BASIC RESEARCH

Assessment of the relationship between cerebral and splanchnic oxygen saturations measured by near-infrared spectroscopy and direct measurements of systemic haemodynamic variables and oxygen transport after the Norwood procedure

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Objectives: To evaluate the clinical utility of near-infrared spectroscopic (NIRS) monitoring of cerebral (Sco_2) and splanchnic (Sso_2) oxygen saturations for estimation of systemic oxygen transport after the Norwood procedure.
Methods: Sco_2 and Sso_2 were measured with NIRS cerebral and thoracolumbar probes (in humans). Respiratory mass spectrometry was used to measure systemic oxygen consumption ($\dot{V}o_2$). Arterial (Sao_2), superior vena caval (Svo2) and pulmonary venous oxygen saturations were measured at 2 to 4 h intervals to derive pulmonary (Qp) and systemic blood flow (Qs), systemic oxygen delivery (Do_2) and oxygen extraction ratio (ERo_2). Mixed linear regression was used to test correlations. A study of 7 pigs after cardiopulmonary bypass (study 1) was followed by a study of 11 children after the Norwood procedure (study 2).
Results: *Study 1.* Sco_2 moderately correlated with Svo_2, mean arterial pressure, Qs, Do_2 and ERo_2 (slope 0.30, 0.64. 2.30, 0.017 and -32.5, $p < 0.0001$) but not with Sao_2, arterial oxygen pressure (Pao_2), haemoglobin and $\dot{V}o_2$. *Study 2.* Sco_2 correlated well with Svo_2, Sao_2, Pao_2 and mean arterial pressure (slope 0.43, 0.61, 0.99 and 0.52, $p < 0.0001$) but not with haemoglobin (slope 0.24, $p > 0.05$). Sco_2 correlated weakly with $\dot{V}o_2$ (slope -0.07, $p = 0.05$) and moderately with Qs, Do_2 and ERo_2 (slope 3.2, 0.03, -33.2, $p < 0.0001$). Sso_2 showed similar but weaker correlations.
Conclusions: Sco_2 and Sso_2 may reflect the influence of haemodynamic variables and oxygen transport after the Norwood procedure. However, the interpretation of NIRS data, in terms of both absolute values and trends, is difficult to rely on clinically.

Adequate systemic oxygen delivery (Do_2) balanced with systemic oxygen consumption ($\dot{V}o_2$) is crucial to the care of any child requiring intensive care but is particularly difficult to assess after the Norwood procedure.[1] Direct measurement of Do_2 and $\dot{V}o_2$ is most desirable but is rarely performed outside of investigational protocols, and methods often use inappropriate surrogates. Consequently, indirect markers of oxygen balance, such as superior vena caval oxygen saturation (Svo_2), arterial (Sao_2) and venous oxygen saturation difference and blood lactate, are commonly used to estimate the adequacy of Do_2 in these patients.[2–4] The disadvantages of these techniques include the need for repeated blood sampling and the necessarily intermittent nature of the data accrual.

Near-infrared spectroscopy (NIRS) provides a non-invasive, continuous method to monitor regional tissue oxygenation.[5 6] This technique depends on the transparency of biological tissue to light in the infrared region of the spectrum of tissue chromophores, such as haemoglobin and cytochrome aa3. Changes in absorption at several wavelengths can be converted into signals of oxyhaemoglobin, deoxyhaemoglobin and oxidised cytochrome aa3. Furthermore, newer generations of NIRS devices permit quantitative measurement of the ratio of oxyhaemoglobin to total haemoglobin, representing the tissue oxygenation index as an absolute term, independent of a tissue path length factor.[7 8]

NIRS has been extensively evaluated in the cerebral[6 9–13] and splanchnic circulations of newborn infants.[14–16] Because of its relative ease of use, it is also increasingly used in intensive care units as surrogates of Svo_2 and systemic oxygenation.[17–19] Good correlations have been generally reported between splanchnic (Sso_2) or cerebral oxygen saturation (Sco_2) and Svo_2 in various patient groups.[17–19] But there are few data in children with congenital heart disease, either preoperatively or postoperatively. Importantly, all the previous studies have used interindividual single-point assessments in a relatively large population, and NIRS has not been validated against directly measured systemic haemodynamic variables and oxygen transport. Thus, we performed two studies. Study 1 examined pigs after cardiopulmonary bypass (CPB) with normal circulation. Study 2 was a clinical study of neonates during the 72 h

