

# ANESTHESIOLOGY™ 2014

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Session: L117  
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## Management of Severe ARDS in the ICU With ECMO

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**Disclosures:** This presenter has no financial relationships with commercial interests

### Stem Case and Key Questions Content

As the ICU attending, you are asked to care for a 21 year old, 50 kg female with no significant past medical history transferred from the ER with a diagnosis of pneumonia. She is intubated and mechanically ventilated. Her vital signs are:

P 140 beats/min BP 85/55 RR 25 SpO<sub>2</sub> 93%

Ventilator settings

AC rate 10 tidal volume 600 FIO<sub>2</sub>: 0.7 peep 5

She is sedated with an ativan infusion. Her CXR reveals multifocal infiltrates with a moderate sized right pleural effusion. The ED physician started empiric levofloxacin, and sent blood, urine and endotracheal aspirate cultures.

Her first arterial blood gas results are:

7.26/42/69.6/18/-7.8/93%

**Question 1:** Does this patient meet criteria for ARDS? What ventilator changes should be made?

**Question 2:** Is a computed tomography (CT) scan indicated at this time? What additional information could it provide to guide clinical-decision-making of this presentation?

**Question 3:** Culture data is pending. Should the antibiotic regimen be altered and how?

**Question 4:** What additional monitoring would you require to care for this patient?

CT scan demonstrates a loculated pleural effusion. A chest tube is placed by interventional radiology, which drains purulent fluid. Shortly after the procedure her chest tube develops a continuous air leak. Her vital signs and ventilator settings are

P 135 bpm, BP 75/32 mmHg, RR 25 breaths/min, SpO<sub>2</sub> 94 % (100 FIO<sub>2</sub>)

**Question 6:** Is any invasive hemodynamic monitoring indicated? Would a pulmonary arterial catheter add any useful information?

**Question 7:** What other therapies may benefit this patient? Would you consider proning this patient?

**Question 8:** What do you expect the intravascular volume status to be? Would you aggressively resuscitate to increase intravascular volume?

The patient's condition continues to worsen after switching to low stretch ventilation. The ventilator settings are AC Vt 300 RR 28 Peep 10 FIO<sub>2</sub> 1.0 and the ABG is 7.18/58/72/25/-

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3.6%/89% on flolan and a vecuronium infusion. VSS are T 100 HR 120 BP 85/50

**Question 10:** Would you consider ECMO for this patient? Would you use veno-venous or arterial? What further information do you need to decide?

**Question 11:** When discussing ECMO with the family, what time frame would you offer for possible recovery and length of time on ECMO?

You decide that the patient would benefit from ECMO support, and the patient is transported to the operating room for percutaneous veno-venous ECMO. The intraoperative echocardiogram shows normal biventricular function. Bicaval cannulation is successful and veno-venous ECMO is initiated. After initiation of ECMO, the patient's ABG is 7.38/32/428/19/-5.5/100%.

**Question 11:** What ventilator changes should be made based on this ABG and the presence of a large bronchopleural fistula?

**Question 12:** What anticoagulant would you use to prevent thromboembolic complications of ECMO? If the patient had a known PFO would that change your management? What are your goals for level of anticoagulation (labs)?

The patient develops bleeding from the chest tube that is draining the empyema. The volume of bleeding is about 200 mL/hour. Platelet count is 93 and the fibrinogen level is 300 mg/dL.

**Question 13:** What would your red cell transfusion trigger be for this patient? Would you transfuse platelets? Would you stop the anticoagulation? If so, for how long?

After stopping the heparin temporarily, the bleeding stops. The bleeding starts again after restarting heparin and the patient is taken for a VATS procedure when a large effusion is visualized on chest x-ray. After cauterization of bleeding, the patient has no further bleeding. Over the course of the next several weeks, the patient's lungs improve.

**Question 14:** How would you wean the ECMO as the patient improves to evaluate the patient's ability to maintain independent function once decannulated?

**Question 15:** At what point would you perform a tracheostomy on the patient since she is on therapeutic anticoagulation?

## Model Discussion Content

ARDS remains a common problem associated with a high rate of mortality and morbidity. A thorough understanding of the pathophysiology and lung protective ventilation strategies is crucial in the management of this disease. Furthermore, evidence is emerging that therapies such as neuromuscular blockade, prone positioning, and ECMO may improve survival. ARDS is an inflammatory reaction in the lungs in response to pulmonary or systemic insults characterized by acute hypoxemia and bilateral pulmonary infiltrates in the absence of left heart failure.(1) Inflammatory cytokines and leukocytes cause injury to pulmonary vascular and alveolar endothelium resulting in profound capillary leak with protein rich alveolar edema. This damage impairs gas exchange and culminates in hypoxemia and hypercarbia. The heterogeneous nature of inflammation and damage produces regions of lung with vastly different compliance. The severely damaged areas are relatively non-compliant and areas with

less injury are more compliant. The use of life-saving mechanical ventilation may temporarily improve oxygenation and carbon dioxide excretion, but places the patient at risk for ventilator induced lung injury. The sentinel ARDSnet study defined the open lung, low-stretch ventilation strategy which has become the standard of care in ARDS patients.(2) Lung protective ventilation as defined by the ARDSnet group is a tidal volume of 4-6ml/kg of predicted body weight and a plateau pressure  $\leq 30$  cmH<sub>2</sub>O.

