

# Airway Management in Cardiac Arrest

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- Cardiac arrest • Airway management
- Cardiopulmonary resuscitation • Assisted ventilation
- Intubation • Ventilator

The mantra of airway, breathing, circulation (ABC) is well known to practitioners of emergency medicine. It positions airway management as a preeminent feature of any resuscitation and engenders memories of Dr Peter Safar and his groundbreaking work on artificial respiration. Recent discoveries surrounding the physiology of cardiac arrest have turned the ABC mnemonic around. Although effective exchange of oxygen is important to survival, it seems that maintenance of coronary and cerebral perfusion eclipses airway management as an overriding goal of any cardiac arrest situation. The last iteration of the American Heart Association (AHA) guidelines deemphasizes endotracheal intubation as the penultimate goal of airway intervention in cardiac arrest. Instead, emergency practitioners should focus on the rapid delivery of uninterrupted compressions and other basic life support interventions that have proved to increase both survival and neurologic recovery. Important questions about airway management persist. It is no longer a question of when a patient in cardiac arrest must be intubated. The decision to perform endotracheal intubation results from a complex synthesis of environmental and clinical factors. It may not be feasible for a system that lacks veteran paramedics to mandate securing the airway with an endotracheal tube. Recent advances in the understanding of cardiac arrest physiology suggest that airway management, in the immediate phase following cardiac arrest, is subordinate to interventions such as defibrillation and quality cardiac compressions. When intubation is performed, it should not interfere with the ongoing resuscitation and thereby

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decrease the coronary perfusion that is so vital to survival. This article reviews the state of the art as it applies to airway management in the patient with cardiac arrest. Like any other medical intervention, the plan for airway management should proceed from a thorough understanding of current evidence, provider capability, and emergency medical services (EMS) system resources.

## PARADIGM SHIFTS IN AIRWAY MANAGEMENT

### *Physiology of Cardiac Arrest Ventilation*

Ventilation is the exchange of gas that occurs as a result of air movement caused by changes in pressure. During spontaneous respirations, contractions of respiratory muscles cause an expansion of the chest cavity. A negative intrathoracic pressure (ITP) is generated, resulting in air moving into the lungs.<sup>1</sup> This negative-pressure ventilation is replaced with positive pressures when medical personnel attempt resuscitation of a patient during cardiac arrest. Although positive-pressure ventilation (PPV) has been an essential component of resuscitating critically ill patients, understanding the physiology of PPV and how it relates to patients in cardiac arrest is crucial to optimizing conditions favoring successful resuscitations.

The physiologic effect of PPV reducing coronary perfusion has long been studied.<sup>2</sup> PPV forces air into the respiratory system, increasing ITP. This increased ITP impedes blood flow returning to the heart and causes a marked decrease in left and right ventricular end-diastolic volumes, thereby reducing preload.<sup>3</sup> The Starling law reveals that this reduction in preload results in a direct decrease in cardiac output during chest compressions, thus reducing coronary perfusion. Therefore, PPV has the paradoxical and potentially harmful effect of reducing cardiac output during the resuscitation of a patient during cardiac arrest.

In addition to reducing coronary perfusion, PPV reduces the effectiveness of chest compressions by preventing negative ITP from being generated during recoil of the chest wall.<sup>4</sup> This negative ITP during the recoil phase of chest compressions assists with blood return to the chest cavity.<sup>5</sup> Drawing blood back to the heart increases preload, which in turn increases cardiac output with the next chest compression. However, while performing PPV, the generation of a negative ITP during chest wall recoil is hindered. PPV can consequently make chest compressions less effective at generating sufficient cardiac output.

Furthermore, PPV causes an increase in intracranial pressure.<sup>6</sup> The excess intrathoracic pressure generated during PPV is transmitted to the intracranial space via the venous vasculature. This process can result in a reduction in cerebral blood flow. Even if the patient were to be successfully resuscitated, the potential for anoxic brain injury or other profound complications from reduced cerebral blood flow is a possible drawback of PPV. The reductions in coronary and cerebral perfusion during PPV have profound implications for the management of patients in cardiac arrest.

### *De-emphasizing PPV*

The deleterious effects of PPV mandate that it must be avoided, or at least reduced. Despite AHA guidelines addressing concerns for hyperventilation, providers often ventilate at rates exceeding 30 breaths per minute.<sup>7</sup> So even when providers are specifically trained not to overventilate, they often continue to do so. A de-emphasis of PPV during training of providers, along with constant instruction by EMS medical directors and other continuing medical educators must highlight the potential harmfulness of PPV. Given the negative effects associated with ventilation, as discussed earlier, it is imperative to reduce PPV during cardiac arrest. The ABC

mantra of resuscitation has been the standard teaching since at least the early 1960s,<sup>8</sup> so the reduction of PPV represents a major paradigm shift in the management of patients in cardiac arrest, for which the emphasis on early airway management including intubation has long been emphasized.