Abbreviations: Cao_2, systemic arterial oxygen contents; CPB, cardiopulmonary bypass; $Cpvo_2$, systemic pulmonary venous oxygen contents, Cvo_2, systemic superior vena caval oxygen contents; Do_2, systemic oxygen delivery; ERo_2, oxygen extraction ratio; NIRS, near-infrared spectroscopy; Pao_2, arterial oxygen pressure; Qp, pulmonary blood flow; Qs, systemic blood flow; Sao_2, arterial oxygen saturation; Sco_2, cerebral oxygen saturation; Sso_2, splanchnic oxygen saturation; Svo_2, superior vena caval oxygen saturation; $\dot{V}o_2$, systemic oxygen consumption after the Norwood procedure. The Norwood group was chosen because it is a particularly challenging subset of patients in which adequate DO_2 is difficult to assess and would be highly advantageous to assess non-invasively. Our original hypothesis was that NIRS would accurately reflect systemic oxygen transport when compared with direct measurements. We therefore obtained continuous NIRS measurements of ScO_2 and SsO_2, and continuous measurement of $\dot{V}O_2$ and, in combination with blood gases, derived repeated and quantitative measurements of pulmonary (Qp) and systemic blood flows (Qs), DO_2 and oxygen extraction ratio (ERO_2). We examined the correlation of ScO_2 and SsO_2 with each of the elements, as well as interindividual and intraindividual variability, to determine the clinical usefulness of NIRS for monitoring systemic haemodynamic function and oxygen transport in patients after the Norwood procedure.

MATERIALS AND METHODS

Study 1

After review and approval by the Institutional Animal Care and Use Committee of the Research Institute in The Hospital for Sick Children, Toronto, Canada, seven Yorkshire pigs weighing 18.5 (1.6) kg were studied. Techniques for anaesthesia, CPB and postoperative management were as described elsewhere.[20] Briefly, the animals were studied during general anaesthesia, with inhaled isoflurene (2%) and intravenous infusion of pancuronium (0.8 μg/kg/min). After median sternotomy the pigs underwent a total of 3 h of CPB at 32℃. After rewarming the animal were weaned from CPB, with continuous infusion of dopamine 5–10 μg/kg/min when necessary.

Study 2

Patients

This study was approved by the institutional Research Ethics Board. Written informed consent was obtained from the parents of 11 children (10 boys, aged from 4 to 92 days, median 7 days) undergoing the Norwood procedure between April and October 2004. Table 1 shows the patients' demographics.

Intraoperative procedures

All patients were intubated with cuffed endotracheal tubes (microcuff Heidelberg paediatric; Microcuff GmbH, Weinheim, Germany). General anaesthesia was maintained with inhaled isoflurane, intravenous fentanyl and pancuronium bromide. Low-flow CPB and selective cerebral perfusion was used in 10 of 11 patients. A standard Norwood procedure with 3.5 mm right modified Blalock–Taussig shunt was used.[21] Phenoxybenzamine 0.25 mg/kg was given at initiation of CPB. Milrinone (100 μg/kg) was given before termination of CPB. Dopamine (5 μg/kg/min) was initiated for the immediate time around cessation of CPB and was subsequently discontinued if the haemodynamic and ventricular functions were good. A pulmonary venous line was inserted into the orifice of the right upper pulmonary vein.

Postoperative management

The central temperature (oesophageal) was maintained at 36–37℃. Postoperative monitoring included arterial, superior vena caval and pulmonary venous pressures and heart rate. Sedation was maintained by a continuous intravenous infusion of morphine and intermittent injections of a muscle relaxant (pancuronium) and lorazepam. Infants were ventilated with volume control and pressure support. Ventilation volume and rate were adjusted to maintain $PaCO_2$ between 40–50 mm Hg. Inotropic agents, vasoactive drugs (milrinone, dopamine, phenoxybenzamine and vasopressin) and volume infusions (5% albumin or blood) were given according to our standard protocol.[22]

Methods of measurement

ScO_2 and SsO_2

NIRS probes consist of a near-infrared light emitter optode and a receiver optode with a distance of 5 cm. In pigs, they were placed on the right and left sides of the forehead. In patients, we chose to replicate previously published techniques of probe placement in this group of patients.[13] Briefly, the probes were placed on the patient's forehead in the midline (ScO_2) and slightly to the right of the midline on the thoracic–lumbar flank (SsO_2). The probes were monitored by a dual-detector device (INVOS 5100A; Somanetics, Troy, Michigan, USA) and recordings were made at 1 min intervals.

$\dot{V}O_2$

$\dot{V}O_2$ was measured continuously with an AMIS2000 mass spectrometer (Innovision A/S, Odense, Denmark). This is a sensitive and accurate method that permits simultaneous measurements of multiple gas fractions. We have described the details elsewhere.[23]

Calculations of systemic haemodynamic variables and oxygen transport

Blood samples were taken from the arterial, superior vena cava and pulmonary vein lines for the measurements of blood gases. Qp and Qs were then calculated by the direct Fick method: $Qp = \dot{V}O_2/(CpvO_2 - CaO_2)$ and $Qs = \dot{V}O_2/(CaO_2 - CvO_2)$, where CaO_2, $CpvO_2$ and CvO_2 indicate systemic

