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The use of cephalad cannulae to monitor jugular venous oxygen content during extracorporeal membrane oxygenation

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Abstract

Background: When used during extracorporeal membrane oxygenation (ECMO), jugular venous bulb catheters, known as cephalad cannulae, increase venous drainage, augment circuit flow and decompress cerebral venous pressure. Optimized cerebral oxygen delivery during ECMO may contribute to a reduction in neurological morbidity. This study describes the use of cephalad cannulae and identifies rudimentary data for jugular venous oxygen saturation (JVO_2) and arterial to jugular venous oxygen saturation difference ($AVDO_2$) in this patient population.

Results: Patients on venoarterial (VA) ECMO displayed higher JVO_2 ($P < 0.01$) and lower $AVDO_2$ ($P = 0.01$) than patients on venovenous (VV) ECMO ($P < 0.01$). During VV ECMO, JVO_2 was higher and $AVDO_2$ lower when systemic pH was < 7.35 rather than > 7.4 ($P = 0.01$). During VA ECMO, similar differences in $AVDO_2$ but not in JVO_2 were observed at different pH levels ($P = 0.01$).

Conclusions: Jugular venous saturation and $AVDO_2$ were influenced by systemic pH, ECMO type and patient age. These data provide the foundation for normative values of JVO_2 and $AVDO_2$ in neonates and children treated with ECMO.

extracorporeal membrane oxygenation venovenous ECMO, venoarterial ECMO, cephalad cannulae, jugular venous oxygen content

Introduction

Extracorporeal membrane oxygenation (ECMO) is used to treat newborn infants and children experiencing life-threatening cardiorespiratory failure unresponsive to conventional medical therapy [1,2]. Infants meeting the required criteria are estimated to have 80% mortality if they do not receive ECMO compared to approximately 80% survival for those who do receive the treatment [3]. This survival is not without significant cost and morbidity [2]. Substantial investigative interest has focused on the neurological outcome of patients treated with ECMO. Optimized cerebral oxygen delivery during ECMO may limit neurological morbidity associated with hypoxia.

Monitoring jugular venous oxygen saturation (JVO_2) as a method of approximating global cerebral oxygenation via a jugular venous bulb drainage catheter is a safe and reliable method in both adults and children [4,5], including neonates [6,7]. Jugular venous oximetry is used in the management of patients with increased intracranial pressure [8-11], as well as intra-operatively during cardiopulmonary bypass [12] and during neurosurgical procedures [13]. Jugular venous sampling enables calculation of arterial to jugular venous oxygen saturation difference ($AVDO_2$) for more precise monitoring of cerebral oxygen content and to aid in the assurance of adequate oxygen delivery [14,15].

When used during ECMO, jugular venous bulb catheters, also known as cephalad cannulae, increase venous drainage, augment circuit flow, and decompress cerebral venous circulation. Currently there are insufficient data available to clarify the results of samples obtained from cephalad cannulae used as monitoring tools during ECMO. The purpose of this study was to describe the use of cephalad cannulae and the data obtained from jugular venous blood samples as an additional tool in the management of the ECMO patient. Our goal was to

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identify rudimentary data that would be foundation for normative data for JVO_2 and $AVDO_2$ in this population of patients.

Materials and methods

Data collection

In this retrospective study, we reviewed the medical records of all the patients treated with ECMO in whom a cephalad cannula was placed. Data collected included vital signs, arterial blood gases, jugular venous blood gases, ECMO flow rate, as well as the type of ECMO used. These data were recorded every 8 h at the time of jugular venous blood sampling as per our ECMO protocol. Patient data were compared using the following categories: neonatal, pediatric, and the type of ECMO utilized [venoarterial (VA) or venovenous (VV)].

ECMO procedure

Cephalad cannulae are inserted via an arterial catheter into the jugular vein. The size of the catheter is based on patient weight and blood vessel diameter. In neonates this is most commonly a catheter between 10 and 14 F. In pediatric patients, the same size or one size smaller than the venous drainage catheter is used. The catheter is then advanced in a retrograde fashion into the jugular vein until resistance is met. Optimal cannula flow is considered to be between one-third and one-half of total ECMO flow. The insertion of the cephalad catheter is performed at the time of ECMO cannulation.

All neonates unergoing ECMO received sedation with morphine and lorazepam without neuromuscular blockade. Pediatric patients routinely received sedation with an opioid (fentanyl or morphine) and a benzodiazepine (midazolam or lorazepam). Neuromuscular blockade was achieved in the pediatric patients with either vecuronium or atracurium.

Measurements

Arteriovenous oxygen content differece was calculated using the formula:

$AVDO_2$ = arterial oxygen content (CaO_2) - venous oxygen content (CVO2)

where CaO_2 (vol%) = [hemoglobin × arterial saturation (%) × 1.36] + [arterial PO_2 × 0.0031] and CVO_2 (vol%) = [hemoglobin × venous saturation (%) × 1.36] + [venous PO_2 × 0.0031].

