± 5	46 ± 8	47 ± 12		
Isoflurane (%)	0.23 ± 0.09	0.25 ± 0.09	0.27 ± 0.13	0.49 ± 0.07	0.47 ± 0.08
CPB flow (mL · kg ⁻¹ · min ⁻¹)				195 ± 62	103 ± 47
CPB flow (L · m ⁻² · min ⁻¹)				3.11 ± 0.99	1.65 ± 0.70
Da-rO ₂ cerebral (%)	21 ± 9	26 ± 11	26 ± 10	11 ± 9	12 ± 8
Da-rO ₂ somatic (%)	22 ± 6	30 ± 9	36 ± 17	22 ± 13	31 ± 20
DrSO ₂ cerebral-somatic	-1 ± 6	-3 ± 5	6 ± 10	12 ± 13	20 ± 19
CaO ₂ · PP index	658 ± 106	628 ± 145	573 ± 115	322 ± 94	168 ± 46

Detailed hemodynamic data at each of the 11 identified intervals presented as mean ± SD. Cells with incomplete data are signified by @.

MABP, Mean arterial blood pressure; CVP, central venous pressure; PP, perfusion pressure; AoX, aortic crossclamp; MUF, modified ultrafiltration.

TABLE 4. Effect of CPB on determinants of cerebral rSO₂

Parameter	Pre-CPB coefficient	P value	Post-CPB coefficient	P value	P value for difference
MABP (mm Hg)	0.14 ± 0.05	.002	0.43 ± 0.07	<.001	<.001
SaO ₂ (%)	0.19 ± 0.05	<.001	0.19 ± 0.05	<.001	—
CVP (mm Hg)	-0.14 ± 0.06	.026	-0.14 ± 0.06	.026	—
Hematocrit (%)	-1.39 ± 0.03	<.001	1.14 ± 0.07	<.001	<.001
Paco ₂ (torr)	0.38 ± 0.03	<.001	0.80 ± 0.05	<.001	<.001
Temperature (centigrade)	-0.7 ± 0.11	<.001	-4.65 ± 0.32	<.001	<.001
Isoflurane (%)	-21.5 ± 1.33	<.001	26.1 ± 1.5	<.001	<.001
Constant	106.5 ± 8.3	<.001	106.5 ± 8.3	<.001	—

Table shows the coefficients for parameters that were significantly related to cerebral rSO₂ in a GLS regression model using all pre- and post-CPB data points (N = 2390). Parameter coefficient shows change in rSO₂ for unit change in parameter. Model R² was 0.75 within patient, 0.89 between patients, and 0.79 overall. P value for difference compares pre-CPB with post-CPB coefficient. The effects of MABP, hematocrit, Paco₂, temperature, and isoflurane concentration were significantly altered by CPB.

GLS, Generalized least square.

oxygen delivery. Our findings provide direct evidence for an effect of phenoxybenzamine to preserve perfusion in somatic beds. However, the cerebral circulation may be more vulnerable if splanchnic vasoconstriction is pharmacologically limited. Further study is necessary to quantify the potential effect of phenoxybenzamine to redistribute blood flow away from the cerebral circulation.

The decreased cerebral rSO₂ corresponded in time to a decrease in isoflurane concentration; isoflurane decreases both cerebral oxygen consumption and cerebrovascular resistance, with a resultant increase in cerebral oxygenation.³⁸

Although isoflurane similarly alters autoregulation in all systemic beds by its vasodilatory effects, additional somatic vasodilation may not have occurred in the presence of phenoxybenzamine and milrinone. Thus, the effect of isoflurane to increase cerebral blood flow may be enhanced in the presence of additional pharmacologic reduction in somatic resistance.

Cerebral resistance is increased after hypothermic circulatory arrest and hypothermic CPB.^{1,5,15} This may occur as result of altered dynamic autoregulation from changes in Pco₂ and pH as a result of impaired autoregulation,^{15,16}