Table 1 Clinical data for the 11 patients

Patient	Age (days)	Weight (kg)	BSA (m^2)	CPB (min)	ACC (min)	Circulatory arrest (min)	Cerebral perfusion (min)	Diagnosis
1	7	3.5	0.23	108	47	12	35	HLHS, AS, MS
2	4	3.7	0.25	151	100	35	53	HLHS, AS, MS
3	7	4	0.26	105	47	3	44	HLHS, AS, MS,
4	16	3.5	0.24	133	39	34	0	HLHS, endocardial fibroelastosis of LV, AS, MS
5	7	4.2	0.27	122	62	3	60	HLHS, AS, MS
6	12	3.5	0.23	165	75	13	59	DILV, TGA
7	6	3.5	0.23	172	82	9	70	HLHS, AA, MA
8	92	4.1	0.26	105	58	30	17	DILV, TGA,
9	7	4	0.25	167	64	17	44	HLHS, AS, MS
10	6	2.9	0.2	142	62	1	62	HLHS, AS, MS
11	9	3.6	0.24	109	50	4	44	HLHS, AS, MS

AA, aortic atresia; AS, aortic stenosis; ACC, aortic cross clamp time; BSA, body surface area; CPB, cardiopulmonary bypass; DILV, double inlet left ventricle; HLHS, hypoplastic left heart syndrome; LV, left ventricle; MA, mitral atresia; MS, mitral stenosis; TGA, transposition of great arteries.

arterial (equal to pulmonary arterial), pulmonary venous and superior vena caval oxygen contents, respectively. DO_2 and ERO_2 were calculated by standard equations: $DO_2 = Qs \times CaO_2$ and $ERO_2 = VO_2/DO_2$. All values in patients were indexed to body surface area and in pigs, to body weight.

Study protocols

The animal study (study 1) was performed during the first 6 h after CPB. Six sets of measurements were obtained at 1, 3 and 6 h, with a 10 min interval between each set of measurements. The clinical study (study 2) was performed during the first 72 h after the patient's arrival in the cardiac intensive care unit. Values of haemodynamic function, oxygen transport and central body temperature were collected at 2 h intervals during the first 24 h and at 4 h intervals during hours 25 through 72. Sampling was avoided if sedation, paralysis and ventilatory or haemodynamic treatment were changed within the prior 15 min.

Data analysis

Data are expressed as mean (SD). Interrelationships between the measures were sought by using mixed linear regression analysis for repeated measures without regard to time. When a significant correlation was found ($p < 0.05$), interindividual differences were further analysed. The extent of the correlation was indicated by the intercept and slope values. All data were analysed with SAS statistical software V.8 (SAS Institute, Inc, Cary, North Carolina, USA).

RESULTS

Pigs

Two pigs died before the end of the 6 h study period. As the NIRS measures of ScO_2 on the two sides were similar, the mean values were used for analysis.

Patients

ScO_2 was obtained in all the patients and SsO_2 in five patients, in three of whom measurements were stopped at 28 and 48 h, respectively, due to technical issues in two patients (patients 1 and 3) and extubation in the other (patient 11). There was no incidence of circulatory collapse or death during the study period. All patients survived to hospital discharge. Extubation was done between 2–16 days (median seven days) after the procedure except in one child who had vocal cord complications. Extubation for that infant was done at 90 days, after a bidirectional cavopulmonary anastomosis (table 1).

Correlations of ScO_2 and SsO_2 with systemic haemodynamic variables and oxygen transport

Table 2 and figs 1–3 detail the results of the correlations of ScO_2 and SsO_2 with haemodynamic variables and oxygen transport in the animal and clinical studies.

Study 1

We obtained 106 sets of measurements in the seven pigs. ScO_2 ranged from 30–59%, SvO_2 from 46.6–86.1%, SaO_2 from

Table 2 Correlations (mixed linear regression) of ScO_2, SsO_2 and SvO_2 with haemodynamic and oxygen transport variables for the entire group and individually in 7 pigs and 11 patients

