Cerebral oxygenation during paediatric cardiac surgery: identification of vulnerable periods using near infrared spectroscopy


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Abstract

Objective: Neurologic sequelae remain a well recognised complication of paediatric cardiac surgery. Monitoring of cerebral oxygenation may be a useful technique for identifying vulnerable periods for the development of neurologic injury. We sought to measure regional cerebral oxygenation in children undergoing cardiac surgery using near infrared spectroscopy to ascertain such vulnerable periods. Methods: Observational study of 18 children (median age 1.3 years) undergoing cardiac surgery (17 with cardiopulmonary bypass, 8 with circulatory arrest). Regional cerebral oxygenation was monitored using the INVOS 3100 cerebral oximeter and related to haemodynamic parameters at each stage of the procedure. Results: Prior to the onset of bypass, 10 patients had a decrease in regional cerebral oxygenation of E15% points, reaching an absolute haemoglobin saturation less than 35% in 5 cases. The most common cause was handling and dissection around the heart prior to and during caval cannulation. With institution of bypass, regional cerebral oxygenation increased by a mean 18% points to a mean maximum of 75%. During circulatory arrest regional cerebral oxygenation decreased with rate of decay influenced by temperature at onset of arrest (0.25%/min at < 20°C; 2%/min at > 20°C). Reperfusion caused an immediate increase in regional cerebral oxygenation followed by a decrease during rewarming. Discontinuation of bypass caused a precipitous decrease in regional cerebral oxygenation in 5 patients, reaching less than 50% in 3 patients. Conclusions: These observations suggest that the pre- and early post-bypass periods are vulnerable times for provision of adequate cerebral oxygenation. Near infrared spectroscopy is a promising tool for monitoring O₂ supply/demand relationships especially during circulatory arrest. © 1998 Elsevier Science B.V. All rights reserved.

Keywords: Cerebral oximetry; Children; Cardiopulmonary bypass; Deep hypothermic circulatory arrest; Near infrared spectroscopy

1. Introduction

In recent decades there has been a marked reduction in surgical mortality for many forms of congenital heart disease. Neurologic sequelae remain, however, a well recognised and potentially devastating complication of paediatric cardiac surgery [1,2]. These complications appear to occur more commonly after deep hypothermic circulatory arrest. Although the aetiology of neurologic injury is almost certainly multifactorial [2,3], imbalance between cerebral oxygen supply and demand is likely to play an important role [3]. The ability to monitor cerebral oxygenation during, and immediately after, cardiac surgery offers the possibility for predicting and preventing neurological injury. We have previously shown that near infrared spectroscopy is a simple, noninvasive tool for monitoring cerebral oxygenation in infants and children [4]. In this study we have used near infrared spectroscopy to help identify periods of mismatch between cerebral oxygen supply and demand during paediatric cardiac surgery, including deep hypothermic circulatory arrest.
containing tripeprazine (3mg:volving premedication with 0.5 ml:(total of 14 episodes of circulatory arrest).pass including eight with hypothermic circulatory arrest procedures were carried out on cardiopulmonary by-and procedures are shown in Table 1. A total of 17 circumference 46 cm (range 34–55 cm). Their diagnoses dian age was 1.2 years (range 2 weeks to 12.5 years), tored during cardiac surgery. There were 11 boys. Me-

2. Materials and methods

Following institutional approval (September 1993) and informed parental consent, 18 children were moni-
tored during cardiac surgery. There were 11 boys. Median age was 1.2 years (range 2 weeks to 12.5 years), weight 8.4 kg (range 3–52 kg) and occipito-frontal head circumference 46 cm (range 34–55 cm). Their diagnoses and procedures are shown in Table 1. A total of 17 procedures were carried out on cardiopulmonary bypass including eight with hypothermic circulatory arrest (total of 14 episodes of circulatory arrest).

