Perinatal-Pediatrics Bulletin

May/June '99

American Association for Respiratory Care

Notes from the Chair: CPG Revisions Sought

by Peter Betit, RRT

The AARC Clinical Practice Guideline Steering Committee needs our assistance in amending the various Clinical Practice Guidelines (CPGs) published by the Association. The Steering Committee has proposed that the development and revision of CPGs incorporate an evidence-based approach. The CPGs that are revised via this approach will focus on clinical problems and disease management rather than respiratory care procedures. Additionally, they will address outcomes, patient education, patient satisfaction, and quality of life. Where appropriate, age-specific issues will also be incorporated into the CPGs.

Evidence-based medicine is an approach to practice and teaching that is grounded not only in the evidence upon which practice is based, but also in the strength of that evidence. Evidence-based medicine emphasizes the need to base clinical decisions, whenever possible, on the results of rigorously controlled investigations. The approach calls for caution when clinical decisions rest only on the results of data from non-human studies and uncontrolled observations. (Cook et al, Crit Care Med 1996;24:334-337)

Essentially, a systematic review, including an extensive literature search, is performed. The literature is graded; randomized controlled trials with low false-positive and false-negative results receive the most weight, while case series without control subjects receive the least weight. Treatment algorithms are developed in conjunction with the CPG.

Members of our section can make a significant contribution to the Steering Committee's efforts by volunteering to review the various CPGs related to neonatal and pediatric respiratory care. Perinatal-Pediatric Resource Panel members will be called upon to assist in this endeavor.

Copies of all the CPGs published by the AARC are available at the following web site: http://www.rcjournal.com/online_resources/cpgs/cpg_index.html
CPGs that section members may be interested in revising include:

1. Neonatal time-triggered, pressure-limited, time-cycled mechanical ventilation.
   Respir Care 1994;39(8):808-816
2. Application of CPAP to neonates via nasal prongs or nasal pharyngeal tube.
   Respir Care 1994;39(8):817-823
3. Transcutaneous blood gas monitoring for neonatal and pediatric patients.
   Respir Care 1994;39(12):1176-1179
4. Capillary blood gas sampling for neonatal and pediatric patients.
   Respir Care 1994;39(12):1180-1183
5. Infant/toddler pulmonary function testing.
   Respir Care 1995;40(7):761-768
6. Selection of an aerosol delivery device for neonatal and pediatric patients.
   Respir Care 1995;40(12):1325-1335
7. Selection of an oxygen delivery device for neonatal and pediatric patients.
   Respir Care 1996;41(7):637-646

Suggestions for CPG revisions should be e-mailed to the chair of the Steering Committee, Dean Hess, PhD, RRT, FAARC, at dhess@partners.org. Please remember to include specifics and references. ■

Notes from the Guest Editor

by Douglas Eric Petsinger, BS, RRT

Greetings, fellow neonatal and pediatric RTs, from sunny Atlanta, GA. Before I jump head first into the *Bulletin's* contents, I'd like to take my hat off to Peter Betit for the tremendous efforts he has made as the editor of the *Bulletin* over the past few issues. I truly did not realize the amount of "sweat equity" involved in this difficult and strenuous task.

This issue of the *Bulletin* covers a variety of topics that I feel will be of great interest to all. Several of the authors are experts in their particular fields, which range from ECMO to neonatal developmental care to cardiothoracic management. My intent is to challenge you, as a clinician, to question yourself daily and ask, "Am I doing the very best for my patient?" The role of the RT today is completely different than it was five years ago. Due to decentralization and/or lack of adequate staffing coverage (RT or RN), therapists have had to take on more nontraditional skills.

In my heart, I truly believe that in order to succeed as an expert clinician one cannot ever stop learning. This means developing new skills that will elevate the care we give. At Egleston Children's Hospital, we have experienced decentralization of the traditional respiratory care department to specific ICUs and general care populations. While this has worked very well in the ICUs, the result has not been as positive in the general care areas. But whatever the departmental configuration, the more specialized and educated RTs are the ones who become invaluable and irreplaceable team members.

The multidisciplinary team approach works very well in our institution, especially in the CICU. Since decentralization in 1995, the addition of the RT to "the team" has provided a consistent and proactive respiratory management strategy that has resulted in decreased length of stay (LOS) and decreased cost, largely by decreasing unnecessary respiratory procedures. The latter was easily tracked by comparing pre-decentralization respiratory procedures with AARC time standards to post-decentralization time standards.

When considering the impact on LOS, one must look at mechanical ventilation practices and empowering the RT in the areas of appropriateness and accountability. For example, we don't utilize a weaning protocol in the CICU, mainly due to the staff's maturity, years of experience, and common goals for the patient (advocacy). But this was not an overnight phenomena – it was developed through patience, education, and trust from the team, all of which helped to establish the RT as an acceptable and welcome addition to the multidisciplinary team. Indeed, these factors have taken RTs at our hospital one step further. In addition to the role we play on the team itself, the management teams in the CICU, PICU, and NICU at Egleston each include at least one RT as an equal member. This, in itself, speaks well for our profession.