Dependent variable	Independent variable	Intercept	Group slope	p Value	Range of individual slopes	p Value for interindividual slope difference
Pig data						
ScO_2	SvO_2	28.1	0.30	<0.0001	0.14–0.67	0.0005
	SaO_2	57.6	−0.11	0.82		
	PaO_2	48.0	−0.01	0.60		
	Haemoglobin	52.4	−0.36	0.28		
	MAP	18.6	0.64	<0.0001	−0.71–1.16	<0.0001
	CO	39.7	2.30	<0.0001	1.56–6.42	0.0006
	DO_2	39.0	0.017	<0.0001	0.02–0.07	0.006
	VO_2	39.9	0.01	0.48		
	ERO_2	59.7	−32.5	<0.0001	−65.4–16.4	0.002
Patient data						
ScO_2	SvO_2	28.5	0.43	<0.0001	0.17–0.97	<0.0001
	SaO_2	4.6	0.61	<0.0001	0.14–2.13	0.0001
	PaO_2	12.1	0.99	<0.0001	0.36–1.93	<0.0001
	Haemoglobin	47.5	0.24	0.54		
	MAP	26.0	0.52	<0.0001	0.14–1.11	0.21
	Qp	43.6	3.72	0.001	0.86–6.72	0.03
	Qs	42.1	3.22	<0.0001	0.27–13.64	0.002
	DO_2	42.4	0.03	<0.0001	0.01–0.10	0.001
	VO_2	56.8	−0.07	0.046	−0.33–0.39	<0.0001
	ERO_2	61.1	−33.2	<0.0001	−91.7–−5.3	<0.0001
SsO_2	SvO_2	49.2	0.26	<0.0001	0.11–0.54	0.32
	SaO_2	33.9	0.39	0.0001	Infinite likelihood	
	PaO_2	46.9	0.41	0.002	0.19–0.83	0.22
	Haemoglobin	58.2	0.30	0.48		
	MAP	51.7	0.22	0.03	Infinite likelihood	
	Qp	60.0	1.48	0.23		
	Qs	59.3	1.73	0.03	0.46–10.3	0.28
	DO_2	58.2	0.015	0.004	0.006–0.066	0.11
	VO_2	63.3	−0.007	0.84		
	ERO_2	68.9	−20.3	0.0002	Infinite likelihood	
SvO_2	Qp	47.1	2.49	0.02	−9.3–10.8	0.04
	Qs	40.5	5.82	<0.0001	3.1–11.3	0.02
	DO_2	36.4	0.05	<0.0001	0.032–0.097	0.001
	VO_2	69.7	−0.20	<0.0001	−0.45–0.028	0.008
	ERO_2	77.9	−83.2	<0.0001	−91.9–−69.4	0.18

DO_2, systemic oxygen delivery; ERO_2, oxygen extraction ratio; MAP, mean arterial pressure; PaO_2, arterial oxygen pressure; Qp, pulmonary blood flow; Qs, systemic blood flow; SaO_2, arterial oxygen saturation; ScO_2, cerebral oxygen saturation; SsO_2, splanchnic oxygen saturation; SvO_2, superior vena caval oxygen saturation; VO_2, systemic oxygen consumption.