RCP	CPB	AoX off	Off CPB	MUF performed	Closure	Overall total
58 ± 10	7 ± 5	59 ± 9	19 ± 5	61 ± 12	11 ± 8	353 ± 39
81 ± 9	76 ± 15	77 ± 10	56 ± 7	52 ± 9	48 ± 7	70 ± 15
41 ± 7	56 ± 17	86 ± 8	79 ± 8	76 ± 7	78 ± 4	67 ± 18
98 ± 4	97 ± 5	95 ± 8	83 ± 3	82 ± 4	81 ± 3	92 ± 8
89 ± 8	76 ± 17	79 ± 13	63 ± 6	61 ± 6	62 ± 4	77 ± 16
19.6 ± 2.9	20.5 ± 3.2	32.1 ± 5.1	35.2 ± 2.2	36.1 ± 0.7	36.5 ± 0.5	30 ± 8
27 ± 3	31 ± 6	32 ± 4	38 ± 4	42 ± 3	45 ± 3	35 ± 8
17 ± 8	27 ± 9	44 ± 8	49 ± 4	53 ± 3	52 ± 3	39 ± 14
4 ± 2	3 ± 2	6 ± 4	10 ± 3	11 ± 2	10 ± 2	7 ± 4
13 ± 8	24 ± 9	38 ± 9	38 ± 6	42 ± 4	42 ± 4	32 ± 12
0.51 ± 0.14	0.53 ± 0.17	0.64 ± 0.22	0.62 ± 0.20	0.51 ± 0.10	0.51 ± 0.14	0.45 ± 0.20
41 ± 8	41 ± 6	39 ± 4	40 ± 3	42 ± 5	46 ± 6	44 ± 9
		15 ± 14	29 ± 5	32 ± 6	35 ± 5	27 ± 19
0.51 ± 0.06	0.49 ± 0.09	0.52 ± 0.08	0.34 ± 0.21	0.28 ± 0.21	0.04 ± 0.07	0.38 ± 0.17
46 ± 22	111 ± 68	214 ± 49				70 ± 9
0.74 ± 0.35	1.75 ± 1.06	3.41 ± 0.77				1.1 ± 1.5
17 ± 11	21 ± 13	17 ± 13	28 ± 8	30 ± 9	33 ± 8	22 ± 12
59 ± 7	44 ± 17	10 ± 10	6 ± 9	6 ± 9	2 ± 2	25 ± 22
42 ± 10	27 ± 22	-8 ± 14	-24 ± 13	-21 ± 13	-27 ± 7	3.1 ± 24
153 ± 100	312 ± 94	529 ± 210	556 ± 145	652 ± 110	679 ± 87	474 ± 224

endothelial dysfunction,^{3,6,8,9} tissue edema, microvascular occlusion, cerebral edema, or other mechanical factors. Perfusion at excessive pressure has been implicated in the cerebrovascular lesion induced by hypothermic CPB.^{12,16,39} Although we targeted a cerebral rSO₂ greater than 75% during RCP, we did not systematically reduce flow rates in patients with cerebral rSO₂ greater than 90%. Although the RCP flows in our patients were in the same range (25-75 mL · kg · min) as reported by others,^{12-14,39,40} excessive flow during RCP may also contribute to the increased cerebrovascular resistance seen after hypothermic CPB. A target of cerebral rSO₂ or jugular bulb saturation greater than 90% during CPB cooling, as in preparation for DHCA,⁵ thus may not be advisable if RCP will be used.

The use of a pH-stat perfusion strategy, although enhancing recovery of cerebral energetics⁴¹ if post-CPB PP is controlled, may contribute to this overperfusion lesion through alteration of cerebral autoregulation.^{16,42} Our results indicate that hypercapnia during pre-CPB management and pH-stat CPB management may reset cerebrovascular autoregulation for CO₂ to a higher level. The enhanced sensitivity of rSO₂ to changes in PP and CO₂ post-CPB (Table 4) is consistent with altered autoregulation.¹⁶ The increased dependency of the cerebral circulation on PP constitutes a physiologic risk in the patient with parallel circulation, because increases in systemic blood pressure and SVR will reduce systemic blood flow.⁴³ On the other hand, CO₂ responsiveness was en-

hanced post-CPB in our patients, indicating that more aggressive use of hypercapnia may improve cerebral rSO₂ even in the face of adequate global oxygen delivery measures.

Our findings emphasize the direct effect of hypothermic CPB and RCP on cerebrovascular resistance^{11,16,41} despite efforts to optimize regional oxygen delivery,⁴⁴ including adjuncts to minimize the endothelial dysfunction from CPB.^{6,8,9} Modifications in perfusion strategy to limit changes in rSO₂ may provide a means to avoid both supply-dependent oxygen conditions⁴⁰ and overperfusion during hypothermia.¹² Use of more moderate hypothermia during CPB and RCP may be justifiable under regional rSO₂ guidance to reduce the alterations in cerebral resistance that result from deep hypothermia. However, the relationship between the effects of DHCA and hypothermic RCP on cerebral blood flow and metabolism, and any subsequent neurologic injury, is still unclear.

The cerebral circulation remains at risk post-CPB despite continuous cerebral perfusion, and neurologic dysfunction may result from postoperative cerebral ischemia independently of CPB techniques. Our results indicate that extended monitoring of brain oxygenation in the immediate postoperative period, when reduced global oxygen delivery is likely to be superimposed on increased cerebrovascular resistance, may reveal further opportunities for hemodynamic interventions to reduce the risk of cerebral ischemia.

Figure 3. The risk of regional saturation less than 50% is shown as the absolute binomial risk (and 95% CI of this risk estimate) at each operative period. Significant somatic desaturation risk occurred after incision and during RCP. The risk of cerebral desaturation progressively increased post-CPB. CPB, Cardiopulmonary bypass; RCP, regional cerebral perfusion.