General anaesthesia used a standard technique involving premedication with 0.5 ml/kg of a mixture containing tripeprazine (3mg/ml), atropine (60 μg/ml), and morphine (1mg/ml), intravenous induction with 3–5 mg/kg thiopentone and paralysis with vecuronium or pancuronium. Maintenance was with 0.5–1% isoflurane in nitrous oxide and oxygen. Morphine or fentanyl were infused for analgesia. Ventilation was adjusted to maintain normocarbia, as assessed by end-
tidal CO₂ sampling and intermittent arterial blood gas analysis. Cardiopulmonary bypass utilised a non-pul-
satile roller pump (Stockert, Munich, Germany) and membrane oxygenator (Sorin, Mirandola, Italy). Standard pump flow rates were 100–120 ml/kg per min although in selected patients periods of low flow bypass were employed. α-Stat management of acid–base status was used during cardiopulmonary bypass.

Near infrared monitoring utilised the INVOS 3100 cerebral oximeter (Somanetics, Troy, MI). This mea-
sures intracerebral haemoglobin oxygen saturation by spectroscopy of reflected near infrared light. Since the cerebral microcirculation contains arterial, venous and capillary components, the regional cerebral oxygen saturation (rSO₂) represents a weighted average assuming the venous component to be predominant (estimated as 75% by volume) [5]. The sensor consists of a near infrared light transmitter and two photodetectors (optodes) positioned 3 and 4 cm from the infrared source. This arrangement allows spatial resolution since the optode nearest the light source receives a signal from the light that has travelled in an arc through superficial tissues whilst the more distant optode receives light that has passed through superficial and deeper tissues. ‘Subtraction’ processing of the two signals allows calculation of oxygen saturation of haemoglobin from cerebral tissue whilst minimising ‘contamination’ from superficial (extracerebral) sources [6,7]. The cerebral oximeter displays the regional cere-
bral haemoglobin oxygen saturation. The light source and optodes are contained within a flexible adhesive pad which was placed over the forehead lateral to the midline to avoid the superior sagittal sinus and at least 2 cm above the eyebrows to avoid the frontal sinus. Monitoring was commenced shortly after induction of general anaesthesia and continued throughout the procedure until the child left the operating room. Eleven patients had retrograde cannulation of the internal jugular vein for comparison of regional cerebral oxygen saturation (rSO₂) as measured by near infrared spec-
troscopy with jugular bulb oxygen saturations. For

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AR, aortic regurgitation; AS, aortic stenosis; ASD, atrial septal defect; AVR, aortic valve replacement; AVSD, atriointerventricular septal defect; CPB, cardiopulmonary bypass; DHCA, deep hypothermic circulatory arrest (≤20°C); MR, mitral regurgitation; MV, mitral valve; MVR, mitral valve replacement; PA, pulmonary artery; PA VSD, pulmonary atresia with ventricular septal defect; T, tetralogy; TAPVD, total anomalous pulmonary venous drainage; TGA, transposition of the great arteries; VSD, ventricular septal defect; and WCA, ‘warm’ circulatory arrest (>20°C).
individual patients, correlations between rSO2 and jugular bulb oxygen saturations were excellent; these results have been previously reported [4].

The following variables were monitored approximately every 5 min during surgery, or more frequently when events were changing rapidly: rSO2, systemic arterial saturation, heart rate, mean blood pressure, central venous pressure, cardiopulmonary bypass flow rates and nasopharyngeal and oesophageal temperatures.

The rSO2 profiles were described in five distinct periods: prebypass, bypass, circulatory arrest, rewarming/reperfusion and post-bypass. In the bypass period, the relationship between rSO2 at 20°C and rate of cooling was explored. During circulatory arrest, rates of decline in rSO2 were noted and the relationship between rate of decay of rSO2 was correlated with temperature at onset of arrest, rSO2 at onset of arrest and rate of bypass cooling. During the rewarming period the relationship between rSO2 and nasopharyngeal temperature was examined.

3. Results

There was one operative death (Case 10) in a child undergoing late arterial switch for right ventricular failure after prior Mustard procedure. There were no neurologic complications either acutely nor during at least 1 year of follow-up. The procedures were divided into the following stages to aid analysis: pre-bypass, bypass, circulatory arrest, reperfusion/rewarming and post-bypass.