On another note, I would like to express my concern regarding the recent decision to limit the areas of expertise that can be listed by members of our Perinatal-Pediatric Resource Panel. (See your March-April issue of the *Bulletin*.) Clinically, when using advanced therapies, the treatment strategy varies from disease state to disease state. Let's look at two clinical arenas that come to mind: high frequency oscillatory ventilation (HFOV) and extracorporeal membrane oxygenation (ECMO). Comparing an open mediasternotomy HFOV strategy to a low lung volume HFOV strategy is not possible. Is all HFOV the same?

Clinical strategies vary according to disease states, location (intra and extra hospital), and physician coverage. The Egleston ECMO experience and strategies vary greatly from ICU to ICU because the disease states, as well as physician management, vary so greatly. A cardiac ECMO run cannot be compared to a neonatal ECMO run, plain and simple. The real intent of the Resource Panel is to provide a way for section members to network, share ideas, and, yes, maybe even create beneficial new strategies. My belief is that limiting the areas of expertise to two or three may not capture all the nuances of a particular area of interest. My question to all of you is, "should the topics be categorized by specific disease states?"

I hope that the information in this issue of the *Bulletin* sparks your interest in enhancing our practice as clinicians. If any questions or comments arise regarding the contents, please don't hesitate to contact either myself or Peter Betit at the addresses/numbers listed on this page. ■

Perinatal-Pediatrics Bulletin
is published by the
American Association for Respiratory Car e
11030 Ables Lane
Dallas, TX 75229-4593
(972) 243-2272
FAX (972) 484-2720
e-mail: info@aarc.org

Kelli Hagen
AARC communications coordinator

Debbie Bunch
Bulletin managing editor

Edwards Printing
Bulletin typesetting

Section Chair and Bulletin Editor
Peter Betit, RR T
Children's Hospital
300 Longwood Avenue
Boston, MA 02118
(617) 355-6118
FAX (617) 738-0338
e-mail: betit@a1.tch.harvard.edu

Medical Advisor
Mark Wilson, MD (ACAI)
(402) 390-5624
email: mwilson@childrens_omaha.com

Consultant Panel
Mike Czervinske, RR T
University of Kansas Medical Center
Department of Respiratory Care Education
3901 Rainbow Blvd.
Kansas City, KS 66106-7606
(913) 588-4631
FAX (913) 588-4631
email: mczervin@kumc.edu

V̇CO2 Monitoring and Such (Metabolic Ramblings)

by Douglas Eric Petsinger, BS, RRT

During breakout sessions at the AARC International Congress last year, a wide variety of topics were eloquently presented by many gifted clinicians (many of your peers). One in particular, V̇CO2 evaluation, was presented twice in the same session by Jenni L Raake from CHMC in Cincinnati, OH, and John Emberger from Christiana Care Health Services in Newark, DE. Both Jenni and John revealed the extreme importance of tracking V̇CO2 in two very tenuous types of patients: the post op congenital heart disease (CHD) neonate and low birth weight (LBW) neonate.

Carbon dioxide production (V̇CO2), as we all remember, is result of cellular metabolism – or more to the point, the degree of work placed at the cellular level. For the patient advocate in all of us, we, as a collective group, must ask the question: is our CO2 production in line with our CO2 removal? When monitoring V̇CO2, is the value a reflection of steady state metabolism, overfeeding, inappropriate weaning, increased pulmonary blood flow (PBF), or worsening pulmonary artery hypertension (PAH)?

Speaking for the RTs in Sibley Heart Center at Egleston Children's Hospital, we rely on continuous V̇CO2 monitoring with Novametrix's CO2SMO+®. We utilize ETCO2 and V̇CO2 continuous monitoring for two different subsets of post op CHD (Norwood Stage 1 Palliation and post op PAH) patients. The Norwood Stage I Palliation's post operative course is managed via a balancing act between pulmonary blood flow (Qp) and systemic blood flow (Qs); in essence, the management approach is more of a Gaustalt than the traditional "knee jerk" reaction. (*Oh no, the patient's toe temp is 27.9˚ C. Start Nipride stat, and increase the Epi to 0.15 mcg/kg/m.*)

What we attempt to do is not "micromanage" but look at the entire picture of arterial pressure, central venous pressure (CVP), and left atrial pressure (LAP); urine output (UOP); distal periphal-cutaneous temperature; acid-base balance; lactic acid levels; and SVC, SVO2, SaO2, and V̇CO2 measurements to aid in our "fact-based decision making." Granted, V̇CO2 is not the magic bullet for Norwood Palliation, but it does give us a reliable look at PBF or Qp. We know that in "steady state" CO2, production is in a very predictable cc/kg/min relationship. In fact, 5-6 cc/kg/min is considered a "normal" accepted value.

In Norwood physiology, due to a large mixing chamber (the common atrium), V̇CO2s are in the range of 3.5-5 cc/kg/min. If V̇CO2 levels rise, PBF has increased and the Qp > Qs. Then the sequella of "low systemic cardiac output" ensues, with stagnant hypoxia resulting in an increased production of lactic acid and metabolic acid furthering low cardiac output to patient hemodynamic collapse. Not a pretty sight, and one that is preventable.