Systemic venous saturation (SVO_2) was not measured since recirculation and return of ECMO derived oxygenated blood into the venous circulaton with VV ECMO would render this measurement inaccurate.

Data analysis

All data are presented as mean ± standard deviation. Data analyses of changes in JVO_2 or $AVDO_2$ over time were performed using analysis of variance (ANOVA) for repeated measures. Analyses of data between groups and under different clinical conditions were performed utilizing ANOVA with *post hoc* analysis using Fisher's test of least squares. Linear and non-linear correlation analysis was used to determine any correlation between physiologic parameters, ECMO flow, and JVO_2 or $AVDO_2$. Probabilities of <0.05 were considered statistically significant.

Results

Patient population

Forty-seven patients were studied including 36 neonates and 11 pediatric patients. The demographic characteristics of the patient population are described in Table 1.

Three patients were removed from the study due to malfunction of the cephalad cannulae or incomplete data collection. Three hundred and eight measurements were reviewed. Neonatal ECMO patients carried a mortality of 11%, while the mortality of pediatric ECMO patients was 18%. Both pediatric deaths occurred in patients with underlying cardiac anomalies. The diagnoses of all patients are shown in Table 2.

Monitoring

Demographic data collected included name, age, diagnosis and weight. Blood gas results were collected every 8 h for the first 3 days of the ECMO run, and included patient arterial (postductal in neonates), cephalad venous, pre-membrane venous and post-membrane measurements. Vital signs and ECMO flow were also collected to coincide with the time of blood gas analysis.

There was no correlation between JVO_2 and mean arterial blood pressure, heart rate, PaO_2, $PaCO_2$, peripheral saturation or ECMO flow. Similarly, there was no correlation between these parameters and $AVDO_2$. The above mentioned clinical parameters were maintained within a normal range during the ECMO run. The number of values obtained at extremes was small.

Table 1 Demographic data

	Neonatal	Pediatric
Total in Group		
VV with ceph	31	7
VA with ceph	5	4
Total	36	11
Weight	2.5-4.7 kg	2.7-61 kg
Age	5-168 h	3 months-16 years
Sex		
Male	25	7
Female	11	4

VV = venovenous, VA = venoarterial, ceph = cephalad drain.

Table 2 Diagnoses

		VV	VA	Total
Neonatal	Meconium aspiration	12	1	13
	Sepsis	4	0	4
	Persistent pulmonary hypertension	8	1	9
	Congenital diaphragmatic hernia	3	2	5
	Lung hypoplasis	2	0	2
	Respiratory distress syndrome	2	1	3
Pediatric	Acute respiratory distress syndrome	3	0	3
	Near drowning	1	0	1
	Myocarditis	1	1	2
	Asthma	2	0	2
	Necrotizing tracheitis	0	1	1
	Respiratory syncytial virus	0	1	1
	Pneumonitis	0	1	1

W = venovenous; VA = venoarterial.

Mean JVO_2 and $AVDO_2$ changed over the course of the ECMO run in patients treated with VV ECMO, but not in patients treated with VA ECMO. Patients on VA ECMO had higher JVO_2 ($P < 0.01$) and lower $AVDO_2$ ($P = 0.01$) than patients on VV ECMO. Neonates had lower JVO_2 and higher $AVDO_2$ than pediatric patients. When the type of ECMO was considered, neonates on VA ECMO had lower JVO_2 and higher $AVDO_2$ than pediatric patients on VA ECMO. Neonates on VV ECMO had higher $AVDO_2$ than pediatric patients, but JVO_2 was similar. Multivariate analysis showed that the type of ECMO was more important than the patient's age group in determining both $AVDO_2$ and JVO_2.

During VV ECMO, JVO_2 was higher and $AVDO_2$ was lower when the systemic pH was < 7.35 than when the pH was >7.4. During VA ECMO, similar difference in $AVDO_2$, but not in JVO_2, were observed at different pH levels ($P = 0.01$).

There were no complications (ie increased bleeding, venous thrombosis, infection or limitation of ECMO flow) due to the cephalad cannulae. Clotting of the cephalad cannula necessitated its removal in four out of 47 cases (8.5%). Clots were identified by visual inspection and/or blood flow decreasing to less than 50 cm^3/min as measured by a transit time flowmeter (Transonic Systems Inc, Ithica, NY, USA). Clotted catheters were identified and removed at 5, 10, 120 and 254 h of ECMO. The remaining catheters were removed at the end of ECMO therapy. All catheters were removed without incident. No morbidity was suffered by any patient who had their cephalad cannula removed due to clot identification or decreased flow. There were no reported incidents of intracranial hemorrhage in any of the patients with cephalad catheters. Long-term neurologic follow-up was unavailable due to the retrospective nature of our patients who are referrals from other institutions, specifically sent for ECMO, then returned to the referral area once support is terminated.