3.1. Pre-bypass

A total of 18 patients were analysed. See Table 1. Patient 5 was included because, although non-bypass, the intention at the time of induction of anaesthesia had been to undergo complete correction using cardiopulmonary bypass. Ten of the 18 patients had a decrease in rSO2 ≥ 15% points for a minimum of 3 min. In 5 patients rSO2 reached < 35% (20, 27, 28, 28 and 33%). In 3 cases this fall was associated with handling and dissection of the heart (especially the superior vena cava) prior to going onto cardiopulmonary bypass (Case 15, Fig. 1a). In one patient (Case 8), decrease in rSO2 was due to anaesthetic-induced cyanotic spell, and in another (Case 5) it was associated with bradycardia and cardiovascular collapse during sternotomy. Under moderate hypothermic conditions (where cerebral autoregulation would be expected to be preserved), near infrared spectroscopy confirmed that marked swings in blood pressure were not associated with swings of rSO2 (Fig. 1b).

3.2. Bypass

Altogether, 17 patients underwent 23 periods of cardiopulmonary bypass. The first period of bypass raised the rSO2 by a mean 18% points (S.D. 9) to a mean maximum of 75% (S.D. 8, \( P < 0.01 \)). During cardiopulmonary bypass, patients were cooled to between 13 and 32°C with 7 patients being cooled to less than 20°C (Fig. 1a–c). The mean bypass cooling time to achieve a nasopharyngeal temperature of 20°C in these 7 patients was 14 min. rSO2 at the time that this temperature was reached varied inversely with rate of cooling

\[
\text{rSO}_2 - 20°C = -20.5 \times \text{rate of cooling (°C/min)} + 97.4
\]

\( r = -0.77, \ P = 0.04. \)

3.3. Circulatory arrest

A total of 8 patients underwent 14 episodes of circulatory arrest. For 11 of these episodes, the temperature at the onset of circulatory arrest was less than 20°C. rSO2 at onset of these 11 episodes ranged from 62–94% (mean 82%). Mean arrest time for this group was 45 min (S.D. 13, range 23–61) and mean rate of fall of rSO2 during circulatory arrest was 0.25% points/min (S.D. 0.16), though the fall was not linear. The decay in rSO2 during circulatory arrest is demonstrated in Fig. 1c and Fig. 2. In 3 patients (three episodes), circulatory arrest was commenced at a temperature greater than 20°C (21, 22 and 26°C). The period of circulatory arrest ranged from 7–27 min and the rate of decrease in rSO2 was 6–11 times higher (2.0% points/min, range 1.5–2.7) than for episodes of deep hypothermic circulatory arrest (< 20°C) (Fig. 2).

Rate of decay of rSO2 was correlated with temperature at onset of arrest

\[
\text{rate decrease in rSO}_2 = 0.19 \times \text{temperature (°C)} - 2.5
\]

\( r = 0.84, \ P = 0.0002, \ n = 14, \) but not with rSO2 at onset of arrest \( (r = 0.40, \ P = 0.16), \) nor rate of cooling \( (r = 0.4, \ P = 0.17). \) The mean rSO2 at the end of circulatory arrest was 66% (S.D. 16, range 30–88).

3.4. Rewarming/reperfusion

Three clinical scenarios were encountered: (a) no circulatory arrest, rewarmin on cardiopulmonary bypass—for patients not undergoing circulatory arrest \( (n = 9), \) rewarmin resulted in a universal decrease in rSO2 (mean 12% points, S.D. 15, range 2–44) (e.g. Fig. 1a,b). There was an inverse relationship during rewarmin between the rSO2 and nasopharyngeal temperature

\[
\text{rSO}_2 = -2.9 \times \text{nasopharyngeal temperature} + 148
\]