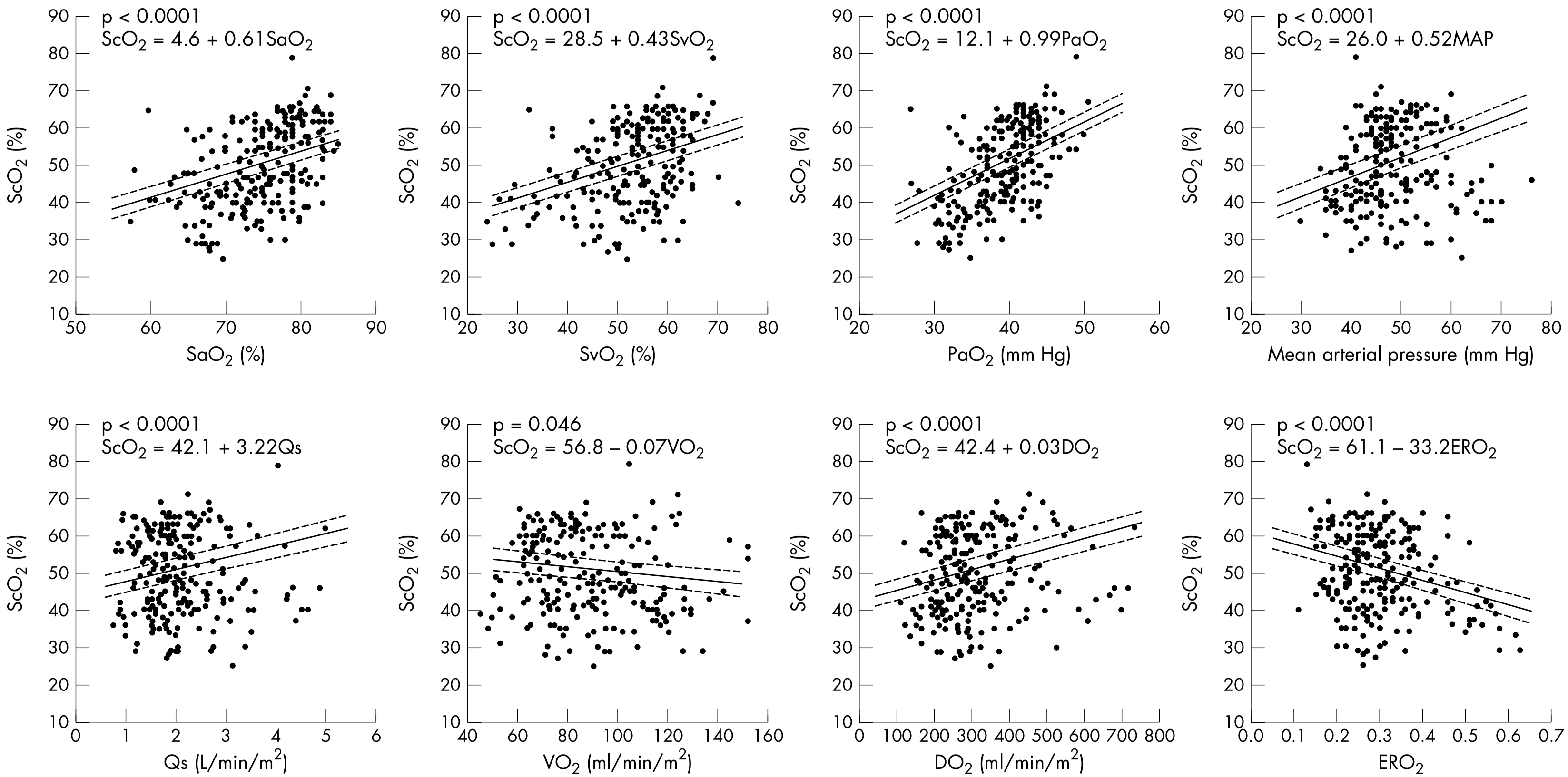

Figure 1 Correlations between cerebral oxygen saturation (ScO_2) and systemic haemodynamic and oxygen transport variables of arterial oxygen saturation (SaO_2), superior vena caval oxygen saturation (SvO_2), arterial partial oxygen pressure (PaO_2), mean arterial pressure (MAP), pulmonary (Qp) and systemic blood flows (Qs), systemic oxygen consumption ($\dot{V}O_2$), oxygen delivery (DO_2) and oxygen extraction ratio (ERO_2) in patients during the first 72 h after arrival in the cardiac intensive care unit.

