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1

Ventilation and gas exchange management after cardiac arrest

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For several decades, physicians had integrated several interventions aiming to improve the outcomes in post-cardiac arrest patients. However, the mortality rate after cardiac arrest is still as high as 50%. Post-cardiac arrest syndrome is associated with high morbidity and mortality due to not only poor neurological outcome and cardiovascular failure but also respiratory dysfunction. To minimize ventilator-associated lung injury, protective mechanical ventilation by using low tidal volume ventilation and driving pressure may decrease pulmonary complications and improve survival. Low level of positive end-expiratory pressure (PEEP) can be initiated and titrated with careful cardiac output and respiratory mechanics monitoring. Furthermore, optimizing gas exchange by avoiding hypoxia and hyperoxia as well as maintaining normocarbia may improve neurological and survival outcome. Early multidisciplinary cardiac rehabilitation intervention is recommended. Minimally invasive monitoring techniques, that is, echocardiography, transpulmonary thermodilution method measuring extravascular lung water, as well as transcranial Doppler ultrasound, might be useful to improve appropriate management of post-cardiac arrest patients.

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Introduction

In these last years, physicians had integrated several interventions such as early revascularization-targeted temperature management (TTM), use of vasopressor drugs, controlling of seizures as well as metabolic disturbance aiming to improve the outcomes in post-cardiac arrest patients. Furthermore, early cardiopulmonary resuscitation (CPR) before the arrival of emergency medical services (EMS) was associated with the improvement of survival rate compared with no CPR before the arrival of EMS [1,2].

However, the mortality rate after cardiac arrest is still high as 50%, and it is believed to be secondary to a unique pathophysiological process after return of spontaneous circulation (ROSC) namely the post-cardiac arrest syndrome. Post-cardiac arrest syndrome is the phenomenon including systemic inflammatory response aggravated by the ischemia–reperfusion injury as a result of multiple organ dysfunctions. The post-cardiac arrest syndrome is defined as neurological injury, myocardial dysfunction, and whole-body ischemia and reperfusion response [3,4]. Not only brain injury but also cardiovascular instability and respiratory dysfunction may be associated with poor mortality outcome. Thus, the optimal setting of mechanical ventilation after cardiac arrest may improve the patient's condition by maintenance of normocapnia, prevention of hypoxemia, improvement of brain perfusion, hemodynamic stability, and minimizing ventilator-associated lung injury (VALI). However, there are limited data on the role of different ventilatory strategies and gas-exchange management on mortality and neurological outcome of these patients.

In the present review, we systematically searched the PubMed database for articles published from 2000 to 2015 using the following keywords: Cardiac arrest or Heart arrest, cardiopulmonary resuscitation, Acute Respiratory Distress Syndrome, Acute Lung Injuries, Ventilator-Induced Lung Injury, pneumonia, Mechanical Ventilation, pulmonary gas exchange, oxygen, carbon dioxide, and blood gas analysis. We aim to discuss the following: (1) pathophysiology of pulmonary complications after cardiac arrest and clinical outcomes; (2) ventilation and oxygenation during CPR; (3) the prevention of lung injury after cardiac arrest; (4) the gas-exchange management in patients with cardiac arrest after ROSC; and finally (5) we propose the general bundle of ventilator management in and monitoring cardiac arrest patients.

Pathophysiology of pulmonary complications after cardiac arrest

Robert et al. have recorded extra-cerebral organ dysfunction using the sequential organ failure assessment (SOFA) score over the first 72 h after ROSC in adult patients resuscitated from cardiac arrest. They showed that the highest cardiovascular- and respiratory-specific SOFA scores were independently associated with in-hospital mortality after adjustment for cerebral injury [5]. Pulmonary complications after CPR may be caused by the redistribution of pulmonary circulation associated with the aggravation of immunological reaction, and those lead to pulmonary edema, decreased respiratory system compliance, impaired gas exchange, and increased extravascular lung water (EVLW). Positive pressure ventilation (PPV) may yield positive effects in patients with pulmonary edema. The application of PEEP can increase alveolar fluid clearance and decrease EVLW. Pneumonia within the first 4 days after intensive care unit (ICU) admission is another serious complication after ROSC with the risk of aspiration and emergency airway access during cardiac arrest, and it may be difficult to diagnosed due to confounding factors, that is, the signs of systemic inflammatory response syndrome after ROSC that mimic sepsis and the introduction of therapeutic hypothermia that impairs the leukocyte function, and it may increase the risk of pneumonia and mask the fever response [6]. The rate of early-onset pneumonia is associated with the longer need of mechanical ventilation and the length of ICU stay [7]. We summarized the pulmonary complications and associated pathophysiology in patients with cardiac arrest as shown in Table 1.

Prehospital management: ventilation and oxygenation

Oxygenation during CPR

At the onset of cardiac arrest, an abrupt decrease in oxygen delivery caused subsequent decline of tissue partial pressure oxygen. Oxygen demand increases, whereas the oxygen supply decreases, being