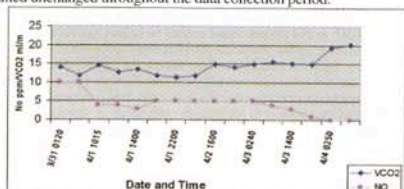


Mini StatCO₂ use w/Epinephrine

CASE STUDY: USING VOLUMETRIC CAPNOGRAPHY (VCO₂) TO AID IN THE WEANING OF INHALED NITRIC OXIDE (INO) IN A CRITICALLY ILL INFANT.
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INTRODUCTION: Monitoring and managing infants with PPHN can be very problematic as it is critical to understand what is dynamically occurring at the pulmonary capillary interface. Pulse oximetry (SpO₂) can also be limiting because these patients are often hyperoxygenated to facilitate the decrease of pulmonary vascular resistance. An infant's SpO₂ will not decrease below 100% until the arterial oxygenation (PaO₂) decreases to ~58 torr due to the presence fetal hemoglobin. This delay can make titrating Nitric Oxide to therapeutic levels difficult. The NICO monitor measures volumetric carbon dioxide (VCO₂), and MValv (alveolar minute ventilation). VCO₂ has been shown to be a sensitive indicator of pulmonary blood flow when measured in conjunction with MValv. If MValv remains constant, and VCO₂ decreases this represents a decrease in pulmonary blood flow, as less CO₂ is eliminated even though minute ventilation at the alveolar level did not change. This is measured noninvasively and continuously to provide a real time dynamic measurement indicative of pulmonary blood flow and to alert clinicians of acute increases in pulmonary vascular resistance.

CASE STUDY: Infant delivered via Cesarean section at 38.4/7 weeks gestation secondary to non-reassuring fetal heart tracing. The infant had respiratory failure at birth secondary to meconium aspiration with progression to persistent pulmonary hypertension of the newborn. The patient was placed on conventional ventilator and INO. The NICO[®] Respiratory Profile Monitor was placed inline with the ventilator circuit. Data was collected on the patient for five days. With each INO wean the VCO₂ blood gas results and ventilator settings were documented. By trending VCO₂ we were able to detect changes in the CO₂ production and dead space prior to obtaining a blood gas sample (see graph below). The patient's oxygen and ventilator settings remained unchanged throughout the data collection period.



DISCUSSION: INO improves oxygenation and V/Q matching in patients with hypoxic lung disease. Using VCO₂ in conjunction with INO therapy can aid the therapist in adjusting respiratory support within prescribed limits. In this patient we were able to effectively wean INO by trending VCO₂ with the NICO[®] monitor. Drawing blood gases only when monitored values fall out of the prescribed range could improve respiratory treatment, reduce the incidence of barotrauma, reduce the exposure time to hypoxemia and hypercapnia and reduce the number and total volume of blood samples required.

CONCLUSION: By studying the VCO₂ graph, we were able to observe ventilation by the shape of the graph and observe immediate feedback on V/Q changes. In this case, we were able to wean and discontinue INO therapy using VCO₂.
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CASE STUDY: A TRIAL COMPARING THE EFFECTIVENESS OF THE MERCURY MINI-STATCO₂ AND THE NELLCOR PEDI-CAP[®] IN THE PRESENCE OF ENDOTRACHEALLY ADMINISTERED MEDICATIONS.
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INTRODUCTION: The new American Heart Association guidelines for intra-hospital resuscitation recommend the use of an exhaled carbon dioxide (CO₂) as the primary means of confirming correct endotracheal tube (ETT) placement. We compared two exhaled CO₂ detectors, Nellcor PEDI-Cap[®] and Mercury Mini-STAT[®], for accuracy of CO₂ detection in a neonatal resuscitation situation in which medications are administered via the ETT.

CASE SUMMARY: We tested several drugs that might be given via the ETT during resuscitation for their effect on the two exhaled CO₂ detectors. Normal saline solution (NSS) was used as the control. Each brand of exhaled CO₂ detector was tested in two ways. First, several drops of each drug were put directly onto the detection filter of the two CO₂ detectors and color change recorded (see chart below). Next, each drug was delivered in an appropriate neonatal dose into a neonatal size test lung via an ETT. A resuscitation bag and CO₂ detector were attached to the test lung and several breaths delivered. Color change in the CO₂ detector was again recorded (see chart below).