One other use for V̇CO2 monitoring on the Norwood Stage I Palliation is to monitor the integrity or patency of the shunt. What happens after careful administration of clotting factors, especially platelets? We have (regrettably) discovered that with a Gortex®Shunt, the inner-diameter is significantly narrowed. This enables the bedside clinician to ask, "What's wrong with my patient?" before total hemodynamic collapse accompanied by profound CO2 retention, hypoxemia, hypotension occurs.

We have also found continuous V̇CO2 monitoring to be extremely useful in managing pulmonary artery hypertension (PAH). This is usually associated with status-post (s/p) total anomalous veneous return (TAPVR), truncus, transposition of the great arteries (TGA)-switch, complete atrio-ventricular canal (CAVC), and ventricular septal defect (VSD). Evaluation of forward flow from the RV can be enhanced by coupling V̇CO2, CVP, and LAP. If the patient becomes "super systemic," the bedside clinician will see an increasing CVP and a falling V̇CO2 and LAP, then act accordingly to decrease the PVR. In the CICU iNO Protocol for Post Operative Pulmonary Hypertension, V̇CO2 trending is included in the "gold standard" for ideal hemodynamic indicators of improved diaphragmatic cardiac output (C.O.) along with UOP, toe temperature, and lactic acid production.

Besides hemodynamic uses for V̇CO2, believe it or not there are some equally important respiratory indications for V̇CO2 monitoring. Weaning strategies from long-term mechanical ventilation vary from institution to institution, but as a collective group we must ask the question, "Are we hurting or helping the patient?" The clinician must look at the balance of CO2 elimination and CO2 production, along with oxygen consumption (VOW), in a global sense. When we ask our patients to wean, we are really telling their diaphragm and accessory muscles to begin working. With increased muscle work, the clinician is faced with an increased C.O. requirement and an increased VOW due to an increased cellular metabolism.

When the work load at the cellular level increases, the metabolic demand increases V̇O2, V̇CO2, and finally, caloric consumption. If we increase caloric consumption for spontaneous WOB, are we indeed risking increased ventilator days, decreased wound healing, decreased weight gain, and probably increased risk for nosocomial infection(s)? Are we increasing the patient's WOB because of the weaning strategy or the nutritional strategy? These are sobering questions, but necessary ones if we want to make a difference at the bedside and in the face of an ever-changing world of health care.

The challenge of successful weaning from mechanical ventilation has to be approached from several directions. Common sense dictates that adequate pulmonary recruitment is necessary to enhance proper CO2 elimination, both in full mechanical support and during a weaning strategy. This, then, begins the juggling act between deadspace (Vd/Vt), intrapulmonary shunt, and alveolar ventilation, with the goal being to decrease both Vd/Vt and shunt and optimize alveolar ventilation for CO2 clearance.

The nutritional strategy is also a balancing act, this time between providing adequate calories to promote growth and wound healing and not increasing the CO2 production at the cellular level (avoidance of high carbohydrate load). In the adult population, indirect calorimitry is often utilized to assess a proper nutritional strategy in conjunction with a mechanical ventilation weaning strategy. However, so far I have not been satisfied with the accuracy of indirect calorimitry in the neo/ped population. Currently, we are employing trending V̇CO2 along with an estimated V̇O2. This then allows us to calculate a crude respiratory quotient (RQ) on mechanical breaths and spontaneous breaths. The nutritionists are fairly convinced that they are not "overfeeding," and I'm convinced that we are weaning too quickly.

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In the future, improvements in weaning strategies must encompass markers for success and failure that are easily recognized and accepted throughout the community. I believe that V̇CO2 and V̇O2 are especially important, but obtaining a V̇O2 on all patients is not feasible or practical. However, the Novametrix Company has introduced a noninvasive cardiac output monitor, NICO2™, to the adult community. The basis for noninvasive cardiac output utilizes the derivation of the Fick Principle with partial CO2 rebreathing or C.O.=ΔV̇CO2 / S ΔPetCO2, with S representing the slope of the CO2 dissociation curve. The equation for C.O. is based on two assumptions: 1) that C.O., CvCO2, and Vd/Vt are constant during the measurement period and 2) that a shunt correction is added to the final equation, based on FiO2 and SpO2 (pulse oximeter).

The possibility of evaluating the effects of inotropic or mechanical manipulations without instrumenting a patient with a Swan Ganz catheter and adding to intravascular volume will greatly affect the overall care in the ICU. I feel that by monitoring V̇CO2 and C.O. we will be able to optimize our ventilatory strategies to meet the patient's needs more efficiently and, hopefully, improve outcomes. In the CICU population, I have high hopes that when a neo/peds adapter for the NICO2™ is developed, we will be able to demonstrate improved C.O. during iNO administration, and, with the Norwood Stage I Palliation, improved C.O. during mechanical and N2 administration. The latter use for NICO2™ would occur if there can be a correlation for intracardiac shunting. The future is here. Are we up for the challenge of improving the care we strive to deliver?

