



Adult Acute Care

May / June 2002

Bulletin

Notes from the Editor

by Jeff Whitnack, RRT, RPFT

In this issue we feature an article that holds significant implications for respiratory therapists. Here's why:

Unique among the body organs, the lungs "avail themselves" to therapeutic and resuscitative mechanical intervention. Not the heart, nor the brain, nor the liver, kidneys, etc., can compare. And once that boundary has been traversed the clinician must then confront the ramifications and signals pertaining to and arising from the other organ systems. Does mechanical ventilation impair or improve hemodynamics? How does one apply mechanical ventilation in the setting of a closed head injury? If the abdomen is swelling, what is the best ventilation strategy? Then there are specific lung pathologies, such as ARDS and asthma, which mandate that ventilatory strategies stray a bit from the norm in large part because of the potential impact on other organ systems.

Keith Lamb's case study addresses these issues by exploring the intricacies of ventilating patients with the complicated condition known as abdominal compartment syndrome. I know I found the article intriguing, and I believe you will as well. ♦

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Polytraumatic Abdominal Compartment Syndrome: Part One

by Keith D. Lamb, RCP, RRT, charge respiratory therapist,
Christiana Care Health Services, Newark, Del

Many critically ill patients suffer from a serious complication known as abdominal compartment syndrome (ACS). ACS can be a manifestation of multi-system organ failure (MSOF), systemic inflammatory response syndrome (SIRS) and many other etiologies, including serious injury. Vigilance is required on the part of the respiratory therapist in recognizing this syndrome in its early stages and most importantly, RTs must be capable of recommending appropriate methods to effectively ventilate these challenging patients.

Recent literature relates severity of ACS to fluid input/output and peak inspiratory pressures. Following surgical intervention of severe abdominal trauma, the closure of the abdomen can cause severe tension, reduction of cardiac output, reduction of perfusion to all abdominal organs except the adrenal gland, renal failure, and grossly impaired ventilatory mechanics. These clinical manifestations are generally described as ACS.

The following case study pertains to a typical polytrauma victim.

Day one

A 47-year-old female presented to our emergency department s/p MVC, vehicle versus vehicle. She was the restrained driver of a side impact collision with a 20% intrusion on the driver's side of her subcompact car. The patient was found responsive in the field with a GCS of 13. At the scene, she was complaining of dyspnea, suprasternal pain and moderate abdominal pain. She was slightly hypotensive, tachypneic and tachycardic (sinus). Breath sounds were clear and slightly diminished on the left with no crepitice. She was cannulated with two large bore IVs and fluid management was initiated with crystalloids. Supplemental oxygen was administered via NRB. Extrication time from her vehicle was approximately 20 minutes.

Upon arrival at our emergency department her condition had deteriorated somewhat. Her initial survey revealed an intact airway. Breath sounds were clear and very diminished on the left. She was hypotensive, tachypneic, tachycardic, diaphoretic and complaining of extreme dyspnea. Her abdomen was very tender and she was complaining of extreme pain in her left arm. She had an obvious left humerus fracture. Rectal exam was normal. There was no obvious step off or other spinal deformity. She had slight abrasions on her chest and belly.

Blood was drawn and a trauma panel sent to the lab. She was crossed and matched for blood. A FAST exam of her belly showed probable free fluid, and a DPL was positive for free blood. Plain films of the chest and c-spine were taken. A left chest drain was placed, after which several hundred cc's of blood were drained. Immediately after the chest drain was placed the patient became profoundly hypotensive. She was intubated via RSI. Succinylcholine, lidocaine and etomidate were administered to facilitate intubation with an 8.0 endotracheal tube. Transfusion of whole blood was started via rapid infuser.

The patient's initial CXR showed a left pneumothorax, several rib fractures and a slightly widened mediastinum. C-spine films were unremarkable. The patient was taken to the angiography suite and an aortogram was performed, which was also unremarkable. Her blood pressure was stabilized and she was taken to the operating room. An open laparotomy was performed, and a perforated small bowel was repaired. A splenectomy was also performed.

The patient was closed and taken to the Surgical Critical Care Complex. She was initially placed on CMV (f) 12, (VT) 700ml, FI02 1.0, and (PEEP) 5 via Drager E-4. Central venous access was obtained via a right subclavian line and CVPs were transduced. An arterial line was placed in the operating theater. An initial ABG revealed a metabolic acidemia,

New JCAHO Initiative Targets ICU Quality

Outcomes-based measures for assessing the quality of hospital intensive care units are on their way. Working with the Leapfrog Group, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) is convening an expert panel to determine an ICU measurement framework and formulate an initial set of specific performance measures. As part of this process, JCAHO is issuing a call for existing measures that address care in medical, surgical and medical/surgical intensive care settings.

