Post-Arrest Ventilator Management

Key Article

- *Johnson NJ, Carlborn DJ, Gaieski DF. Ventilator management and respiratory care after cardiac arrest. Chest. 2017. [epub ahead of print]*

Background

- Over 500,000 patients experience cardiac arrest each year in the US
- A number of critical care interventions have been shown to impact outcome
    - Targeted temperature management
    - Hemodynamic optimization
    - Appropriate neuroprognostication
    - Meticulous respiratory care
- Abnormal O2 and CO2 tensions are associated with worse outcomes
- Early pulmonary infection seen in approximately 50% of all patients
- Patients at high risk for development of ARDS
- What is the optimal approach to mechanical ventilation that will improve outcomes in these patients?

Post-Cardiac Arrest Syndrome

- 4 key components to PCAS
    - Brain injury
    - Myocardial dysfunction
    - Systemic ischemia and reperfusion injury
    - Persistent precipitating etiology/mechanism of arrest
- Results in…
    - Oxidative stress
    - Coagulopathy
    - Widespread inflammation
    - Multiorgan dysfunction
- Brain particularly sensitive to alterations in macrocirculatory and microcirculatory blood flow, impaired cerebral autoregulation, and abnormal metabolism
- Hyperoxia may exacerbate free radical production, mitochondrial dysfunction and neuronal injury
- Secondary cerebral injury also common, as patients with PCAS have elevated cerebrovascular resistance

- Finally, PCAS patients are at risk for immune dysfunction – more susceptible to infection
- TTM and hemodynamic optimization shown to improve outcomes in patients with PCAS
- What about oxygenation, ventilation, and low tidal volume strategies?

Oxygenation

- Must strike a balance between sufficient oxygen delivery to meet the metabolic needs of the cells while avoiding hyperoxia and the potential injury from excess oxygen present during ischemia and reperfusion
- Hypoxemia – produces ongoing ischemia, irreversible cellular injury, organ dysfunction
- Hyperoxemia – may increase oxidative stress, amplify free radical production, worsen organ function
- Preclinical studies – animals had improved outcomes and decreased neuronal injury when receiving lower FiO2 post-arrest
- Many retrospective analyses have been performed to further investigate relationship between O2 tension, mortality, and neurologic outcome in post arrest patients
    - 100% FiO2 associated with higher NSE levels (neuron-specific enolase) compared with lower FiO2 levels
    - Hyperoxemia (PaO2 > 300 mm Hg) associated with increased mortality
    - Higher PaO2 associated with mortality and poor neurologic outcome
    - Other studies with mixed results
    - These retrospective studies used a variety of cutoffs for hyperoxemia and hypoxemia, blood gas sampling intervals, and duration of exposure to O2
- The Hyperoxic or Normoxic Therapy trial the only prospective trial of titrated O2 after OHCA
    - All patients had initial rhythm of vfib or pVT and were randomized to titrated O2 therapy to SpO2 of 90-94% or standard therapy with SpO2 > 95%
    - Trial stopped early after just a few patients reached a prespecified end point
    - Provided no real information
- Oxygen-ICU trial
    - Reviewed previously on CCPEM
    - Not sure of how many patients were post-arrest
- **Recommendation**
    - **Titration of FiO2 to maintain SpO2 between 92%-97% (approximates a PaO2 up to 100 mm Hg) immediately after ROSC**

Ventilation

- Numerous patients with ROSC after OHCA have abnormal PaCO2 values
- Arterial hypocarbia has consistently been associated with poor neurologic outcomes after cardiac arrest; can cause excessive cerebral vasoconstriction and worsen ischemia
- Relationship between hypercarbia and neurologic outcome less clear

- Mechanism behind association of arterial CO2 and neurologic outcome may be influenced by…
    - Hemodynamic effects of positive pressure ventilation – leads to an increase in intrathoracic pressure - impairs venous return, increases right ventricular afterload – lowers cardiac output – poor organ perfusion
    - Cerebral vasoconstriction and hypoxia
    - Modulations of ischemia reperfusion injury
    - PaCO2 also directly affects the vasculature after cardiac arrest – major regulatory of cerebral blood flow
- Current studies suggest that mild hypercarbia may be beneficial
    - Phase II and phase III trials
    - Patients randomized to mild hypercarbia had lower concentrations of NSE
    - Emerging concept that requires further study
- **Recommendation**
    - **Titrate respiratory rate to target a PaCO2 of 40-50 mm Hg**

Low Tidal Volumes

- Post-arrest patients at significant risk for lung injury
    - Aspiration – 30% have witnessed emesis periarrest
    - Pulmonary contusion – 40% experience contusions from CPR
    - Ischemia-reperfusion injury – affects lungs
    - VILI
    - Infection – early onset pneumonia is common
- Substantial overlap between pathophysiology of ARDS and PCAS
- Epidemiology of lung injury and ARDS after cardiac arrest still not well characterized
- Optimal mechanical ventilation strategy not well defined – BUT existing data suggests a role for low tidal volumes
- Current studies suggest an association between lower tidal volumes (<= 8 ml/kg PBW) and favorable neurologic function
- **Recommendation**
    - **Patients with ARDS post-arrest should be ventilated according to ARDSNet low-tidal volume strategy with Vt <= 6 ml/kg PBW**
    - **Patients without ARDS post-arrest may benefit from low tidal volumes strategy**

Pulmonary Infection

- Infection is common post-arrest
- Incidence reported to be up to 70%
- Risk factors for pneumonia – witnessed aspiration, pre-existing dementia, noncardiac causes for arrest, elevated levels of PEEP

- There is conflicting data regarding the association between TTM at 33C vs. 36C and the incidence of pneumonia; in a multivariable analysis TTM at 33C was associated with early onset pneumonia
- Diagnosis of pneumonia post-arrest is challenging: reperfusion injury and systemic inflammation can appear similarly
- An approach to diagnosis…
    - Consider prearrest and periarrest factors such as aspiration, altered consciousness, swallowing dysfunction, emesis, and difficult intubation
    - In patients with suspected aspiration - obtain samples and culture from the lower respiratory tract (bronch or endotracheal aspirate)
    - Consider ABX in patients with a high risk of bacterial pneumonia – new opacities on CXR, leukocytosis or leukopenia, purulent tracheobronchial aspirates
- Delaying ABX until diagnostic certainty has been associated with significant mortality

Summary

- **Target normal arterial oxygen tension**
- **Target high-normal CO2 tension**
- **Use tidal volumes of 6 to 8 ml/kg PBW**
- **Carefully evaluate for pneumonia**