The information provided in this article should be enough to start a new thought process about critical care respiratory therapy, but there are still many unanswered questions. Viewing patient monitoring in a different light provides us with an opportunity for discovery and the establishment of a standard of care. The traditional standards for successful intervention or manipulation have to be shared via newer and more effective modalities. I implore all of you to "push the envelope" by thinking more about caloric debt and consumption. (For more information on metabolic ramblings, surf to the Novametrix website – http://www.novametrix.com – and browse around.) ■

Transitioning to a Developmentally Supportive Care Model for Neonates: *The Role of the RCP in a Team Approach*

by Esther Taylor, RRT and Lori Schuyler-Hickey, RN, BSN

Developmentally supportive care creates a framework for the delivery of individualized rather than protocol driven care. The model requires ongoing assessment and evaluation of each infant's behavioral cues to plan appropriate caregiving strategies and provide care in a manner that will enhance optimal neurobehavioral outcomes for the patient.

Over the past decade there has been a general movement within neonatal care from traditional care environments and philosophies to those which are more developmentally appropriate and supportive. Such transitions require moving from an approach to care that is typically task oriented, with specific caregiving roles and activities assigned to specific care providers, to one that is patient driven, with shared responsibilities for the provision of care that is specific to the individual patient and modified in accordance with patient cues that are reflective of response and tolerance.

In our institution, the process of transitioning from a traditional care model to a developmentally supportive model was initiated four years ago with the formation of a Developmental Care Council, which was tasked with completing a literature search and formulating a plan and timeline for transition. From the outset, RTs have been actively involved in this process, with representation on every related working group – including one that looked specifically at role integration and how our RN and RT staff could share certain caregiving activities that had traditionally been carried out by one work group or the other. The goal was to decrease the frequency and number of caretaker interactions and provide increased periods of undisturbed sleep, quiet time, or rest and recovery time for our patients.

The RT staff also participated in a work group that looked at revision of existing policies and development of new policies. This group focused on ensuring that each policy addressed the developmentally supportive aspects of care delivery. Another group that involved RTs focused on providing staff education specific to the definition of developmental care and evaluation of patient behavioral cues to guide delivery of care.

Today, we have progressed to the point where we are practicing patient driven, developmentally supportive care at the bedside. We have found that our RT staff is in a unique position to evaluate the impact of our changes in practice on patient outcomes. To date, we have noted that when "hands on" procedures such as CPT, suctioning, and aerosol treatments are provided in a developmentally supportive environment where patient cues are continually assessed and care is modified in response to specific behavioral cues, patients demonstrate an increased tolerance for caretaking. We also see a decrease in the number of negative physiologic and behavioral consequences frequently encountered with a more traditional approach to provision of these respiratory care interventions.

The implementation of developmentally supportive care and the corresponding increase in patient tolerance related to respiratory care procedures has allowed the RT to focus on completion of the procedure in a timely manner. RTs no longer find themselves repeatedly having to interrupt the procedure to calm the patient. Nor do they have to spend an extended period of time after the procedure is completed responding to negative physiologic consequences such as desaturation episodes and/or significant alterations in heart rate, respirations, or blood pressure. Together, these improvements have decreased overall treatment time. We have also noted a decrease in long-term consequences, such as development of oral aversion and subsequent delays in transition to nipple feedings in our intubated patients. This has the potential to impact the length of hospitalization by decreasing transition time from gavage to nipple feeding, thus resulting in earlier patient discharge.

Although we have not completed the transition from a traditional care model to the developmentally supportive model, we feel that we have clearly established the importance and value of RT staff involvement throughout the process and recommend that other units beginning such a process ensure that their RTs are active participants as well. ■

Principles and Practice Of Venovenous Extracorporeal Membrane Oxygenation

by Michele Labuz, CCRN, Micheal Heard, RN, Robert Pettignano, MD, Reese H. Clark, MD, and J. Devn Cornish, MD

Extracorporeal life support (ECLS) encompasses many different devices and techniques. Venoarterial (VA) ECMO is one of the original techniques that evolved from traditional cardiopulmonary bypass (CPB). VA ECLS is widely used, accounting for approximately 84% of total cases performed. VA ECLS is achieved by draining venous blood, removing carbon dioxide and adding oxygen through an artificial lung, and returning the blood to the circulation via an artery.

Venovenous (VV) ECLS, while not a commonly used therapy in most ECLS centers, is not new. Its long-term use in animals was first described in 1969 by Kolobow et. al., and in 1975 Hanson and colleagues showed that VV perfusion could be used to support adequate gas exchange in lambs breathing nitrogen.

The successful use of VV ECMO for neonatal patients with respiratory failure was first reported by Andrews et. al., in 1982. In 1985, Klein et. al., reported the support of 11 neonates with VV ECMO using the two-site approach. Moler et. al., reported in 1994 an overall survival rate of 73% in 24 pediatric patients treated with VV ECMO. Between 1991 and 1998, Egleston Children's Hospital has supported 143 patients with VV ECMO with an 83% survival rate.