\( r = -0.65, \ P = 0.002). \) (b) Circulatory arrest, reperfu-
Fig. 1. Examples of regional cerebral oxygen saturation (rSO$_2$) profiles. rSO$_2$, mean systemic blood pressure (BP) and nasopharyngeal temperature (NP) are shown on the y-axis and time elapsed from the onset of induction of anaesthesia is shown on the x-axis. (a) Case 15: 6-year-old with ventricular dysfunction and severe mixed aortic and mitral valve disease undergoing double valve replacement. Handling and dissection of the heart (especially the superior vena cava) on the background of anaesthetic induced exacerbation of ventricular dysfunction in the prebypass period results in severe cerebral desaturation. Rewarming during weaning from cardiopulmonary bypass (CPB) results in further profound fall in rSO$_2$. (b) Case 13: 4-year-old undergoing atrial septal defect repair using cardiopulmonary bypass (CPB) and moderate hypothermia. Marked stability in rSO$_2$ is noted despite large swings in systemic blood pressure, demonstrating cerebral autoregulation. (c) Case 2: 1-month-old undergoing ventricular septal defect closure using cardiopulmonary bypass (CPB) and circulatory arrest (CA). Curvilinear decay in rSO$_2$ is noted during circulatory arrest. Immediate restoration of rSO$_2$ occurs during reperfusion followed by gradual decrease during rewarming.

3.5. Post-bypass

Discontinuation of cardiopulmonary bypass was associated with a precipitous fall in rSO$_2$ in 5 patients. In 3 cases the rSO$_2$ fell to less than 50%. In all cases this fall appeared to be due to poor cardiac output with low systemic arterial pressure in the presence of normothermia or during rapid rewarming.

3.6. Non-bypass patient

Case 5 developed cardiovascular collapse and bradycardia during sternotomy for planned arterial switch procedure with ventricular septal defect closure. De-
Fig. 2. Regional cerebral oxygen saturation (rSO$_2$) profiles during circulatory arrest. Slow decay in rSO$_2$ during four separate episodes of deep hypothermic circulatory arrest (13–16°C) in Case 7 are contrasted with a more rapid decline in 3 patients undergoing circulatory arrest at temperatures above 20°C.

Despite only a modest fall in systemic saturations, the rSO$_2$ fell abruptly with fall in systemic blood pressure and bradycardia. It is of interest to note that a gradual decline in rSO$_2$ commencing soon after induction of anaesthesia preceded cardiovascular collapse. At this time clinical and haemodynamic monitoring had not suggested any compromise of cardiac output. Prompt resuscitation with internal cardiac massage and administration of adrenaline and atropine resulted in rapid recovery in rSO$_2$.

4. Discussion

Although neurologic injury during paediatric cardiac surgery is almost certainly multifactorial in origin [1–3], it is self-evident that cerebral oxygen supply must meet metabolic demands. It is known that cooling reduces cerebral metabolism and that the cerebral metabolic rate falls exponentially with temperature reduction [3]. By contrast cerebral blood flow falls linearly with cooling [3]. Thus, induced hypothermia should result in progressive rise in the ratio of cerebral blood flow to metabolism. It might, therefore, be expected that cardiopulmonary bypass under hypothermic conditions should not, in itself, result in inadequate oxygen delivery.

Greeley and colleagues have recently emphasised the need for study of all periods during paediatric cardiac surgery and not just the period of circulatory arrest [3]. The ‘preparation period’ prior to onset of circulatory arrest may be a vital determinant of how well circulatory arrest is tolerated. Important factors at this time might include method and rate of cooling, as well as strategy for acid-base regulation. The early post-bypass and postoperative periods might also be times of increased vulnerability for hypoxic-ischemic damage [3,8,9]. Following profound hypothermia, cerebral autoregulation may remain absent or impaired for several hours. If cardiac output is inadequate following discontinuation of cardiopulmonary bypass, then cerebral oxygen delivery may become insufficient to meet metabolic demands during and after rewarming.