Discussion

Patients requiring ECMO have experienced varying degrees of hypoxia, hypotension, and acidosis [1]. Clinical and laboratory data suggest that severe hypoxia, similar to that occurring in patients requiring ECMO, alters cerebral autoregulation [16-18]. These studies demonstrate significant cerebral hyperemia, characterized by increased volume and velocity of cerebral blood flow after severe hypoxia [19]. The initiation of ECMO also alters cerebral autoregulation in healthy animals [20,21]. In neonates, initiation of VA ECMO causes an increase in cerebral blood flow [22,23]. A better understanding of cerebral oxygen consumption and delivery during ECMO may improve the quality of care that we provide for these patients. Neurological morbidity associated with hypoxia and reperfusion injury may therein be reduced.

Our study demonstrates that, within the normal ranges of mean arterial blood pressure, arterial oxygen and carbon dioxide content, JVO_2 and $AVDO_2$ were consistent over time. In addition, changes in ECMO pump flow were not correlated with changes in JVO_2 or $AVDO_2$. Although it has been suggested that cerebral blood flow is altered during ECMO [20-23], our data imply that cerebral autoregulation may remain intact. In the future, directly monitoring cerebral blood flow may provide the data needed to address this question. Several factors were found to be associated with lower JVO_2 and higher $AVDO_2$. During VV ECMO, there was an initial drop in JVO_2 with a corresponding rise in $AVDO_2$, followed by stabilization of both. The changes were most marked during the first 24 h of ECMO, with stabilization occurring after 32 h. SVO_2 was not measurable and/or inaccurate because of the delivery of oxygenated blood directly into the venous circulation and due to the effects of recirculation on the measurement of SVO_2.

In contrast, there were no changes over time in JVO_2 or $AVDO_2$ in patients treated with VA ECMO. However, the number of patients in this group is small and it is possible that with a larger population a difference would be seen. Throughout their course, patients on VA ECMO had higher JVO_2 and lower $AVDO_2$ than patients on VV ECMO. Similarly, pediatric patients had higher JVO_2 and lower $AVDO_2$ than neonates.

The precise cause of the time-related changes during VV ECMO are unclear. The differences in JVO_2 and $AVDO_2$ between VV and VA ECMO are most likely due to varying oxygen delivery to the brain. During VA ECMO, oxygenated blood from the ECMO circuit is delivered into the ascending aorta immediately adjacent

to the left common carotid artery. As a result, blood entering the left common carotid is completely saturated. During VV ECMO, oxygenated blood is returned to the patient's venous blood near the right atrium. As blood from the ECMO circuit reaches the common carotid artery it is well mixed with the patient's venous blood and is not completely saturated. The potential contribution of this increased oxygen delivery to cerebral reperfusion injury following hypoxia/ischemia in patients undergoing VA ECMO is unknown.

The cause of the difference identified between neonates and pediatric patients is less clear. Our data suggest that JVO_2 and $AVDO_2$ are different in neonates and pediatric patients. There are two possible reasons for this finding. The clinical use of neuromuscular blockade and sedation in our neonatal intensive care unit (ICU) compared to our pediatric ICUs is different. Neonates are not routinely paralyzed and receive less sedation than pediatric patients who are routinely paralyzed and heavily sedated. This may be reflected in an increased oxygen consumption in the neonates giving them a higher $AVDO_2$ level than the pediatric patients. secondly, the global oxygen consumption of a neonate may be higher than that of an older child due to age alone. The significance and implications of the relatively higher JVO_2 associated with both the VA ECMO and pediatric ECMO groups is unclear and will require further study.

Changes in systemic pH were also associated with changes in JVO_2 and $AVDO_2$. We did not find a relationship between $PaCO_2$ and JVO_2 or $AVDO_2$; however, $PaCO_2$ was clinically maintained in a normal range. Cain has demonstrated that, in passively hyperventilated dogs, as pH decreases oxygen consumption also decreases [24]. This may be the explanation for $AVDO_2$ decreasing with pH in our patients. Alkalosis is a well-recognized stimulus for cerebral vasoconstriction [25]. Unfortunately, there are on data that define the optimum pH at which oxygen delivery to the brain is adequate. Conversely, excess or 'luxury' flow [26] may cause cerebral reperfusion injury associated with hypoxic insults. Our data do not allow us to define an optimal range for pH, but they do suggest that small changes in pH affect cerebral blood flow in neonatal and pediatric patients on both VV and VA ECMO.

Summary

In our study population the use of cephalad cannulae was without complications and was useful in the management of the ECMO patient. Cephalad cannulae can provide accurate, consistent readings of JVO_2 during the course of ECMO. Placement of cephalad cannulae at the initiation of ECMO was without adverse effects. We identified several factors that may influence oxygen delivery to the brain during ECMO, including systemic pH, type of ECMO and age of the patient. Future studies should attempt to define optimal oxygen delivery to the brain. This study provides a foundation of normative values for cephalad monitoring in neonates and pediatric patients on ECMO. Additional investigation is required to delineate the role cephalad catheters may play in the clinical monitoring, bedside management and long-term outcome of patients on ECMO. The use of cerebral biochemical Markers taken from jugular venous catheters may help to predict neurodevelopmental outcome in this patient population [27].

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