To submit potential sources of ICU core measures, visit the Joint Commission's web site at www.jcaho.org/perfmeas/coremeas/cm_frm.html, or contact Nancy Lawler, associate project director, at (630) 792-5937 for additional information. ♦

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POLYTRAUMATIC ABDOMINAL COMPARTMENT SYNDROME: PART ONE

hyperoxemia, pH 7.23, a base deficit of 11, and a PaO₂ of ~400 mmHg. FI_{O2} was titrated to 40% within an hour. A STAT portable CXR was obtained after line placement. Hemothorax was resolved while chest drain was on continuous suction. Her humerus fracture was reduced and casted by the ortho resident.

Day two

Twenty-four hours later the patient was not looking well. She was showing signs of sepsis/infection, was febrile, and her CBC was indicative of active new infection. Other labs (bun, creatine, etc.) were also indicative of impending organ involvement. Her CXR was now completely white. FI_{O2} was 1.0 and PEEP was 15.

The patient became extremely agitated. Continuous propofol was started and she again became profoundly hypotensive. A norepinephrine drip, as well as dopamine, was started, and a pulmonary artery catheter was placed. PA pressures were elevated, PAWP was normal, and cardiac output was slightly decreased. CVPs were low.

Upon physical exam, there was no tracheal deviation. Auscultation of the lungs revealed scattered rhonchi that resolved with tracheal suctioning. There were decreased breath sounds on the left, with an identifiable air leak, and chest excursion was equal. Her abdomen was very distended/rigid. An OG tube was set to continuous suction; her left arm was slung and elevated.

An ABG indicated a mixed acidemia; pH 7.11, PaO₂ 48. The lactate level was increased. Other labs were worsening. Her current vent settings were CMV(f) 14, (VT) 700, (PEEP) 15, and (FI_{O2}) 1.0. PIPs were ~65, and static pressures ~40. The patient remained somewhat agitated and her propofol was increased. A cisactacurium drip was started. There were no changes in her static pressures after the NMB. Her abdomen remained rigid, distended. CXR was ominous-looking. Bladder pressures using the foley catheter were ~35 mmHg.

Also of interest is this patient's CVP. Although it had been slightly decreased, it now became significantly decreased. The clinical picture was increasingly looking like a systemic inflammatory response. To rule out cardiogenic issues, an ECHO was performed, which showed no effusion, ventricular dysfunction, or other abnormality. But this patient was still hemodynamically unstable.

The patient was placed on PCV+ (f) 20, (PIP) 35, (FI_{O2}) 1.0, (PEEP) 15, and an (I:E) of 1:1.5. She remained very difficult to oxygenate or ventilate; pH 7.2 and PaO₂ of 55. A couple of amps of HCO₃ were given and THAM (tromethamine) was started, which improved her pH. Her hypoxemia was still unresolved.

Within an hour, one of our trauma attendings was at the bedside to do something about this patient's intra-abdominal pressures. Her abdomen was opened and a Bogota Bag was placed over the abdominal contents. The RRT was present during the procedure and titrated the PIPs to maintain consistent tidal volumes. Serial ABGs were obtained over the next couple of hours, and vent settings were manipulated to maintain isafei distending pressures and reasonable acid base. Her pH and paO₂ slowly improved.

The outcomes

Over the next 96 hours this patient continued to improve. Her pH reached 7.32, paO₂ 90 on PCV (f) 18, (PIP) 20, (FI_{O2}) 60%, and (PEEP) 10, (I:E) 1:2. Pharmacologically, she remained on renal dose dopamine, broad spectrum antibiotics, maintenance fluids, lorazepam and MSO₄. Her propofol was titrated and her cisactracurium stopped.

Over the next two weeks the patient was placed on CMV and then PSV. Unable to be weaned any further due to tracheal secretions, she was tracheostomized at the bedside via percutaneous tracheostomy without complication. She was weaned without incident, placed on trach collar, decannulated, and discharged to home. Her total length of stay was approximately five weeks.

Serious business

Abdominal compartment syndrome is serious business. Never mind that this patient was already seriously compromised due to ARDS and SIRS.

ACS can be very difficult to manage. Many modalities that can be used to "fix" one aspect of the syndrome can exacerbate others. For example, PCV used to treat hypoxemia can cause further detriment to venous return and systemic blood pressure. Fluid challenges to treat hypotension can accelerate the effect of leaky capillaries, interstitial flooding and alveolar flooding, and a resultant decrease in pulmonary compliance ensues. Hyperpermeable capillaries and alveolar flooding most certainly cause a reduction in diffusion capacity and exacerbate hypoxemia.