It is clear that any tool which can assess the cerebral oxygen supply/demand relationship may have major potential for identifying the most vulnerable periods for ischaemic damage during and after paediatric cardiac surgery. Tools for the assessment of cerebral blood flow (e.g. xenon clearance) and metabolism during cardiopulmonary bypass have been well described but are cumbersome and not well suited for continuous monitoring in a clinical setting [10]. Monitoring of jugular bulb venous saturation has been used as a technique for monitoring cerebral oxygen balance during cardiac surgery in both children and adults [11–14]. Although the technique is invasive, it has been suggested that monitoring of jugular bulb oxygen saturation may help predict inadequate cerebral oxygen delivery during rewarming after cardiac surgery [12]. Kuwabara and colleagues demonstrated the potential use of continuous jugular bulb oxygen saturation monitoring during selective cerebral perfusion for aortic arch replacement [13].

These observations suggested that a simple, noninvasive tool capable of providing similar information to
jugular bulb oxygen saturation measurements might be a major advance in the perioperative monitoring of cardiac surgical patients. We have previously shown that rSO₂ measured by near infrared spectroscopy correlates very closely with simultaneous jugular bulb venous saturations in individual children [4]. We therefore postulated that near infrared spectroscopy would prove to be a useful technique for identifying periods of increased vulnerability for ischaemic neurologic damage during paediatric cardiac surgery. Furthermore we recognised this as a potential tool for monitoring cerebral oxygenation during circulatory arrest when jugular bulb oxygen saturation monitoring is not applicable.

Several observations in this preliminary report are noteworthy. Firstly, marked cerebral desaturation was observed in several patients during the pre-bypass period. This was most commonly associated with dissection around the superior vena cava and caval cannulation. Whether this insult is sufficient to cause cerebral damage remains unclear, but it offers the option of adjusting surgical technique if profound falls in rSO₂ occur. Furthermore, we identified one case in which rSO₂ monitoring (in retrospect) offered early clues to impending cardiovascular collapse prior to change in haemodynamic parameters. In almost all patients, institution of cardiopulmonary bypass with simultaneous cooling resulted in a rise in rSO₂, as might be expected based on our understanding of the cerebral response to cooling [3]. These observations are also consistent with changes in jugular bulb saturation during hypothermic cardiopulmonary bypass [12,14]. Since standard temperature monitoring during cardiopulmonary bypass does not reliably predict efficiency of cerebral cooling [11], near infrared spectroscopy may prove to be a useful technique for assessing adequacy of cerebral protection in individuals as well as a suitable research tool for comparing differing strategies of cerebral protection (e.g. differing cooling techniques, optimal management of acid–base status).

Cerebral oxygenation cannot be assessed during circulatory arrest by jugular bulb monitoring. Although magnetic resonance spectroscopy can provide information about high energy phosphorus compounds in the brain [15,16], bedside monitoring is unavailable and therefore this technique is not suitable for clinical use during cardiac surgery. Near infrared spectroscopy is thus a very attractive tool for continuous monitoring during deep hypothermic circulatory arrest as the technique is noninvasive, safe and simple to perform in the operating room [17–19]. We observed a universal fall in rSO₂ during circulatory arrest with the rate of decay correlating with core temperature at the onset of circulatory arrest. Fall in rSO₂ was generally exponential and the trends observed are similar to those reported by others [17–19]. The rate of decrease in rSO₂ under deep hypothermic circulatory arrest was slightly slower than that reported by Ausman and colleagues [17] and by Silvay and co-workers [19] in adult patients. These observations might reflect the relative ease of achieving homogenous cooling of the brain in infants and young children as compared to adults. Alternatively, cerebral O₂ utilisation during deep hypothermic circulatory arrest may be intrinsically lower at younger ages [20]. At temperatures below 20°C, circulatory arrest rarely caused a fall in rSO₂ of more than 15%. This suggests that deep hypothermic circulatory arrest for periods of up to 1 h probably allows adequate oxygen supply to meet metabolic demands providing efficient brain cooling has been achieved prior to deep hypothermic circulatory arrest. It is of particular interest to note that decay in rSO₂ was 6–11 times higher if core temperature at the onset of circulatory arrest exceeded 20°C. At
the present time it is impossible to predict the ‘safe period’ for circulatory arrest in any individual. It seems possible that assessment of cerebral oxygenation at the onset of deep hypothermic circulatory arrest by near infrared spectroscopy, combined with continuous monitoring during deep hypothermic circulatory arrest, might help predict when a patient is at risk for the development of hypoxic-ischaemic cerebral injury.

