Near-infrared spectroscopy to monitor cerebral oxygen saturation in single-ventricle physiology

Marco Ricci, MD,[a] Pierluca Lombardi, MD,[a] Steven Schultz, MD,[b] Alvaro Galindo, MD,[c] Ernesto Coscarella, MD,[d] Amelia Vasquez,[c] and Eliot Rosenkranz, MD[a]

Objectives: Near-infrared spectroscopy monitors cerebral oxygen saturation. This parameter parallels jugular venous oxygen saturation and reflects the balance between cerebral oxygen supply and demand. Experience with near-infrared spectroscopy in univentricular physiology is limited. This study explores the relationship between cerebral oxygen saturation, jugular venous oxygen saturation, and other variables of cerebral perfusion in a model of univentricular physiology.

Methods: Univentricular physiology was created in newborn piglets by means of an aortopulmonary shunt, echocardiography-guided atrial septostomy, tricuspid valve avulsion, and pulmonary artery occlusion. Intra-aortic balloon inflation was used to increase afterload. Cerebral oxygen saturation monitoring (INVOS 5100; Somanetics Corp, Troy, Mich), physiologic recordings, and stable-isotope microsphere determination of cerebral blood flow were performed at baseline and after conversion to univentricular physiology (30 minutes, 120 minutes, and during afterload augmentation).

Results: Univentricular physiology resulted in lower cerebral oxygen saturation, arterial oxygen content, jugular venous oxygen saturation, and cerebral oxygen delivery. Afterload augmentation increased cerebral oxygen saturation, arterial oxygen content, and jugular venous oxygen saturation, whereas cerebral oxygen delivery was unaffected because of lower cerebral blood flow. Cerebral oxygen saturation predicted jugular venous oxygen saturation, arterial oxygen saturation, and arterial oxygen content. No association was found with cerebral oxygen delivery, which decreased in parallel with cerebral oxygen saturation when the single-ventricle physiology model was established but failed to increase during afterload augmentation.

Conclusions: This study shows that in univentricular physiology cerebral oxygen saturation correlates well with jugular venous oxygen saturation, arterial oxygen saturation, and arterial oxygen content. However, our findings suggest that in singe-ventricle physiology changes in cerebral oxygen saturation need to be interpreted in the context of changes in arterial oxygenation.

From the Division of Cardiothoracic Surgery,[a] Pediatric Cardiology,[b] Pediatric Critical Care,[c] and the Department of Neurological Surgery,[d] University of Miami Miller School of Medicine, Miami, Fla.

Supported by the DeWitt Daughtry Family Department of Surgery of the University of Miami Miller School of Medicine.

Read at the Thirty-first Annual Meeting of The Western Thoracic Surgical Association, Victoria, BC, Canada, June 22-25, 2005.

Received for publication May 10, 2005; revisions received June 20, 2005; accepted for publication July 12, 2005.

Address for reprints: Marco Ricci, MD, Division of Cardiothoracic Surgery, University of Miami Miller School of Medicine, Holtz Center 3072 (R-114), 1611 NW 12th Ave, Miami, FL, 33136 (E-mail: mricci@med.miami.edu).

J Thorac Cardiovasc Surg 2006;131:395-402

0022-5223/$32.00

doi:10.1016/j.jtcvs.2005.07.039

Transcranial near-infrared spectroscopy (NIRS) is a novel technology used clinically to monitor regional cerebral O_2 saturation (rSO_2).[1] It allows the noninvasive detection of changes in the ratio of oxyhemoglobin to deoxyhemoglobin (rSO_2 index) in the frontal cortex, thus providing indirect information on the adequacy of cerebral oxygenation.[2]

Previous experience with NIRS in clinical practice has focused primarily on the detection of cerebral hypoxia in the context of normoxia and biventricular physiology.[3,4] In this setting rSO_2 has been shown to parallel jugular venous O_2saturation ($jvSO_2$) consistently.[5] RSO_2 monitoring has been found to be useful in a variety of clinical settings, especially cardiac and vascular surgery, as a surrogate indicator of

Abbreviations and Acronyms
CaO_2 = arterial oxygen content
CBF = cerebral blood flow
dpm = disintegrations per minute
$jvSO_2$ = jugular venous oxygen saturation
NIRS = near-infrared spectroscopy
rSO_2 = cerebral oxygen saturation
SVP = single-ventricle physiology

the adequacy of cerebral perfusion.[6,7] Because NIRS is used as a trend monitor, a decrease in rSO_2 can reflect a shift in the balance between cerebral oxygen supply and demand and therefore suggests cerebral hypoxia.