ACS can have a significant impact on many organ systems, dramatically increasing mortality in the critically ill patient. The patient in this case study was fortunate that issues were found

and dealt with in a timely manner. Oftentimes this is not the case, and optimal outcomes are not achieved. The following list touches on the effects ACS can have on other systems and represents things to consider when helping to manage these complex patients:

SIRS is not uncommon in patients who have experienced shock, in our case hypovolemic and multi-system in nature. It seems to be somewhat more prevalent in patients who have been fluid resuscitated with crystalloids. As a result of this inflammatory response, our patient was compromised in several ways.

Abdomen: Normal intra-abdominal pressures are zero. IAPs > 20 are suggestive of ACS and pressures > 30 are diagnostic. IAPs can be measured directly or indirectly, by use of the orogastric tube or foley catheter.

Pulmonary: Non-cardiogenic pulmonary edema with normal left ventricular function (echo and normal PCWP), diffuse CXR abnormalities, and paO_2/FiO_2 of ~48 paint the classic ARDS picture. Add the ACS and its resultant influence on diaphragmatic excursion and this patient is going to be very difficult to oxygenate and ventilate.

Cardiovascular: If this patient's number one pathology was sepsis, one would have expected her cardiac output to be increased. However, increases in intra-abdominal pressures do funny things to cardiodynamics. Initially, her CVPs were marginally low. This is probably due to the fact that she was slightly under-resuscitated and marginally hypotensive. However when IAPs are elevated to such an extent, there is a resultant increase in intra-thoracic pressures, decrease in venous return via the inferior vena cava and increase in PA pressures and unacceptable pre-load. Finally, a drop in CO will ensue.

Renal: This is a major issue. Resultant compression of renal vasculature due to increased IAPs is very common. Oliguria/anuria are the most common problems. It is not unusual for these patients to need continuous hemodialysis.

Cerebral: Increases in IAPs and intra-thoracic pressures will cause a decrease in jugular venous return and increase in intra-cranial pressures. Systemic hypotension can also cause further decreases in cerebral perfusion pressures.

This case study had a happy ending. Her course was optimal and she escaped many complications of ACS. But consider the following: What if she did not improve after opening her belly? Would you or could you prone her with an open belly? What if she did not begin to ventilate effectively after her abdomen was opened? Could you use tracheal gas insufflation, HFOV, or continue tromethamine infusion? ♦

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Off Pump Better for Bypass

An analysis of data from two studies comparing traditional bypass surgery with off pump bypass concludes the off pump method results in fewer complications and shorter hospital stays.

The report, which appeared in the April 6 issue of *The Lancet*, found off pump patients were 25% less likely to experience atrial fibrillation and 12% less likely to develop a chest infection during the first month after surgery. They were 13% less likely to spend more than a day in the ICU or more than a week in the hospital altogether.

The two studies involved more than 400 patients who were randomly assigned to receive either traditional bypass or the off pump surgery. ♦

The AARC Needs You!

Did you know it takes more than 500 active volunteers to successfully run the vast and varied programs and services offered by the AARC every year? Who should take on these responsibilities? How about you?

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Delayed Treatment Leads to Higher ICU Admissions

A three-year study involving 17 acute-care hospitals in Canada found that initially missed diagnoses and delayed treatment for patients with active tuberculosis (TB) were not only common, but were strongly associated with late admission to the intensive care unit and in-hospital death. According to the researchers, as the number of TB admissions to the hospitals decreased, the outcome for patients worsened, with delays in diagnosis and treatment becoming more frequent, increasing the risk of exposure to TB for hospital personnel and other patients.

The study involved 429 patients newly diagnosed with active pulmonary TB following hospitalization between June 1992 and June 1995. Appropriate treatment was delayed one week or more in 127 cases, or 30%. Among the patients with delayed treatment, the median interval from admission until isolation was 12.5 days. Of the patients who were newly diagnosed with active pulmonary TB following hospitalization, 52, or 12%, died. Death was associated with older age, HIV infection and ICU admission.

The study was published in the first April issue of the *American Journal of Respiratory and Critical Care Medicine*. ♦

Docs Give Conflicting Opinions

An informal survey of about 300 British doctors found nearly all of the physicians polled agreed that patients who are deemed competent should be allowed to refuse life-sustaining treatment. But when asked if they, personally, would be willing to withdraw treatment from a patient who had asked them to do so, even if it meant ending the patient's life, just 61% responded in the affirmative. Twenty-eight percent of the doctors said they would not withdraw the treatment and 11% were unsure how they would respond. About half said they believed the courts should determine whether a patient was competent or not, while the rest either said the courts should not decide or they did not know the answer to the question.

The survey, which was conducted by the British Medical Association's BMA News Review, was spurred by a case in England involving a woman who was fighting for the right to be disconnected from the ventilator that was keeping her alive. ♦

SPECIALTY PRACTITIONER OF THE YEAR:

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