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Discussion

Dr Gil Wernovsky (Philadelphia, Pa). I found this study to be quite innovative, well designed, and thoughtfully presented. It adds to the growing body of information on the complexities of single ventricle physiology, both before and after CPB.

In the past 15 years, there have been consistent and incremental improvements in the outcomes for children undergoing stage 1 reconstruction, and many things have changed because of our clinical experience and laboratory findings.

These include changes in the methods of preoperative stabilization, the timing of surgery (as you have described in regard to the institution of CPB, including a shift to RCP to avoid DHCA), and the medical management of these patients, with increasing use of postoperative, long-acting vasodilators (such as phenoxybenzamine) and frequent postoperative monitoring. Many of these advances have been pioneered by your group in Milwaukee and others in this room.

On the basis of these studies, there has been a shift at many centers to embrace these new practices. However, before this report, there has been little information presented on the potential neurologic morbidities of two of these changes—RCP and the routine use of phenoxybenzamine.

This article contains a substantial amount of data, but I want to comment on two points and then ask Dr Hoffman three questions.

On the basis of clinical laboratory data from a decade ago, many in our field have moved to avoid circulatory arrest whenever possible. However, a similar detailed assessment of this alternative, RCP in the human, is lacking.

The strategy of RCP for an average of approximately 1 hour in this study, which was preceded by bypass of approximately 45 minutes, and another period of 20 minutes or so after the period of RCP, was associated with estimates of adequate cerebral oxygen delivery by NIRS during the procedure, but significantly lower values after the procedure. As the authors state, this was not solely the result of a postoperative decrease in arterial oxygen saturation, but most likely the result of the increased postoperative cerebral vascular resistance caused by impaired autoregulation.

In addition, the patients received a potent systemic vasodilator (phenoxybenzamine) to minimize ventricular afterload.

Thus, following these combined strategies, a situation has been created that conspires to maximally shunt blood away from the central nervous system, high cerebrovascular resistance and low splanchnic resistance.

Now, this may or may not have clinical implications, and we just do not know yet. It is important to emphasize that NIRS is a surrogate variable for CNS oxygenation, and these observations must be followed up with clinical studies to determine the clinical relevance in the long term.

So I would like to ask the following three questions. First, the authors state that the rate of RCP was titrated during the procedure over a wide range (I believe it was 30-70 mL · kg · min) to achieve a target cerebral saturation of 75% or greater during the procedure.

Did you look at whether the decrease in postoperative cerebral oxygenation was related at all to the absolute rate or pressure of the cerebral blood flow that you used? One could hypothesize that

higher flow rates or pressure during profound hypothermia could cause more endothelial damage and impaired autoregulation.

Second, the model that related regional cerebral saturation to the hemodynamic and bypass parameters was elegant and very interesting. The authors found that RCP, treatment with phenoxybenzamine, and a number of potentially modifiable parameters were related to lower cerebral oxygen saturations, including mean arterial pressure, lower hematocrit, and, I think, higher temperature.

Earlier in the session, Dr Gaynor presented data showing in a more heterogeneous group of children that low diastolic pressure in the coronary intensive care unit was related to an increased incidence of periventricular leukomalacia.

Have you looked at the cerebral oxygenation data in the post-bypass period in terms of diastolic pressure in addition to mean arterial pressure, because this could be particularly low in patients with a diastolic runoff through the shunt?

Finally, if you brought this methodology into the intensive care unit, because, as your group has shown, the real vulnerable period is 6 to 12 hours later, would the cerebral somatic difference persist into the postoperative period?

You and your group are really to be commended for continuing to investigate the implications of the changes in our strategies and to provide us with real data rather than dogma as we move forward.

Dr Hoffman. I agree that spectroscopy measurements are surrogate outcome indicators. I anticipate that with collaboration from colleagues we will be able to answer your most important question: What does this have to do with the relationship between NIRS measures and subsequent neurologic outcome?

With regard to the potential conspiracy of afterload reduction and posthypothermic CPB-increased cerebrovascular resistance, I agree that this is a major caveat and find these data compelling indication for regional saturation monitoring in this population.

The controversial question that we didn't answer, because we have largely abandoned the use of total circulatory arrest, is whether the increase in cerebrovascular resistance after DHCA is greater or less than that observed with the continuous RCP techniques.

Your first question related to the effect of perfusion flow rates on changes in cerebrovascular resistance.

I could not do the analysis that you suggested. In actuality, the perfusion flow rates vary somewhat in response to surgical manipulations on a minute-to-minute basis; our data collection technique did not allow the degree of bypass flow tracking to enable the data analysis required to answer your question, so I would have to think about the methodology.