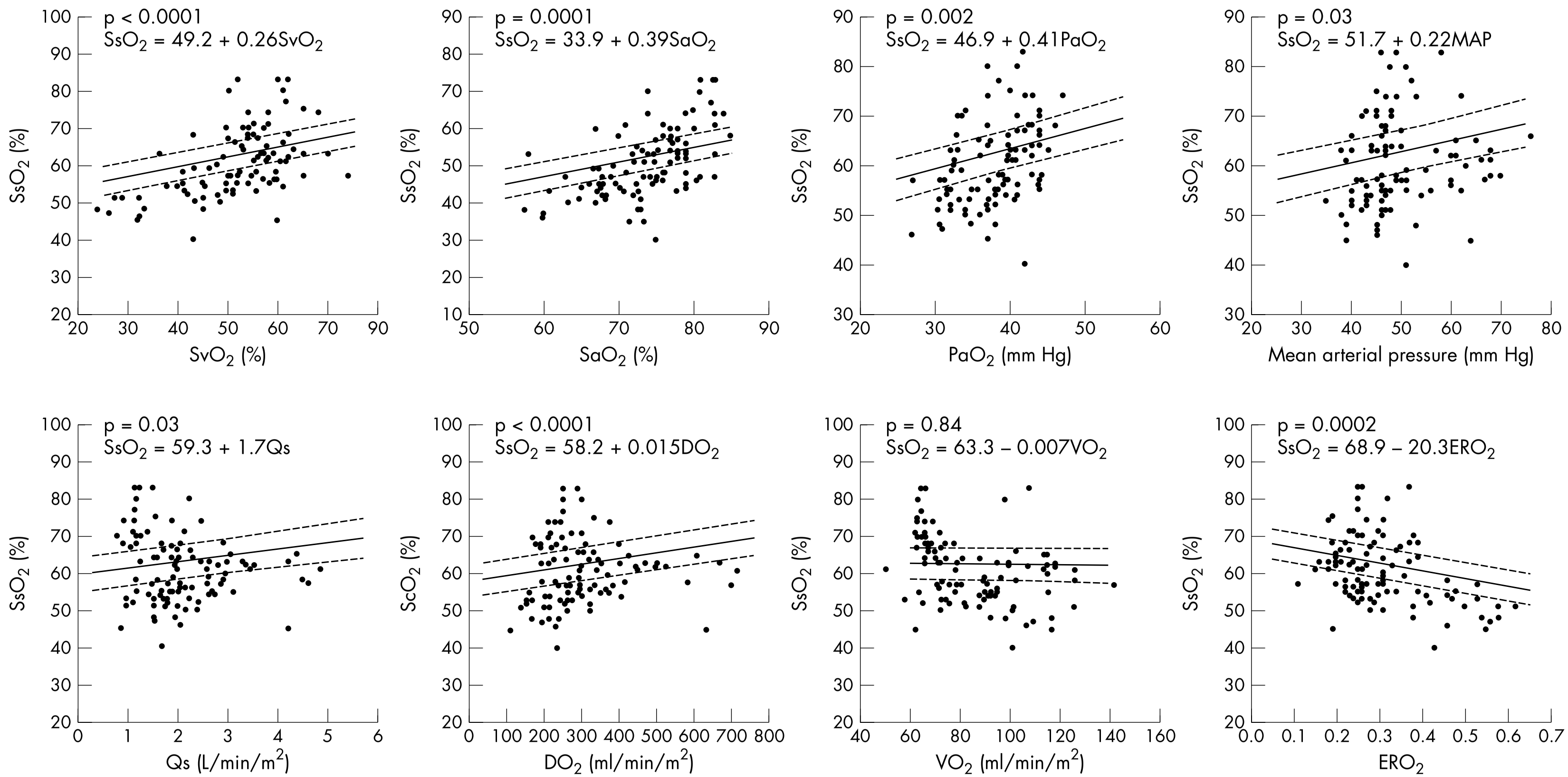

Figure 2 Correlations between splanchnic oxygen saturation (SsO_2) and systemic haemodynamic and oxygen transport variables of superior vena caval oxygen saturation (SvO_2), arterial oxygen saturation (SaO_2), arterial partial oxygen pressure (PaO_2), mean arterial pressure (MAP), pulmonary (Qp) and systemic blood flows (Qs), oxygen delivery (DO_2), systemic oxygen consumption ($\dot{V}O_2$) and oxygen extraction ratio (ERO_2) in patients during the first 72 h after arrival in the cardiac intensive care unit.