Defining the severity of ARDS may help to stratify patients and the need for escalation of therapy. In 2012, the ARDS Definition Task Force Berlin stratified ARDS into mild, moderate or severe based on PaO<sub>2</sub> to FiO<sub>2</sub> ratio, time criteria of one week from known clinical insult or worsening respiratory symptoms, and PEEP level.

Additional therapies have been studied and shown benefit in patients with ARDS. Neuromuscular blockade may improve oxygenation, but may result in ICU-acquired muscle weakness. In 2010, a multi-center randomized trial found that patients with severe ARDS treated with muscle relaxants for 48 hours improved 90 day survival and significantly lower ventilator days.(3) Furthermore, there was no difference in rates of ICU acquired paresis between the neuromuscular blockade and placebo groups.

Prone therapy has gained renewed interest in after the publication of recent trials showing a significant survival benefit.(4) Proposed benefits of prone positioning include improved oxygenation by promoting alveolar recruitment and altering the transpulmonary pressure gradient which may lower the risk of ventilator associated lung injury. The ideal length of time that patients should be left in the prone position is debatable, and rigorous studies comparing durations of prone positioning have yet to be performed. The actual procedure of turning the patient to the prone position requires an experienced team, and risks of proning include dislodgement of cannula and devices, and there is potential for pressure point injury. In addition, there is a limited ability to perform cardiopulmonary resuscitation in the prone position. Early studies analyzing the use of extracorporeal membrane oxygenation (ECMO) as a bridge to survival in patients with severe acute respiratory distress syndrome (ARDS) were not able to show a reduction in mortality. Ventilation management strategies to reduce ventilator associated lung injury have reduced mortality from ARDS, but the high mortality rate of severe ARDS has spurred renewed interest in ECMO as a salvage therapy. Advances in technology including improved membrane oxygenators and pumps have reduced the inflammation and cell trauma associated with ECMO, and decreased the thrombogenic properties of the ECMO circuit. Furthermore, the use of veno-venous ECMO for pulmonary support has reduced the complications associated with arterial cannulation including arterial injury and stroke. For patients with severe ARDS, use of veno-venous ECMO to facilitate low stretch ventilation has been demonstrated to improve survival.(5,6) Results of recent trials and experience with this newer modality will likely facilitate more widespread use of ECMO. At this time there is limited data to suggest which patients would benefit from ECMO, and once instituted, which patients should be continued on ECMO.

Some indications for ECMO in respiratory failure include a PaO<sub>2</sub> to FiO<sub>2</sub> ratio of less than 80 mmHg despite high levels of PEEP, uncompensated hypercapnia with acidemia and excessively high end-inspiratory plateau pressures despite optimized ventilation strategies. An absolute contraindication to ECMO is any condition that inhibits the use of anticoagulation

therapy as the most common life-threatening patient related complications associated with ECMO are thrombosis and bleeding.

There are three general configurations for ECMO: veno-venous (VV), veno-arterial (VA) and arteriovenous (AV). VV and AV are used with patients who have refractory pulmonary failure and stable cardiovascular status, while VA ECMO is used in patients with cardiogenic shock or cardiopulmonary failure.(7) From a physiologic standpoint, the VV approach relies on the artificial ECMO pump to maintain flows while the AV approach which can be thought of as an “artificial lung” does not utilize a pump, but rather on the patient’s own blood pressure to drive flow. As such, AV ECMO requires cardiovascular stability to be successful. Generally, VV ECMO is used in ARDS while AV ECMO is less effective in oxygenation and is reserved for refractory respiratory failure with CO<sub>2</sub> retention in an otherwise stable patient.

In general, the ECMO weaning process can commence as arterial oxygenation, chest radiograph, or lung compliance improves. Ventilator support is increased as the rate of blood flow and FdO<sub>2</sub> in the ECMO circuit is gradually decreased over a period of hours. As ventilator settings are adjusted to levels that would be acceptable without ECMO support and the ECMO FdO<sub>2</sub> is reduced to 0.4 to 0.6 with a PaO<sub>2</sub> greater than 80 mmHg, an “ECMO off” trial can be considered. During this trial blood flow and anti-coagulation are maintained while the oxygenator is disabled. Patient PaO<sub>2</sub> and PaCO<sub>2</sub> and SaO<sub>2</sub> are closely monitored. If the PaO<sub>2</sub> is maintained with acceptable ventilator settings for an hour or more without the ECMO oxygenator, then the patient is ready for decannulation.

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