Nellcor PEDI-Cap [®]			Mercury Mini-STATCO ₂ [®]		
Drug/Dose	Positive	Negative	Drug/Dose	Positive	Negative
Normal Saline		With vapor and drops	Normal Saline 1 cc	With vapor and drops	
Infasur [®]	1 cc turned yellow	With vapor	Infasur [®]	0.8 cc turned yellow	With vapor
Epinephrine 1:10,000 0.1 mcg	With drops and vapor		Epinephrine 1:10,000 0.1 mcg	With drops	With vapor
Atropine 0.1 mcg	With drops	With vapor	Atropine 0.1 mcg	With drops	With vapor
Narcan [®]		0.2 cc to turn <2%	Narcan [®]	0.1 cc turned yellow	0.1 cc to turn 1-2%

DISCUSSION: Our study shows that a false positive CO₂ detection result can occur when resuscitation medications are used. All of the medications tested caused a false positive result when the medication contacted the detection filter directly. This could occur if medication is splashed on the ETT or coughed up the ETT by the infant. When medications were administered through the ETT into a test lung, epinephrine vapor also caused a false positive result with the Nellcor PEDI-Cap[®] within one to two breaths from the resuscitation bag. The Mercury Mini-STAT[®] CO₂ did not give a false positive result with epinephrine vapor. NSS control did not cause a color change on either exhaled CO₂ detector with drops or vapor.

CONCLUSION: Resuscitation medications can cause a false positive result with exhaled CO₂ detectors. This could result in an incorrect assumption that the infant is successfully intubated. Exhaled CO₂ detectors are a good resource for confirming a successful intubation along with auscultation, but caution should be exercised when used during resuscitation.
OF-06-082

SYNCHRONIZING P_{HIGH} AND P_{LOW} DURING AIRWAY PRESSURE RELEASE VENTILATION IN A PEDIATRIC PATIENT.

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Introduction: Airway pressure release ventilation (APRV) is a ventilator mode aimed at recruiting and maintaining lung volume, while preserving spontaneous ventilation¹. In our center, APRV is applied with the Avea[™] ventilator (Viays HealthCare, Palm Springs, CA). We report the use of this mode and the use of its synchronization features in the care of a 14 year-old male who developed ARDS. **Background:** The patient was a child with Bardet Biedl Syndrome admitted for suspected aspiration during induction for an endoscopy. The patient subsequently developed ARDS, and required high-frequency oscillatory ventilation (HFOV) and muscle relaxation due to worsening lung compliance and gas exchange. Over the 7 day HFOV course, gas exchange improved and the patient was transitioned to APRV in order to promote spontaneous ventilation. Initial APRV settings were: F_IO₂ 0.6, P_{HIGH}/P_{LOW} of 25/0 cmH₂O, and T_{HIGH}/T_{LOW} 6.0/0.8 seconds, Paw 22 cmH₂O. At hour 36 of APRV the patient developed a pneumothorax, received a chest tube, and transitioned to PSV/PEEP of 10/8 cmH₂O in order to minimize further barotrauma. Over the next 48 hours, the patient's gas exchange worsened and work of breathing increased. The mode was changed to SIMV-PCV + PSV with no improvement. In an attempt to avoid HFOV and the need for paralysis, a second trial of APRV was employed with the settings: F_IO₂ 0.7, P_{HIGH}/P_{LOW} of 23/0 cmH₂O, and T_{HIGH}/T_{LOW} of 5.5/0.3 sec, Paw 22 cmH₂O. During the transition to APRV, the patient became tachypneic, tachycardic, hypertensive, diaphoretic, and accessory muscle use was noted. The increase in work of breathing was presumed to be from under-recruited lungs and increases in P_{HIGH} were attempted and were unsuccessful. The APRV synchronization features were added: 20% T_{LOW} Synchron for P_{LOW} to P_{HIGH} transition, and 5% T_{HIGH} Synchron for P_{HIGH} to P_{LOW} transition. After these changes were made, work of breathing and RR subsequently decreased, and the patient appeared more comfortable. Blood pressure and heart rate returned to clinically acceptable levels, and there was no recurrence of air leak. Gas exchange improved over the next 9 days and the patient was transitioned from APRV settings of F_IO₂ 0.45 P_{HIGH}/P_{LOW} 16/0 cm H₂O T_{HIGH}/T_{LOW} 10/0.7 sec, to PSV/PEEP of 12/8 cmH₂O. The patient was successfully extubated 2 days later. **Discussion:** The addition of T_{LOW} Synchron and T_{HIGH} Synchron in this case improved the patient/ventilator interaction during APRV. The APRV advanced settings of T_{LOW} Synchron and T_{HIGH} Synchron are adjusted as a percentage of T_{LOW} and T_{HIGH} and are unique to the Avea[™]. It is possible that the lack of improvement and the development of a pneumothorax may have been avoided if the synchronization features were used in the initial APRV attempt. Prior to this patient, we had not used this feature as part of our APRV settings. This case has assisted us with further developing our APRV protocol, and we now routinely synchronize to improve patient/ventilator interactions. Clinical trials are warranted to validate the benefits of synchronization during APRV.

