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Esophageal saturation during antegrade cerebral perfusion: a preliminary report using visible light spectroscopy

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Summary

Background: Visible light spectroscopy (VLS) is newer technology that measures real-time tissue oxygenation. It has been validated in detecting mucosal ischemia in adults. During complex neonatal heart surgery, antegrade cerebral perfusion (ACP) maintains cerebral saturation. Whether ACP maintains peripheral tissue perfusion in humans is not known.

Methods: Five patients undergoing neonatal open heart surgery with hypothermic cardiopulmonary bypass (CPB) were studied using a VLS esophageal probe in addition to bilateral near infrared cerebral oximetry. Three of five patients required ACP for arch repair, while two patients did not. VLS and cerebral saturation data were collected and analyzed in 5 min intervals prior to CPB, during CPB, and during ACP.

Results: In the two patients undergoing heart surgery with routine hypothermic CPB, both cerebral and esophageal saturations were maintained. However in all three neonates requiring ACP, although cerebral saturations did not decrease, esophageal saturation fell below the ischemic threshold (35%). Following establishment of normal CPB, esophageal saturation returned to baseline.

Conclusions: Antegrade cerebral perfusion maintains cerebral oxygen delivery, however, it does not adequately perfuse the esophagus in neonates. This could have clinical implications.

Keywords: visible light spectroscopy; near infrared cerebral oximetry; children*:* somatic perfusion; open heart surgery; cardiopulmonary bypass

Introduction

Visible light spectroscopy (VLS) is a newer, minimally invasive quantitative technology that allows for real time early detection of tissue ischemia using visible light in the range of 475–600 nm(1). VLS is valuable in differentiating between pure hypoxemia and conditions in which ischemia plays a part. Unlike conventional pulse oximetry which becomes unreliable or fails completely under the very conditions during which hypoxia is most likely, VLS technology is not influenced by a lack

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of pulsatile flow, hypothermia or vasoconstriction (2). Near infrared spectroscopy (NIRS) is also noninvasive and has been used successfully to assess changes in cerebral oxyhemoglobin saturation during cardiopulmonary bypass (CPB) in children and uses light in the infrared spectrum of 700–1000 nm (3). The basic differences between VLS and NIRS lie in the depth of penetration and the volume of tissue in which hemoglobin oxygen saturation is measured.

Unlike NIRS or pulse oximetry (SpO_2%), VLS oximetry uses shallow penetrating visible light to measure a local estimate of the microvascular hemoglobin oxygen saturation (StO_2%) in thin, small tissue volumes (1–2 mm). NIRS measures O_2 saturation in larger volume of tissue and has a greater depth of penetration (1–2.5 cm). The latter is therefore useful in measuring brain O_2 saturation where near infrared light can penetrate the skull, whereas VLS can measure mucosal O_2 saturation more accurately because of its shallow depth of penetration.

Complex neonatal open heart surgery (OHS) associated with aortic arch anomalies is performed under hypothermic CPB and either deep hypothermic circulatory arrest (DHCA) or antegrade cerebral perfusion (ACP). During ACP, cerebral perfusion is maintained by cannulating the innominate artery while the arch repair is undertaken. Proponents exist for both techniques (4) and at Stanford University the surgical preference is for ACP.

During DHCA, neither the brain, nor rest of the body receive oxygen supply. On the other hand, ACP at pump flows of 30–80 $ml \cdot kg^{-1} \cdot min^{-1}$ maintains oxygen supply to the brain (4). Whether oxygen supply to rest of the body is maintained is not clearly known. In a neonatal piglet model of CPB, animals were randomized to DHCA or ACP. Using VLS (T-Stat, Spectros Corp., Portola Valley, CA, USA) and an esophageal probe to measure mucosal oxygen saturations (StO_2) we demonstrated that StO_2 was maintained at baseline CPB levels in animals undergoing ACP but decreased significantly in those randomized to DHCA (5).

Based on the above study, we hypothesized that in human neonates undergoing aortic arch repair under CPB and ACP, esophageal mucosal StO_2 would be maintained at baseline levels noted during CPB. To prove our hypothesis, we placed an esophageal probe, and using VLS, monitored esophageal StO_2 as a marker of somatic perfusion in neonates undergoing aortic arch repair where innominate cannulation and ACP were indicated for surgical repair.

Methods

The study was approved by Stanford University review board for human studies and written parental consent was obtained. Infants undergoing OHS requiring CPB and ACP for repair of their cardiac lesion were enrolled. Infants requiring hypothermic CPB but not ACP were controls.

Following routine monitoring (ECG, BP, pulse oximetry) the neonates were anesthetized and orally intubated. Cerebral oximeter probes, routinely used in our institution during OHS, were placed bilaterally in patients undergoing innominate cannulation and only on the right side for routine CPB. The probes were attached to INVOS 5000 for continuous monitoring of cerebral hemoglobin oxygen saturation (rSO_2) (Somanetics Corporation, Troy, MI, USA).