However, there are good data showing that overperfusion, either by pressure or flow, increases the cerebrovascular lesion seen after deep hypothermic CPB.

We typically, as I think other centers do, try to overflow with a pH-stat cooling strategy to achieve a jugular venous saturation or regional NIRS saturation greater than 90% in anticipation of a potential period of cerebral circulatory arrest. I'm not sure that overperfusion is routinely necessary anymore if NIRS monitoring and continuous cerebral perfusion are used, and it may be that overperfusion is contributing to things.

You wondered about the effects of postoperative diastolic blood pressure and global hemodynamic vulnerability on cerebral perfusion. I certainly agree that the first 6 to 24 postoperative hours is the most vulnerable, critical period for these patients. We have some NIRS data for this time period, but it has not been analyzed.

Because of the anticipated postoperative vulnerability, we hope to use this technology in most postoperative patients. We're currently limited by the number of available devices, as most investigators are.

Dr Carlos Troconis (*Caracas, Venezuela*). Dr Hoffman, are we looking at the tip of an iceberg? Is the problem more profound? Should we look back into the cerebral fetal circulation in hypoplastic left heart syndrome? We will probably find many reports about the incidence of microcephaly with this syndrome and undeveloped microcirculation or high resistance in cerebral flow. Can you make comments on that?

Dr Hoffman. I don't know about confounding anomalies of cerebrovascular anatomy in these patients. We have to deal with what we get. We can't change who comes to us. Preoperative management is important and has obvious implications for regional perfusion, and for most people in the room, there is probably less control over that than the immediate perioperative management.

Certainly the Philadelphia group has detected profound cerebral desaturation in a diverse group of cardiac neonates preoperatively. We didn't see that here. We observed relatively normal arterial-to-brain saturation differences in these patients preoperatively, indicating adequate cerebrovascular development but the potential for wide ranges of cerebral flow related to differences in preoperative management. So the potential certainly exists.

We have the advantage of extensive monitoring during a very intense period of operative intervention, but, as you pointed out, the horse could leave the barn before we get to the operating room, or, as Dr Wernovsky anticipated, sometime afterward.

Dr Richard A. Jonas (*Boston, Mass*). I have a question about the positioning of your oximetrics catheter and wonder if it was truly in the jugular bulb. If it was in the superior vena cava, then you were looking at a mix of jugular venous blood and somatic venous blood. And I very much doubt your speculation that the decrease in saturation is not likely to be caused by reduced cerebral oxygen delivery.

You have proposed that there is an increase in cerebrovascular resistance, which presumably reduces cerebral blood flow and cerebral oxygen delivery.

In fact, what I would suggest is that it is very likely that cerebral blood flow, and therefore oxygen delivery, is decreased related to the position of the shunt. I believe you have created a steal from the cerebral circulation. I am going to be fascinated to see you repeat the study, which I'm sure you will, when you perform the right ventricle to pulmonary artery shunt. I believe this will show quite a different result relative to what you presented today.

I think cerebral blood flow might also be decreased because of increased cerebrovascular resistance related to edema, secondary to your use of regional perfusion.

And incidentally, if I could make a plea, RCP has been the abbreviation for retrograde cerebral perfusion, and we are really going to have to come up a different acronym for RCP, otherwise the literature is going to get very confusing.

In conclusion, until we have some mechanism for monitoring the adequacy of cerebral flow or excessive flow, you run a real risk of causing cerebral edema with regional perfusion. So that might be an additional cause for reduced cerebral blood flow. But I'm interested in the catheter position you used.

Dr Hoffman. The superior vena cava catheters are placed approximately 1 cm above the superior vena cava-right atrial junction, and we attempted to thread this up to the jugular bulb in only 1 patient.

So yes, the superior vena cava saturation is not the same as cerebral NIRS saturation. We do have data relating those two, which will be presented subsequently.

I think your point about the innominate take-off and potential cerebral steal is true, and it will be interesting to compare this cerebral NIRS data with patients who derive pulmonary blood flow from a right ventricular shunt.

The point I was trying to make about oxygen delivery was that whole-body oxygen delivery was not decreased. Therefore I agree

completely that this decreased cerebral saturation we observed post-CPB is likely caused by decreased cerebral oxygen delivery related to regional resistance changes.

Dr Ross M. Ungerleider (*Portland, Ore*). On this basis, have you changed your postoperative strategy at all?

I mean, it seems that the most simple and straightforward thing you could do would be to keep PCO_2 higher as you come off bypass, and PCO_2 is related to cerebral blood flow. And in the face of the runoffs you are talking about (that you can't really do very much about it), it would seem that it might modify this. Have you tried that?

Dr Hoffman. We have used this device in a number of patients, and the effect of CO_2 is immediate and apparent to anybody who bothers to look.

So yes, we have used more hypercapnia, as others have suggested, with the advantage of regional oxygenation data to guide this strategy.