For a number of reasons, VV ECMO is probably safer than VA. Most importantly, VVECMO avoids instrumentation and ligation of the carotid artery. When a double lumen cannula (DLC) can be used, cannulation of one, instead of two or three, vessels saves time. During VV ECMO, thromboemboli that enter the body from the ECMO circuit are routed to the pulmonary circulation rather than the cerebral (or systemic) circulation. And, blood entering the cerebral arterial tree is less intensely oxygenated and under less pressure. These differences may decrease the risk of neurological injury. Finally, the potential for ischemic injury to the lungs from decreased pulmonary blood flow during VA ECMO is eliminated.

A disadvantage of VV ECMO is that it does not provide direct circulatory support. Oxygenated blood from the ECMO circuit is returned to the right atrium, not the aorta. Oxygenation may be lower than observed on VA ECMO because the blood returned to the right atrium is mixed with desaturated venous blood. Maximum achievable oxygen delivery, therefore, may be inadequate in larger patients. These problems are aggravated by refractory hypotension and/or increased metabolic rate (e.g., overwhelming sepsis). In such cases, the inability of the circuit to provide a systemic pressure load and deliver supraphysiologic amounts of oxygen may limit therapy, and conversion to VA ECMO may be required.

As with VA ECMO, volume overload is a problem with VV ECMO. Despite preservation of pulsatile flow, VV ECMO is associated with a decline in renal function during the first 48 hours of initiation of bypass. Urine output is often less than 1 cc/kg/hr, even in patients with normal blood pressure and normal serum albumin, despite significantly positive fluid balance.

Other disadvantages of VV ECMO involve the use of two sites for cannulation. These disadvantages include longer operative time required for cannulae placement in separate surgical sites, complications with the groin wound, and possible vascular compromise in the cannulated leg. And finally, the major disadvantage of VV ECMO is recirculation.

Patient selection

In the past, VV ECMO has been reserved for patients with only moderate respiratory failure. Many centers do not consider VV support for patients with severe respiratory failure. At Egleston Children's Hospital, we make no such distinction. We have had substantial success in all patients, including patients suffering from extreme forms of lung disease. Our diagnosis-specific survival rates are equal to, or better than, those reported nationally for VA ECMO. We prefer to use VVperfusion for all patients with acute respiratory failure.

Decisions about the use of VV ECMO for patients with circulatory compromise are not straightforward. VV ECMO does not provide direct circulatory support and may not provide sufficient support in patients with inadequate cardiac performance. However, there have been numerous patients with right, left, or biventricular compromise who have recovered with VV support. We have found that many hypotensive patients with biventricular failure have oxygenated well, improved their mean arterial pressures, and weaned from cardiovascular drugs within a few hours of instituting VV ECMO.

However, we are not prepared to offer specific criteria for selecting patients with cardiovascular compromise for VV support. We do not use VV ECMO for patients with myocardial failure following recent cardiac surgery, a history of recent and severe cardiac arrest, or with refractory rhythm disturbances associated with systemic hypotension.

For children with cardiac depression associated with respiratory failure and its treatment, it is our current practice to isolate both the right common carotid artery and the internal jugular vein during cannulation. After a venous cannulation, we initiate VV ECMO. If the patient's oxygenation and mean arterial pressure improve progressively over the subsequent 15-30 minutes, we proceed with VV support. If the patient does not improve, or if he/she worsens at any time, a catheter is placed in the carotid artery and the patient is converted to VA support.

Patient access

Two-site VV ECMO: Early reports established that adequate oxygen delivery could be provided during acute respiratory failure when blood was drained to the pump from the internal jugular vein and returned to the circulation at the level of the right atrium via a catheter placed in another major vein. The two veins most commonly used were the jugular and femoral, where blood is drawn out of the right atrium through a jugular venous cannula and returned to the femoral vein. This "two-site" form of ECMO is primarily employed in larger pediatric patients due to the unavailability of a double lumen cannulas.

One-site VV ECMO: The most common method utilized at Egleston is one-site VV ECMO. This type of ECMO employs a double-lumen cannula (DLC), inserted into the right internal jugular vein, as both the source and return sites for blood flow. Blood is withdrawn from the right atrium through one port, circulated through the ECMO circuit, and returned to the right atrium through a second port. The key to this type of VV ECMO (DLVV ECMO) is the design of

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the DLC, which minimizes recirculation.

The limitation of DLVV ECMO is based on the lack of available cannula sizes. Presently, the DLC is available in 12 Fr, 14 Fr, 15 Fr, and 18 Fr sizes. In very small infants whose vessels will not accommodate a DL cannula, VA ECMO is employed. Femoral cannulation (to facilitate two-site VV ECMO) in very small patients causes venous congestion and thus should be avoided. The DL cannulas are becoming more available in larger sizes, eliminating the need for two-site VV ECMO.

Circulatory effects of VV ECMO

Many of the characteristics unique to VV ECMO derive from the fact that both drainage to, and reinfusion from, the ECMO circuit occur in the central venous circulation – often through the same blood vessel. This has several important clinical implications.