1. Habashi N, Andrew P (2004). Ventilator strategies for posttraumatic acute respiratory distress syndrome: airway pressure release ventilation and the role of spontaneous breathing in critically ill patients. *Curr Opin Crit Care* 10(6):549.
OF-06-084

CARDIOPULMONARY INTERACTIONS DURING A VENTILATOR MODE CHANGE IN A PEDIATRIC PATIENT Michelle Lilley RRT, Patrice Benjamin RRT, David A. Turner, MD Children's Hospital Boston and Harvard Medical School, Boston, MA

Introduction: Airway pressure release ventilation (APRV) is a mode aimed at recruiting and maintaining lung volume, while preserving spontaneous ventilation¹. We report an episode of dramatic cardiopulmonary interaction involving a pediatric patient who was switched from APRV to PCV+PSV. **Case Summary:** A 3 yr old girl with Kaposiform hemangioendothelioma required prolonged mechanical ventilation following an exploratory laparotomy. PCV+PSV parameters were rate 22, PIP/PEEP 30/10 cmH₂O, PSV 20 cmH₂O, and F_IO₂ 0.85. Paw 19 cmH₂O, mandatory V_T 5 mL/kg, spontaneous V_T 2.0 mL/kg, and ABGs were pH 7.33, PaCO₂ 71 mmHg, PaO₂ 85 mmHg. APRV was initiated due to worsening gas exchange, enlarged abdominal girth, increasing ventilator support, and the need to preserve spontaneous ventilation. Initial APRV settings were P_{HIGH}/P_{LOW} 24/0 cmH₂O, T_{HIGH}/T_{LOW} 7.0/0.4 sec, F_IO₂ 0.6. Paw was 24 cmH₂O, and release V_T 8 mL/kg. ABG was pH 7.36, PaCO₂ 64 mmHg, PaO₂ 130 mmHg. The patient required continuous veno-venous hemofiltration (CVVH). Due to hemodynamic instability, despite inotropic support, CVVH output was limited leaving the patient in a persistent fluid positive state. On day 4 of APRV parameters were P_{HIGH}/P_{LOW} 28/0 cmH₂O, T_{HIGH}/T_{LOW} 5.6/0.4 sec, F_IO₂ 0.45. Paw was 27 cmH₂O, and release V_T 12 mL/kg. ABG was pH 7.20, PaCO₂ 71 mmHg, PaO₂ 65 mmHg. A CXR showed an essentially clear left lung with sharp costophrenic angle and an opacified right lung. The patient continued to require inotropic support, CVVH output was suboptimal and fluid status persistently positive. Due to the lack of appreciable uniform alveolar disease by CXR and ongoing hemodynamic instability, a trial of PCV+PSV was attempted. PCV+PSV parameters were rate 22, PIP/PEEP 30/12 cmH₂O, PSV 20 cmH₂O, and F_IO₂ 0.5, Paw 19 cmH₂O, mandatory V_T 8 mL/kg, and spontaneous V_T 7 mL/kg. ABG was pH 7.23, PaCO₂ 69 mmHg, PaO₂ 134 mmHg, and concomitantly the patient's BP improved, inotropic support was decreased, and CVVH output was optimized. The patient's fluid status became increasingly negative. **Discussion:** While we achieved the goal of preserving spontaneous ventilation in this patient, APRV does not appear to have been the optimal mode. The unilateral nature of the lung disease may have led to lung overdistention and subsequent detrimental cardiovascular effects. Ongoing assessment of clinical data to determine the appropriateness of the settings and mode is an important part of daily clinical practice. We utilize a CPG to assist with patient selection and the use of this mode. Development of a CPG to help guide the team at the bedside is one way to help optimize patient care.

1. Habashi N (2004). Ventilator strategies for posttraumatic acute respiratory distress syndrome: airway pressure release ventilation and the role of spontaneous breathing in critically ill patients. *Curr Opin Crit Care* 10(6):549-57. OF-06-139