Next, a 5-mm esophageal VLS probe was placed in the esophagus [our trans-esophageal echocardiography (TEE) probe has a diameter of 1 cm]. Chest X-ray was obtained to verify position, both of the tip of the tracheal tube (this is the standard institutional practice in all neonates undergoing OHS) and of the VLS probe in the lower 1 cm of the esophagus. This distance was subsequently marked on the probe. Thereafter the VLS probe was removed, to be replaced by a pediatric TEE probe for presurgical assessment. Following baseline TEE examination, the TEE probe was replaced by the VLS probe, inserted to the previously marked distance, for continuous reading of esophageal mucosal StO_2.

Cooling during CPB was accomplished using pH-stat strategy and rewarming with alpha-stat, hematocrit was maintained on CPB between 28–30% and rectal temperature to 18–20°C. Following surgery, during rewarming, the VLS probe was removed and TEE probe reinserted for postsurgical cardiac examination. The study ended at this point. In one patient VLS probe was left in place as this patient was not suitable for TEE examination because of his weight (1.58 kg).

Patient's demographic data, perfusion records, VLS and NIRS data were collected for analysis offline. Continuous data are presented as mean ± SD. NIRS data (rSO_2) was collected every 60 s and was averaged for a 5-min period at the following time periods: under anesthesia prior to surgery, 10 min following start of CPB and cooling, 10 min following start of ACP. VLS data (StO_2) were recorded every second and were also averaged over similar 5-min time periods.

Results

Table 1 gives the demographics for the five patients with complete data. One patient was excluded for incomplete esophageal oximetry data. Figure 1a,b reflects typical intraoperative continuous rSO_2 and esophageal mucosal StO_2 during CPB without ACP (control) (Figure 1a) and in patients undergoing CPB with ACP (Figure 1b).

All patients underwent OHS with CPB, three patients underwent aortic arch repair with CPB and ACP (flow rates 30–80 $ml \cdot kg^{-1} \cdot min^{-1}$). Two of five patients did not require ACP. Baseline StO_2 and rSO_2 saturation values were 64 ± 4% and 67 ± 4% respectively for the three ACP patients and 55 ± 9% and 74 ± 15% respectively for patients not requiring ACP. During CPB, esophageal mucosal StO_2 differed significantly between ACP patients and the controls.

During CPB in the control group, StO_2 remained at or above baseline as did rSO_2. (Figure 1a). In patients who required ACP, during pump flow rates of 30–80 $ml \cdot kg^{-1} \cdot min^{-1}$, esophageal StO_2 declined 20% below baseline in all three instances. In all patients undergoing ACP, mean arterial blood pressure was maintained between 10–40 mmHg (MAP measured in the umbilical artery, femoral artery and in one instance in the axillary artery). The range of StO_2 was 4–17% at flows of 30–80 $ml \cdot kg^{-1} \cdot min^{-1}$. During this period rSO_2 % was maintained at or above baseline (Figure 1b).

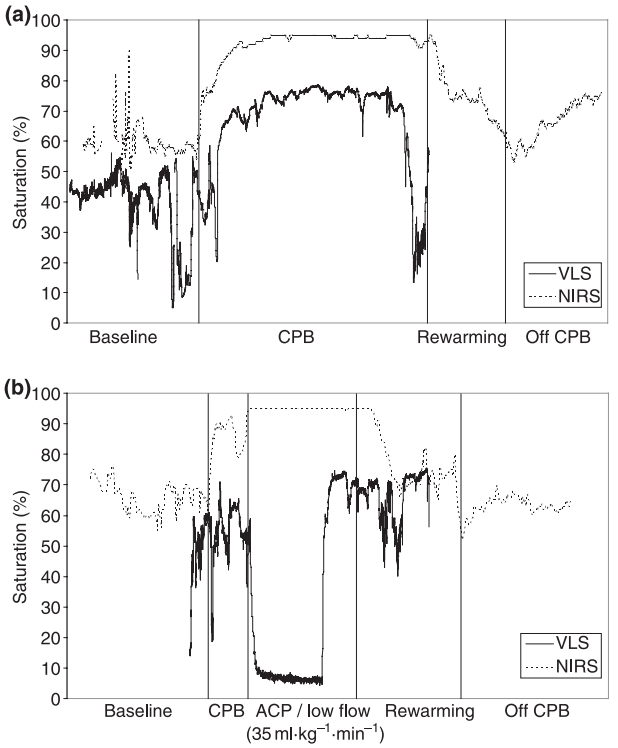

Figure 1
(a) Cerebral and somatic saturations during cardiopulmonary bypass (CPB). (b) Cerebral and somatic saturations during antegrade cerebral perfusion (ACP).