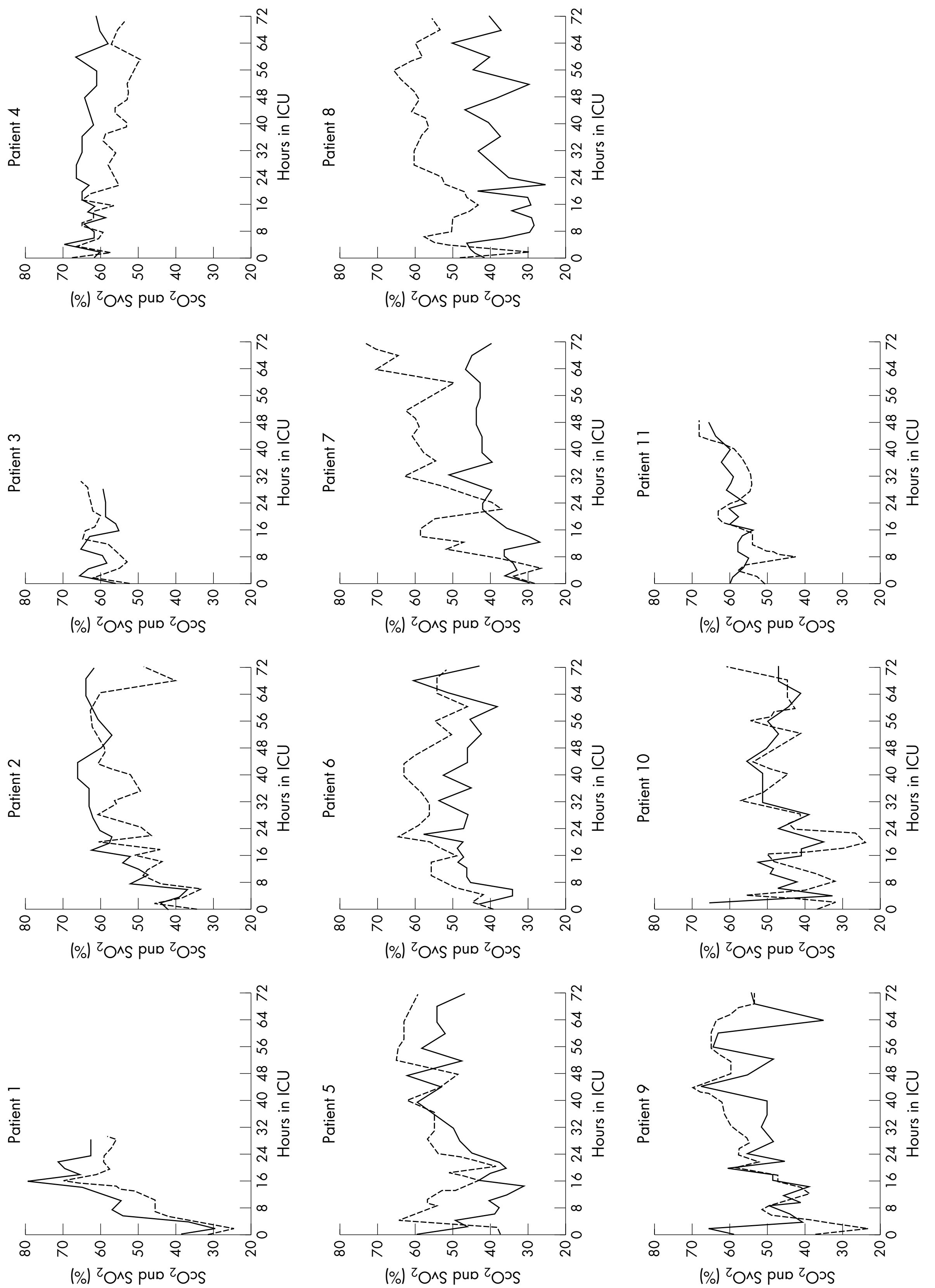

Figure 3 Trends for cerebral oxygen saturation (ScO_2, solid line) and superior vena caval oxygen saturation (SvO_2, dotted line) for each of the 11 patients during the first 72 h after arrival in the cardiac intensive care unit (ICU).

91.1–100% and arterial oxygen pressure (Pa_{O_2}) from 60–237 mm Hg.

Sc_{O_2} was not correlated with Sa_{O_2} (slope −0.11, p = 0.82), Pa_{O_2} (slope −0.01, p = 0.60) or haemoglobin (slope −0.36, p = 0.28). Sc_{O_2} was correlated with Sv_{O_2} (slope 0.30, $p < 0.0001$) but with large interindividual variations (slope 0.14 to 0.67, p = 0.0005). Sc_{O_2} was also correlated with mean arterial pressure (slope 0.64, $p < 0.0001$) with large interindividual variations (slope −0.71 to 1.16, $p < 0.0001$) and moderately correlated with Qs, D_{O_2} and ER_{O_2} (slope 2.3, 0.017, −32.5, respectively, $p < 0.0001$ for all) with significant interindividual variations (table 2). Sc_{O_2} was not correlated with $\dot{V}_{O_2}$ (slope 0.01, p = 0.48) (table 2).

Study 2

We obtained 247 sets of measurements for Sc_{O_2} and 103 sets for Ss_{O_2}. For the entire group, Sc_{O_2} ranged from 25–79% and Ss_{O_2} from 40–83%; Sv_{O_2} ranged from 24–74%, Sa_{O_2} from 55–86% and Pa_{O_2} from 25.0–50.6 mm Hg.

Unlike in pigs, in patients Sc_{O_2} was correlated with both Sa_{O_2} and Pa_{O_2} (slope 0.61 and 0.99, respectively, $p < 0.0001$ for both) but with large interindividual variations (slope 0.17–0.97 for Sa_{O_2}; 0.36–1.93 for Pa_{O_2}; $p < 0.0001$ for both). Sc_{O_2} was not correlated with haemoglobin (slope 0.24, p = 0.54). Similarly to the data in pigs, Sc_{O_2} was correlated with Sv_{O_2} (slope 0.43, $p < 0.0001$) but with large interindividual variations (slope 0.17–0.97 for Sv_{O_2}; 0.14–2.13 for Sa_{O_2}; $p < 0.0001$ for both). It was also correlated with mean arterial pressure (slope 0.52, $p < 0.0001$) but, interestingly, interindividual variations were insignificant (slope 0.14–1.11, p = 0.21). Sc_{O_2} was weakly correlated with $\dot{V}_{O_2}$ (slope −0.07, p = 0.05) and moderately correlated with Qp, Qs, D_{O_2} and ER_{O_2} (slope 3.1, 3.2, 0.03, −33.2, respectively, $p < 0.0001$ for all except for Qp, p = 0.001) with significant interindividual variations (table 2, fig 1).

Ss_{O_2} was similarly, although more weakly, correlated with haemodynamic and oxygen transport variables, with the coefficients being about half those with Sc_{O_2}. Interindividual variations were also large, although they did not achieve significance, probably due to the smaller sample size. Ss_{O_2} was not correlated with $\dot{V}_{O_2}$ (slope −0.007, p = 0.84) (table 2, fig 2).

The correlations of Sc_{O_2} and Ss_{O_2} with Qs, D_{O_2}, $\dot{V}_{O_2}$ and ER_{O_2} were much weaker than those of Sv_{O_2} with these variables (table 2).

Lastly, examples of individual patient plots (fig 3) of the NIRS and the directly derived data show notably variable intraindividual relationships that changed with time and at individual time points.