Few studies on the use of NIRS in children with congenital heart disease have been reported,[8-10] and the experience with this technology in cyanotic children with single-ventricle physiology (SVP) is limited.[11] In contrast to biventricular physiology, rapid changes in systemic and pulmonary vascular resistance can produce changes in cerebral perfusion in SVP, as well as significant fluctuations in pulmonary blood flow and therefore arterial oxygenation. Previous reports have indicated that NIRS is heavily influenced by markers of arterial oxygenation, such as arterial O_2 content (CaO_2) and arterial O_2 saturation.[12] We hypothesized that changes in arterial oxygenation occurring simultaneously with changes in cerebral blood flow (CBF) could influence the ability of transcranial NIRS to predict changes in cerebral oxygenation. In our study we investigated the relationship between rSO_2, as measured with a commercially available NIRS device, and $jvSO_2$ in an animal model of hypoxemia and SVP. We also explored the association between rSO_2, cerebral O_2 delivery, and other physiologic variables of cerebral oxygenation in an environment in which simultaneous changes in CaO_2 and CBF were induced by afterload augmentation.

Methods

Animals

Eight Yorkshire newborn piglets (3.7 ± 0.5 kg) were used. The study was approved by the Animal Care and Use Committee of the University of Miami Miller School of Medicine and carried out in compliance with the 1996 National Research Council guidelines on the Care and Use of Laboratory Animals.

Surgical Preparation

Piglets were anesthetized with intramuscular ketamine (40 mg/kg) and xylaxine (4 mg/kg), intubated through a tracheostomy, and started on volume-control ventilation (tidal volume, 25-30 mL/kg; rate, 25 breaths/min; inspired O_2 fraction, 0.25). Anesthesia was maintained with continuous infusion of fentanyl (50 $\mu g \cdot kg^{-1} \cdot h^{-1}$), pancuronium (0.4 $\mu g \cdot kg^{-1} \cdot h^{-1}$), and midazolam (0.2 $mg \cdot kg^{-1} \cdot h^{-1}$). A catheter was inserted in the femoral artery for pressure monitoring and blood sampling. A 6F introducer sheath to be used for intra-aortic balloon dilation (afterload augmentation) was inserted in the opposite femoral artery. A 7F introducer sheath was placed in the femoral vein for fluid administration and subsequent insertion of an atrial septostomy catheter. A catheter was inserted in the left internal jugular vein for venous blood sampling. Electrocardiography, rectal temperature, and rSO_2 were monitored.

The single-ventricle model resembled that described by others.[13,14] Through a median sternotomy, catheters were placed in the right and left atrium. Heparin was administered (150 U/kg), and a 3.5-mm polytetrafluoroethylene* shunt was interposed between the aorta (proximal to the take-off of the innominate artery) and the pulmonary artery. While the shunt was clamped, a 2-mL balloon septostomy catheter (Medtronic Vascular, Danvers, Mass) was advanced from the right femoral vein into the right atrium. Epicardial 2-dimensional echocardiography was used to direct the catheter across the atrial septum and perform a pull-back septostomy. The same catheter was then advanced into the right ventricle. The tricuspid valve was made incompetent by repeatedly withdrawing the inflated balloon across the valve. Finally, the shunt was opened, and the main pulmonary artery was occluded. This allowed the left ventricle to support both the systemic and pulmonary circulations, reproducing a physiology similar to that of pulmonary atresia with intact ventricular septum.

Experimental Protocol

Eight piglets were included in the study group. Animals were ventilated at a constant fraction of inspired oxygen of 25%, adjusting the respiratory rate to maintain a P_{CO_2} of between 35 and 45 mm Hg. Rectal temperature was kept at 35.5°C to 36.5°C. On the basis of experience from previous pilot experiments, normal saline (4 $mL \cdot kg^{-1} \cdot h^{-1}$), dopamine (5-10 $\mu g \cdot kg^{-1} \cdot min^{-1}$), and epinephrine (0.05-0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$) were administered in all animals throughout the entire experiment to maintain cardiovascular stability and avoid hemodynamic perturbations. Calcium gluconate and sodium bicarbonate were given as needed. Fresh whole blood was obtained from an adult pig and infused to maintain a hemoglobin concentration as close to baseline as possible.

Data collection was carried out at baseline and after conversion to SVP (30 minutes, 120 minutes, and during afterload augmentation). Measurements during afterload augmentation were obtained 15 minutes after inflating a balloon in the distal descending thoracic aorta. Data collected included hemodynamic parameters, blood sampling (arterial, central venous, and cerebral venous, hemoglobin, and lactate), determination of total cardiac output with an electromagnetic flowmeter, determination of CBF with stable-isotope microsphere injections, and transcranial measurement of rSO_2 by using NIRS. At completion, piglets were killed with KCl and fentanyl. An autopsy was performed to confirm the correct positioning of all indwelling catheters and the adequacy of the atrial septostomy and that of the aortopulmonary shunt anastomoses. The brain was removed and weighed for blood flow determinations.

*Gore-Tex shunt, registered trademark of W.L. Gore & Associates, Inc, Newark, Del.

ET