Cerebral saturation returned to baseline post-bypass in all patients (control and ACP group). StO_2 data during rewarming and after bypass was not available in four of five patients as the TEE probe

Table 1
Patient information

Case no.	*Age (days)*	*Weight (kg)*	*Diagnosis*	*Baseline SpO_2%*	*Baseline StO_2%*	*Baseline NIRS%*
1	2	2.8	Critical AS, with arch obstruction	85	61	64
2	19	1.58	Incomplete aortic arch, VSD repair	100	68	76
3	3	2.8	Aortic atresia, hypoplastic ascending aorta, VSD	76	63	67
4	8	3.9	TGA, ASD, VSD, PS, PDA	89	64	88
5	7	3.2	TGA, DORV, PDA	93	47	63

Cases 1, 2, and 3 underwent CPB + ACP and cases 4 and 5 underwent CPB not requiring ACP.
AS, aortic stenosis; VSD, ventricular septal defect; TGA, transposition of the great arteries; ASD, atrial septal defect; PS, pulmonic stenosis; PDA, patent ductus arteriosis; DORV, double outlet right ventricle NIRS, near infrared spectroscopy.

was reinserted at this time for cardiac evaluation. In the one case where postbypass data were available, StO_2 returned to baseline of 67%.

Discussion

Using VLS technology we found that during routine CPB, oxygen delivery to the brain and systemic circulation (esophagus) is maintained at or above baseline. In contrast, during ACP, although cerebral oxygen delivery is maintained with innominate artery perfusion at flow rates of 30–80 $ml \cdot kg^{-1} \cdot min^{-1}$, systemic saturation plummeted to levels consistent with tissue ischemia. As VLS offers the ability in real time, to quantify tissue oxygenation, this technology may be useful in the early detection of ischemia in infants and children at risk for hypoxic injury.

The anatomical difference in the arterial circulation between humans and piglets perhaps accounts for the observed difference between our laboratory study and our observations in humans. In piglets, the aorta gives rise to the innominate artery and the left subclavian artery. The former, divides into a right subclavian as well as the right and left carotid arteries. Innominate cannulation therefore, perfuses both cerebral hemispheres, and the body, via the right subclavian. Perhaps collaterals from the right subclavian contribute to esophageal perfusion and thus maintain normal tissue oxygen saturation even during ACP. On the other hand, in humans, when the innominate is cannulated, only the right cerebral hemisphere is perfused and a patent Circle of Willis allows flow to the left cerebral hemisphere. This was evidenced in our study by bilateral normal cerebral oxygen saturation during periods of ACP flow. Both subclavian arteries are excluded from perfusion during ACP in humans. In humans, the lower third of the esophagus is commonly perfused by the esophageal branches of the thoracic aorta and esophageal branch of left gastric artery (given off by the celiac trunk arising from the abdominal aorta). We speculate that esophageal perfusion was not maintained during ACP as the flow in the thoracic and abdominal aorta are insufficient.

In the nine neonates studied by Hoffman *et al.*, somatic saturations were detected via NIRS probes placed in the thoraco-lumbar region. In these neonates, rSO_2 fell from 59% to 41% during ACP (31% decrease) (6). This level is very close to the ischemic NIRS threshold of 33–44% (7). Pigula et al studied 15 neonates requiring ACP and/or DHCA (8). Using NIRS in the quadriceps region, they reported that somatic saturation was maintained close to baseline during periods of ACP (68% at baseline to 57% during ACP, 16% decrease). Contrary to the author's conclusion that ACP maintains somatic perfusion, when the reported standard deviation of 25 in that study is factored into a mean of 57, it suggests some patients in Pigula's study also had somatic saturation (rSO_2, 32%) that was below the ischemic threshold (8). Our results concur with these findings. NIRS measures much larger tissue samples than VLS, hence standard deviation of the data is large, unlike tissue-oximetry. This was noted in previous publications (1). Furthermore in the study by Pigula *et al.*, alpha-stat blood gas management was used during ACP and DHCA. It is our practice to use pH-stat blood gas management during hypothermia and ACP. We speculate that the blood gas management strategy preserved cerebral blood flow at the expense of somatic blood flow, accounting for very low tissue oxygen saturation during ACP in our study.

The limitations of this study are its size and the lack of outcome correlations. A much larger study would be required to determine if low esophageal tissue saturations during ACP would result in postoperative gastrointestinal complications. Larger, prospective studies are planned to address these concerns.

In conclusion, VLS oximetry provides a continuous, noninvasive, and localized measurement of tissue oxygen saturation. During CPB with ACP, although bilateral cerebral oxygen saturation is maintained, tissue oxygen saturation measured by VLS oximetry is not. The clinical implication of this finding should be evaluated in a larger prospective trial in children undergoing heart surgery.

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