Both VV and VA ECMO are referred to as "bypass," but unlike VA ECMO, VV does not really "bypass" anything. Since the volume of blood drained from and returned to the central venous system is equal, VV ECMO does not decrease right ventricular preload, pulmonary blood flow, left atrial return, or left ventricular output. The absence of a change in left ventricular afterload may eliminate the isolated left ventricular "stun" syndrome seen in a subset of VA-supported patients. However, right ventricular stun is a complication that has been associated with VV ECMO. The consequences of right and left ventricular stun are similar. Neonates who develop right ventricular stun often have severe pulmonary hypertension before initiation of ECMO. On ECMO, the right ventricle becomes dilated and works poorly. The dilated right ventricle causes the ventricular septum to bow into the left ventricle. Left ventricular filling is compromised and cardiac output can be decreased. Careful echocardiographic assessment and subsequent use of pressor agents directed at reducing right ventricular afterload can be helpful in reversing these problems.

Echocardiographic studies demonstrate that patients managed on VV ECMO have normal cardiac function. VV ECMO may even indirectly improve cardiac performance by improving mixed venous oxygen content. In contrast to VA ECMO, the oxygen saturation of the blood delivered to the pulmonary artery is higher because oxygenated blood is delivered to the right atrium. Pulmonary vascular resistance and right ventricular afterload may be decreased by the effects of higher mixed venous oxygen saturation in the pulmonary arteries. In addition, avoidance of increased left ventricular afterload and improved oxygen delivery to the coronary arteries may improve myocardial performance.

Another advantage of VV ECMO is that, unlike VA, it preserves physiologic pulsatility. When compared to nonpulsatile flow, pulsatile flow decreases vascular resistance, decreases afterload, and improves organ perfusion.

The success of venovenous ECMO depends on the manipulation and management of various factors unique to this therapy. The ECMO centers that *preferentially* utilize VVECMO understand the subtleties involved in the management of oxygenation and ventilation during these runs.

Cephalad cannula

A cephalad cannula is defined as a catheter inserted into the right internal jugular vein toward the head and advanced into the jugular venous bulb. Initially, this catheter was employed to decompress the cerebral venous circulation in order to decrease the incidence of intracranial pathology. Extra-axial fluid was a common finding on cranial ultrasound, and many centers believed that use of this catheter would reduce the incidence of its occurrence. As ECMO centers gained experience with the use of this catheter, the other benefits associated with its use became apparent.

The use of a cephalad venous catheter has been shown to decrease recirculation and increase the amount of oxygen that can be added to the blood as it passes through the membrane lung. Additionally, the flow from the cephalad catheter augments the amount of venous drainage, thereby increasing the amount of maximum obtainable pump flow. Typically, 1/3 to 1/2 of the total ECMO flow is obtained from a well placed cephalad cannula. This flow is measured continuously using a Transonics Flowmeter.

Recirculation

An understanding of the concept of recirculation is critical to successful application of VV ECMO, as well as to appropriate interpretation of blood gas results obtained from patients on VV ECMO. Recirculation fraction is the portion of blood returning to the ECMO circuit immediately after being returned to the patient from the ECMO circuit.

All patients on VV ECMO have some element of recirculation. Clinically, recirculation may present as decreasing patient arterial saturations, increasing pre-membrane saturations, and decreasing $AVDO_2$. Additionally, it can be noted that the blood draining from the right atrium is the same color as the blood returning from the pump. Mathematically, recirculation fraction (R) can be estimated as:

$$R = \frac{S_{preOx} - S_{vO2}}{S_{postOx} - S_{vO2}}$$

where SpreOx is the oxygen saturation of the blood entering the oxygenator, SpostOx is the oxygen saturation of the blood exiting the oxygenator, and SvO2 is the true mixed venous oxygen saturation in the patient. For example, if the pre-oxygenator saturation is 90% and the mixed venous saturation is 55% (post-oxygenator saturation is always 100%) then the recirculation fraction is 77% – this is very BAD! However, if the pre-oxygenator saturation is 78% and the mixed venous saturation is 65%, the recirculation fraction is 37% – this is better (30% recirculation is average).

Unfortunately, it is impossible to measure mixed venous saturation during VV ECMO because oxygenated blood from the ECMO circuit has been added to the blood in the pulmonary artery. Approximations of the mixed venous saturation during VV ECMO may be obtained by sampling blood from another major vein not affected by recirculation, such as the cephalad.

It is less important to calculate recirculation fraction than it is to understand the factors that affect it. There are four factors that can affect recirculation: pump flow, catheter position, cardiac output, and right atrial size (or intravascular volume).

Pump flow

The impact of pump flow on recirculation is straightforward. If pump flow is high, the suction pressure drawing blood from the right atrium back into the ECMO circuit is higher. The increased suction pressure causes streaming of oxygenated blood from the oxygen delivery catheter to the venous drainage catheter. Recirculation fraction increases almost linearly with increasing pump flow. The amount of oxygen provided to the patient first increases and then decreases as

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pump flow increases beyond optimal flow and minimal recirculation.

In fact, the point at which "effective oxygen delivery" begins to decrease is 500 cc/min flow. The reason for the decreased effective oxygen delivery is that the recirculation proportion has limited the amount of oxygen provided to the patient. In numerical terms, effective flow may be described by the equation:

Effective Flow = Total Flow - (Total Flow * Recirculation Fraction)

When total flow is zero, the effective flow is zero. At some maximal flow, the recirculation fraction is 100% and effective flow again becomes zero. As pump flow increases, the effective flow first increases, then stabilizes, then decreases. Ideal pump flow provides the highest effective flow at the lowest revolutions per minute of the pump, yielding the least degree of tubing wear and hemolysis.

