Med Evac Up-2-Date

Resuscitation prior to intubation

May 2019

The objective of this article is to identify patients predisposed to cardiac arrest during emergency endotracheal intubation, consider the physiological factors that put patients at risk for cardiac arrest, and perform preventative measures that must be performed to secure a safe, successful, definitive airway.

HOP Killers
Hemodynamic instability/Induction agents/Paralytics
Oxygenation
pH

Hemodynamic Instability

Pre-intubation hypotension is the largest predictor of cardiac arrest during intubation. (1) Patients in shock experience a sympathetic catecholamine surge to maintain adequate perfusion.

The administration of induction agents used for rapid sequence intubation suppress the sympathetic response to shock. Additionally, taking away the patient's ability to spontaneously breathe and providing positive pressure ventilations will reduce venous return to the heart, resulting in decreased cardiac output and further hypotension. Take measures to support the patient's cardiac output and blood pressure prior to performing RSI, and further anticipate the steps necessary to facilitate a successful intubation. Empirically correct hypovolemia with intravenous crystalloid fluids/blood and FFP prior to RSI. In septic shock unresponsive to IVF, resuscitate with vasopressors prior to induction (2,3). Not only do vasopressors support the patient's blood pressure and mean arterial pressure, vasopressors will augment the delivery of induction agents and paralytics. Bare in mind that hypotension is the largest predictor of cardiac arrest peri-intubation (1). Prepare push dose Epinephrine (10mcg/ml) for rapid administration for immediate blood pressure support (2,3)

Induction Agents

Theoretically, in the hemodynamically unstable patient the pharmacokinetics of sedative agents are altered and can be administered at a significantly lower dose than required for a normotensive patient. (2,3) Administration of standard RSI doses can cause further unwanted hypotension with some agents, such as propofol (4). Depending on the patient's catecholamine reserve, ketamine may aid in supporting cardiac output by increasing HR, BP, and MAP, however studies have also observed hypotension in ketamine-treated patients with high shock index >0.9 (5).

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The rapid onset of action and potent analgesia of ketamine are also desirable characteristics of the drug, whereas other induction agents, such as etomidate, do not possess any analgesic properties and will require administration of an additional analgesic agent. All induction agents and sedatives may cause hypotension, so consider a patient's predisposition to hypotension prior to selecting an agent.

Paralytics

In the hemodynamically unstable patient, perfusion to the peripheral vascular system is decreased in a compensatory effort to perfuse vital organs. A reduced blood volume in the peripheral vascular system results in decreased perfusion to the peripheral musculature, where paralytic medications act at the neuromuscular junction. Theoretically, as a result, the time to achieve full paralysis in the hemodynamically unstable patient will be prolonged. Paralytic medications are cardiac output dependent.

Supporting the patient's blood pressure and increasing cardiac output prior to administration of paralytics is the most effective measure to improve the delivery of medications to the peripheral musculature.

Oxygenation

Inadequate oxygenation leads to rapid hemoglobin desaturation during a period of apnea. The oxyhemoglobin disassociation curve and critical hemoglobin desaturation demonstrates a rapid decline in saturation levels over time. Desaturation to below 70% puts patients at risk for dysrhythmia, hemodynamic decompensation, hypoxic brain injury, and death. (6) The time of desaturation is exacerbated in a hemodynamically unstable patient, which makes adequate pre-oxygenation a priority in order to extend the period of safe apnea.

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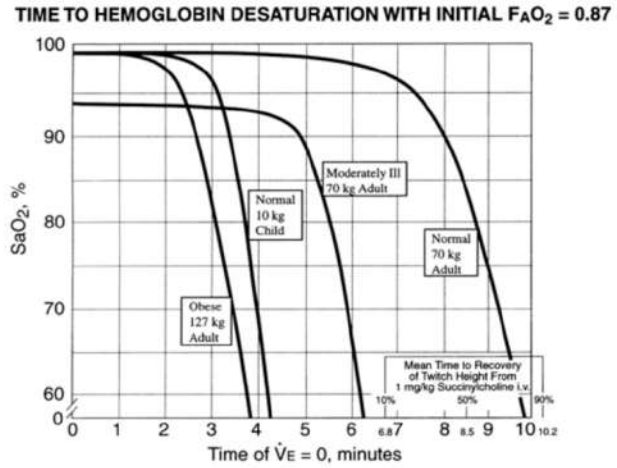

Oxyhemoglobin -Jonathan L. Benumof, Rachel Dagg, Reuben Benumof; Critical Hemoglobin Desaturation Will Occur before Return to an Unparalyzed State following 1 mg/kg Intravenous Succinylcholine . Anesthesiology 1997;87(4):979-982.

Physiologically, a patient in shock is experiencing increased sympathetic tone. In the spontaneously breathing patient, negative intrathoracic pressure created by spontaneous respiration supports venous return to the heart, thus increasing cardiac output.

By performing a rapid sequence intubation, we are inhibiting the bodies positive physiologic response to shock by depressing the sympathetic nervous system and providing positive pressure ventilation. Both of these necessary steps decrease cardiac output. It is imperative to obtain adequate pre-oxygenation by attempting to raise pulse oximetry readings as close to 100% as possible. Methods of providing 100% FiO2 can be referenced here (7/8):

Scott Weingart. Podcast 173 – LaMW – Oxygenation Kills Part I. EMCrit Blog. Published on May 2, 2016. Accessed on April 30th 2019. Available at [https://emcrit.org/emcrit/lamw-oxygenation-kills/]

pH (metabolic acidosis as physiologic cause of peri-intubation morbidity and mortality)

Experimental data clearly demonstrates that metabolic acidosis, including lactic acidosis, contributes to a reduction in cardiac contractility and leads to a hypo-vascular response to vasopressors through various mechanisms.

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Metabolic acidosis can ultimately lead to increased morbidity and mortality. In metabolic acidosis, the body will attempt to raise the pH of arterial blood by offloading CO2 through hyperventilation. While performing rapid sequence intubation, we are inhibiting the body's ability to compensate through respiration. Without proper apneic oxygenation at appropriate rates to support respiratory compensation, a rise in PaCO2 can have deleterious effects. Utilizing EtCO2 to observe the patient's exhaled CO2 levels can provide guidance for ventilatory rates during the intubation. Increased rates of assisted and mechanical ventilation may be required to maintain ETCO2 levels, in theory, supporting compensatory factors to maintain pH. After securing the patient's airway, adjust the rate of mechanical ventilation to maintain ETCO2 levels while obtaining an arterial blood gas to guide ventilator settings. (10)

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