Reperfusion following circulatory arrest caused rSO₂ to rise. When reperfusion was associated with rewarming there was an initial rise and then a fall in the rSO₂. These patterns are very similar to those described by Kurth and colleagues [18] and are consistent with our understanding of the physiology of cardiopulmonary bypass. The initial rise presumably reflects the reinstatement of cerebral oxygen delivery and the subsequent fall during rewarming, the increased cerebral oxygen demand of enhanced cerebral metabolism. Obvious targets for further study with this technique include the physiological effects of repeated episodes of circulatory arrest interspersed with recirculation, and the comparison of deep hypothermic circulatory arrest with low-flow cardiopulmonary bypass. It should be possible to establish the minimum flow rate required to maintain stable rSO₂ in any individual setting.

Rewarming on cardiopulmonary bypass (not following circulatory arrest) caused rSO₂ to fall 2.9% per degree rewarmed. Nakajima observed an almost identical relationship whilst monitoring jugular venous oxygen saturation by continuous oximetry in adult patients. (3% fall in jugular bulb oxygen saturation per degree rise in temperature) [12]. Our observations support the contention of other authors [3,8,9], that the period early after discontinuation of cardiopulmonary bypass may be a vulnerable period for provision of adequate oxygen delivery to meet cerebral metabolic demands. During this period cerebral metabolic rate will rise but ability to increase oxygen delivery may be impaired. This may be particularly problematic following deep hypothermic circulatory arrest when cerebral autoregulation may remain impaired for a variable period of time after discontinuation of cardiopulmonary bypass. In the presence of poor ventricular function and systemic hypotension, cerebral oxygen delivery may be compromised. The ability to assess noninvasively the cerebral oxygen supply/demand relationship at this critical time might allow for modifications in clinical management. For example, one might adjust the rate of weaning from cardiopulmonary bypass, rewarw more judiciously or augment inotropic support to prevent precipitous fall in rSO₂.

These preliminary observations suggest that pre- and early post-bypass are vulnerable periods for maintenance of cerebral oxygenation during paediatric cardiac surgery and that near infrared spectroscopy is a promising tool for monitoring the cerebral oxygen supply/demand relationship, especially during circulatory arrest when other monitoring modalities are unavailable. Use of near infrared spectroscopy remains a research tool in the setting of congenital heart surgery. An appreciation of potential vulnerable periods for neurologic injury has led us, however, to examine more critically current practices. Various strategies may be employed to reduce the risk of cerebral injury. These include a longer period of cooling on cardiopulmonary bypass prior to instituting circulatory arrest, increased usage of superficial cooling of the head with ice packs, avoidance of ‘warm’ (> 20°C) circulatory arrest and short periods of intermittent reperfusion during long episodes of deep hypothermic circulatory arrest. Near infrared spectroscopy offers a suitable technique to assess whether these strategies can help maintain adequate cerebral oxygenation.

Several technical considerations remain to be explored in greater detail. These include the possibility of ‘contamination’ of signal by superficial extracerebral tissues [21,22] (perhaps less problematic in the paediatric age group), the validity of assumptions about the constancy of the path length of light in the rapidly changing conditions (e.g. alterations in haematocrit) encountered during cardiopulmonary bypass and circulatory arrest [20] and the possibility that intravascular oxygenation may be an inadequate surrogate for intracellular oxygenation [23]. Ultimately the potential of this technique for predicting neurologic injury during paediatric cardiac surgery can only be established by large-scale, long-term prospective clinical trials.

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**References**