DISCUSSION

This is the first comprehensive investigation of the relationship between NIRS measurements of Sc_{O_2} and Ss_{O_2}, as a non-invasive clinical haemodynamic monitor of systemic oxygenation, and the directly measured systemic haemodynamic and oxygen transport variables. We assessed the utility of NIRS after CPB in two different circulations—namely, the normal biventricular circulation in an animal model and the more complex parallel circulation, with residual arterial desaturation, in children after the Norwood procedure. Our data showed that, in patients with varied and relatively low values of Pa_{O_2} and Sa_{O_2}, Sc_{O_2} was closely correlated with both of these values, but not in pigs with fully saturated arterial oxygenation. Most important, Sc_{O_2} was, similarly in both groups, closely correlated with mean arterial pressure, loosely correlated with Sv_{O_2}, Sa_{O_2}, Qp, Qs, D_{O_2} and ER_{O_2}, poorly correlated with $\dot{V}_{O_2}$ and not significantly correlated with either haemoglobin or Qp. Whereas the correlation between NIRS-derived oxygen saturations and many of the directly measured indices were highly significant, the relatively loose correlations at an absolute level, combined with wide interindividual variability, cast doubt on the potential clinical utility of such measurements.

Previous studies have evaluated single-point comparisons between Sc_{O_2} or Ss_{O_2} and Sv_{O_2} in individual children in a larger population.[17–19] Good correlations have generally been reported, but large interindividual differences are apparent, even in these studies. Our study was the first to evaluate NIRS in the setting of postoperative complex congenital heart disease, comparing the non-invasive data with directly measured indices of oxygen transport during the first three days after the Norwood procedure, and confirmed the previous findings. Our data showed that an increase in Sc_{O_2} or Ss_{O_2} of 1% explained 0.43% or 0.26%, respectively, of the increase in Sv_{O_2}. However, interindividual variations were large, ranging from 0.17–0.97% for Sc_{O_2} and from 0.11–0.54% for Ss_{O_2}. Furthermore, the individual trends shown in fig 3 show an inconsistent relationship between Sc_{O_2} and Sv_{O_2} throughout the measurement period. Although differences in absolute values and even wide interindividual variability may be tolerated if NIRS is used for trend analysis, the second issue of an unreliable intraindividual relationship between NIRS and Sv_{O_2}, for example, casts doubt on its potential utility as a precise tool for monitoring haemodynamic trends.

This is, however, not surprising. NIRS measures the equilibrium of oxyhaemoglobin and deoxyhaemoglobin in a mixture of veins, arteries and capillaries in the underlying tissue and reflects a regional state of oxygenation. Although NIRS has been extensively used to monitor cerebral and splanchnic oxygenation in various clinical situations including during CPB, deep hypothermic circulatory arrest[13 24] and in other high risk newborns,[6 15 19 25] and has been found to be helpful in predicting cerebrovascular dysfunction[24 25] and splanchnic ischaemia,[15] it has rarely been rigorously examined for its validity, particularly in the setting of complex parallel circulations such as those that exist after the Norwood procedure. However, whereas the venous portion predominantly determines NIRS measurement of the underlying tissue oxygenation, direct measurements, such as jugular bulb oxygen saturation and hepatic venous oxygen saturation, are well known to correlate variably with NIRS measurement of that organ.[11 14] This may be a particular problem in children with complex congenital heart disease, where the contribution of the venous portion has been shown to range from 60–100% of Sc_{O_2} with varied systemic oxygen saturation as seen in our patients after the Norwood procedure.[12] It must also be remembered that NIRS measures oxygenation in a small part of the target organ, and the regional venous oxygen saturation reflects the balance of oxygen delivery and consumption of the whole organ. Sv_{O_2}, conversely, reflects the balance of systemic oxygen transport, presumably explaining the discrepancy that Yeh T Jr *et al*[26] observed between jugular bulb oxygen saturation and Sv_{O_2} in children undergoing CPB. Extracerebral tissue factors, such as ischaemia,[27] oedema and the location of the probe,[28] may also affect the robustness of NIRS signals.

Limitations

The superior vena cava was used to measure systemic venous saturation for the calculations of Qs and D_{O_2}. This measure does not account for potential differences in inferior vena cava saturation[29 30] and may at least partly account for the poorer correlations with Ss_{O_2}. Conversely, it could be argued that, by sampling upper body venous saturation, we have a more representative measurement, incorporating cerebral blood flow. Nonetheless, jugular bulb saturation would have been better in this regard but was not appropriate in this clinical protocol.[31]

Furthermore, we chose the usual position (posterior flank) to assess Sso_2 by NIRS. Recently, Fortune *et al*[15] used the site of just below the umbilicus to monitor splanchnic oxygenation and found a sensitivity of 90% to detect splanchnic ischaemia in neonates during apnoeic episodes. Sso_2 measured in such a way may reflect more Do_2 and $\dot{V}o_2$, and thus warrants further investigation in children with heart disease.

Conclusions

NIRS measurement of Sco_2 and Sso_2 reflects the changes in haemodynamic variables and oxygen transport during the early postoperative period after the Norwood procedure. However, large interindividual differences and intraindividual temporal variability make interpretation difficult and may limit the utility of NIRS as a continuous monitor of systemic haemodynamic function and oxygen transport in critically ill patients.

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