Pump flow is the factor affecting recirculation over which we have the most control. If recirculation is high (i.e., patient sats are 85%, pre-membrane sats are 86%, venous sats are 53%) WEAN the pump flow. If patient saturations improve or stay the same, wean again. If they decrease, go back up on flow and seek other causes for recirculation. Optimum flow is not fixed because there are at least three other factors that affect recirculation fraction.

Catheter position

Catheter position is another major determinant of recirculation. Surgical positioning during cannulation is critical for optimal support during VV ECMO. For example, if the DLC is positioned high in the SVC, blood flow from the catheter will tend to remain within the confines of the vessel and be drained back into the ECMO pump before it reaches the RA. When using the DLC, the patient's head should be almost midline, and the arterial reinfusion port should be flat against the patient's neck behind the ear. In larger patients, if the tips of the drainage and infusion catheters (e.g., catheters inserted in the femoral vein and the internal jugular vein) are directed at each other from close range, the recirculation fraction will be high.

If recirculation appears to be high, inadvertent changes in catheter position should be ruled out. Catheter position can change when degree of lung inflation changes, neck edema increases, or the patient moves. Catheter position problems may be diagnosed with a chest x-ray. If catheter position is the problem, try repositioning the patient's head, add or decrease neck rolls (depending on whether high or low), or add slight tension on the cannula. If the noninvasive techniques are unsuccessful, then surgical repositioning must be considered.

As discussed earlier, a cephalad cannula with good flow can effectively reduce recirculation. Blood draining from this catheter is more desaturated than the blood in the right atrium because it is not mixed with blood returning from the ECMO circuit. Also, venous saturation measurement from the cephalad cannula can be applied to the recirculation fraction equation. Of note, this saturation will be the last to be compromised due to autoregulation and physiologic attempts to maintain cerebral oxygenation.

From a practical standpoint, compare the color of the blood draining from the cephalad to the color of the blood draining from the right atrium. If the colors are similar then recirculation is low. If the colors are very different (cephalad dark or "blue" and RA bright or red) then the recirculation is high. Using this simple assessment as a guide, the specialist can manipulate the cannula or patient position to try and improve the color differential (therefore decreasing recirculation).

Cardiac output

Cardiac output also affects recirculation. If the oxygenated blood delivered to the right atrium is rapidly moved into the right ventricle, it is less accessible to the drainage catheter. In contrast, during cardiac standstill, all of the oxygenated blood flowing into the right atrium would drain back into the ECMO circuit, since it has nowhere else to go.

Cardiac output (CO) is the product of heart rate and stroke volume. Tachycardia should be managed with adequate sedation and a minimum stimulation environment, and if necessary, adenosine or cardioversion for SVT or VT. Stroke volume should be optimized by increasing intravascular volume and the use of cardiotonic drugs as indicated.

Right atrial size

For obvious reasons, intravascular volume, or more precisely, right atrial volume, also influences recirculation. When oxygenated blood is delivered to a very small right atrium it is more likely to be aspirated directly back into the ECMO circuit than if the oxygenated blood is diluted in a larger volume of desaturated blood in a normal right atrium. Obviously, if hypovolemia is the issue, assess the need for volume expanders and blood products and give as indicated.

Patient management

Since VV ECMO provides no direct circulatory support, it can be difficult to achieve the same level of oxygen delivery during VV as during VA ECMO. However, when recirculation fraction is minimal and cardiac output is supported, oxygen provided to the patient from the ECMO circuit can be similar to that seen with VA ECMO. Oxygenation is optimized when the hemoglobin concentration is around 15 g/dL, when recirculation fraction is low, and when the venous-drainage catheter is large enough to achieve approximately 120-140 ml/kg/min flow. The use of a cephalad catheter can augment the amount of oxygen provided to the patient by decreasing recirculation. In neonates, we have found that the addition of a jugular-venous bulb catheter during DLVV ECMO allows us to provide the same amount of oxygen to the patient as we provide during VA ECMO.

At our center, the ECMO flow required to support adequate gas exchange during DLVV ECMO *with* a cephalad drain in place is similar to the flow required during VA ECMO. In contrast, the flow required to support adequate gas exchange in DLVV ECMO *without* a cephalad drain in place is higher.

At present there is no simple, direct measure of oxygenation during VV ECMO. As discussed earlier, mixed venous oxygen saturation readings from the pulmonary artery are not interpretable because of the highly oxygenated blood returned from the ECMO circuit to this side of the circulation. Pre-oxygenator venous-saturation readings do not reflect changes in the patient's mixed-venous saturation alone, because this measure is strongly influenced by the recirculation fraction. When recirculation is high, blood sampled from the pre-oxygenator site will have a high saturation, even if the patient's mixed-venous saturation is dropping and oxygen delivery is inadequate.

The pre-oxygenator saturation value, taken in combination with an arterial saturation reading (e.g., from a pulse oximeter), can yield information about the changing balance of recirculation and systemic oxygen delivery. For example, if the pre-oxygenator saturation rises and

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the patient's arterial oxygen saturation falls, the recirculation fraction must have increased. Alternatively, if the pre-oxygenator saturation falls and the patient's arterial oxygen saturation rises, the recirculation fraction must have decreased. Improvement in the patient's lung function and cardiac output is generally marked by increases in the patient's saturation, with no change or a slight increase in pre-oxygenator saturation. The pre-oxygenator saturation increases because the patient's mixed venous saturation is increasing. The patient's arterial saturation improves because the respiratory system is adding oxygen to the pulmonary blood and because the mixed venous saturation is higher.

Another approach is to measure a venous saturation not affected by recirculation as an approximation of mixed-venous saturation. For example, saturations can be measured from a catheter in the inferior vena cava or the jugular-venous bulb to provide an estimation of oxygen sufficiency. Unfortunately, measurements of jugular-venous bulb saturations are not very sensitive to changes in the patient's cardiopulmonary status since cerebral blood flow is preserved even in patients with life-threatening cardiopulmonary failure.

While cephalad saturations do not readily reflect the changes in cardiopulmonary status, we have found them to be very sensitive to the changes in ventilatory status. As one would expect with intact autoregulation, changes in levels of PaCO2 directly affect the rate of blood flow measured in the cephalad cannula. As levels of PaCO2 rise, the rate of blood flow measured in the cephalad cannula also rises. As PaCO2 levels decrease, the rate of blood flow measured in the cephalad cannula also decreases. Similarly, cephalad saturations rise and fall with changes in levels of PaCO2. It has also been demonstrated that there is an indirect correlation between the arterial pH and the measured cephalad blood flow and saturations.

Clinically, measurements of cephalad flow and saturation are useful in assessing patient response to ventilator manipulations and lung recruitment, as well as acute deterioration in pulmonary status (i.e., pneumothorax).

For VV ECMO, flow is initiated at 20 ml/kg/min and advanced over 15-20 minutes to a maximum calculated flow of 150 ml/kg/min. Most patients do not require this much flow to maintain their oxygenation needs. Definition of the recirculation curve must be done early in the run, (within hours of cannulation), and frequently during the run. Flow is decreased slowly and incrementally, watching the patient's pulse oximeter, until the saturations fall. By returning to the previous flow prior to saturations falling, optimal flow for oxygenation is obtained. As described in the discussion of recirculation, this will often not be the maximum calculated flow. In general, we find flows of 90-140 ml/kg/min to be optimal.

Positive results with VV ECMO should prove reproducible from one center to another for the great majority of patients. The capacity to provide extracorporeal perfusion support to patients with refractory and life-threatening respiratory failure without the need to ligate a major artery may decrease the risk associated with this procedure. ■

Opportunity Knocks!

Does your career need a professional growth spurt? Feel as though you would like to make more of a contribution to the field? Here are two opportunities that may help.

Perinatal-Pediatric Specialty Section *Bulletin* co-editor: As co-editor you will assist with the composition and compilation of items for this *Bulletin*. You will facilitate opportunities for the section membership to participate and assist guest editors.

Perinatal-Pediatric Section chair-elect: As chair-elect you will be oriented to the role of section chair. You will join the chair at the 1999 AARC Congress in Las Vegas and assist with some of the conference activities, including the annual section meeting.

If you are interested please contact Peter Betit at the addresses/numbers listed on page 2. ■

UAB Offers Traineeship

The University of Alabama at Birmingham (UAB) Pediatric Pulmonary Center, located at Children's Hospital in Birmingham, AL, continually accepts applications from baccalaureate level respiratory therapists wishing to improve their knowledge of pediatric pulmonary care while pursuing a master's degree in education/allied health sciences.

The year-long traineeship includes 20 hours per week participating with other graduate level health professionals in faculty and guest lectures, hospital rounds, outpatient pulmonary clinics, field trips, patient and family education, discharge coordination, and research. Other activities are planned according to the trainee's individual needs and interests.

Successful completion of the traineeship and five additional graduate-level UAB courses can earn the trainee a master's degree in as little as one year. Applicants who are chosen for the program receive $1,500 in tuition assistance and a $500 per month stipend for the year of their traineeship.

The UAB traineeship has been in existence for nearly ten years. For more information about the program, contact Julie McDougal, RRT, MAE, Pediatric Pulmonary Center, 1600 7th Ave., S., ACC 620, Birmingham, AL35233, (205) 939-9583, FAX (205) 975-5983, e-mail: jmcdougal@peds.uab.edu. ■

1999 Summer Forum

The AARC will hold its annual Summer Forum July 16-18 in Phoenix, AZ. This outstanding meeting promises to provide a wealth of information for practitioners holding positions in management and education and should be of interest to anyone wanting up-to-the-minute information about the profession and where it is headed as we prepare to enter the new millennium.

For more information about the Forum and how you can attend this important meeting, see your April issue of *AARC Times* or visit the AARC's web site at www.aarc.org. ■