Shifting the focus of resuscitations away from ventilations may improve other aspects that have proved to be more beneficial, such as increasing the amount of time compressing the chest. The cardiocerebral resuscitation (CCR) algorithm reduces interruptions to compressions by specifically concentrating on continuous chest compressions.<sup>9</sup> CCR is composed of cycles of 200 continuous compressions, each followed by defibrillation, if indicated. No PPV is given until at least the third cycle, when intubation may be considered. Instead, rescuers use passive insufflations with nonrebreather masks. Kellum and colleagues<sup>10</sup> found that CCR greatly improved outcomes, with 48% of patients being discharged from hospital with good neurologic functions, versus 15% using standard cardiopulmonary resuscitation (CPR). CCR represents a major shift in the standard resuscitative techniques because it inverts ABC to CAB (circulation, airway, breathing).

The need to not interrupt chest compressions, at least during initial phases of resuscitation, cannot be overemphasized. Despite compression/ventilation ratios that underscore the need for more time compressing the chest, less than half the time spent resuscitating a patient by rescuers, particular EMS providers, includes performing chest compressions.<sup>11</sup> Chest compressions are vital to achieving needed coronary perfusion pressures (CPP). A study by Reynolds and colleagues<sup>12</sup> found that higher CPPs than previously thought were associated with return of spontaneous circulation (ROSC) after cardiac arrest. Interrupting compressions to ventilate results in lower CPP, thereby reducing ROSC. Continuous chest compression has been shown to improve neurologic outcome following cardiac arrest.<sup>13</sup> Given the importance of perfusion during resuscitation, good chest compressions while minimizing PPV should be emphasized. Resuscitation methods that reduce any interruptions to chest compressions are highly desirable.

The concept of uninterrupted compressions is further supported by data from compression-only resuscitation by bystanders. A large study in Japan convincingly found that compression-only resuscitation resulted in more favorable neurologic outcomes compared with traditional CPR in patients who were apneic, had a shockable rhythm, or whose resuscitations were begun within 4 minutes of cardiac arrest.<sup>14</sup> Moreover, chest compressions may also provide some ventilation in the form of passive chest recoil.<sup>15</sup> In this respect, chest compressions may be a form of ventilation management. As such, uninterrupted chest compressions should be stressed and prioritized rather than mechanical ventilation.

## **EMERGENCY MEDICINE AIRWAY MANAGEMENT: FROM PREHOSPITAL TO THE EMERGENCY DEPARTMENT**

Recent studies and the latest iteration of the AHA guidelines have challenged the idea of endotracheal intubation as the gold standard for airway control in the arrested patient.<sup>16</sup> Prehospital providers, although capable of performing endotracheal intubation, are implementing less invasive strategies such as passive ventilation and supraglottic airways. The direct impact of endotracheal intubation to patient survival is a matter of debate. The focus of cardiac arrest management has shifted toward the delivery of excellent basic life support, uninterrupted compressions, and prompt defibrillation. Prehospital providers, emergency clinicians, and other critical care providers must have a thorough appreciation of the evidence base surrounding airway

management of the patient in cardiac arrest. Definitive airway control in the form of endotracheal intubation should not be achieved at the expense of other interventions linked to increased survival and improved neurologic outcome.

### ***Passive Airway Management***

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PPV, although long considered a staple of CPR, is being reevaluated. A retrospective study by Bobrow and colleagues<sup>17</sup> examined patients with out-of-hospital cardiac arrest who underwent airway management via passive insufflation or bag-valve-mask (BVM) ventilation. Responding paramedics were permitted, at their discretion, to deliver oxygen via BVM or nonrebreather facemask. In the subset of patients with witnessed arrest, passive ventilation was associated with increased neurologically intact survival to discharge. Despite study limitations that included a retrospective design and a lack of control for postarrest care, the association between neurologic recovery and passive oxygenation deserves consideration. Oxygen delivery via face mask does not require additional skills or equipment. It avoids the complications of hyperventilation and may minimize gastric distention. Although 1 retrospective study does not constitute a robust evidence base, it nevertheless reminds rescuers that a single strategy of airway control has potential complications. However, the AHA guidelines do not recommend removal of ventilation from CPR performed by advanced cardiac life support providers.<sup>16</sup>

### ***Noninvasive Airway Management***

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BVM ventilation is a cornerstone of emergency airway management. BVM strategies are taught to all levels of EMS personnel, from basic to paramedic. AHA guidelines affirm that all health care providers should be familiar with BVM techniques.<sup>16</sup> Airway management of the patient in cardiac arrest requires considerable attention to detail. First, rescuers must ensure avoidance of hyperventilation. Overzealous inflation of the BVM is associated with negative patient outcomes and complications such as hypotension.<sup>18</sup> As previously discussed, hyperventilation increases intrathoracic pressure, diminishes venous return, and decreases coronary perfusion.<sup>7,19</sup> Current AHA guidelines recommend low-volume (600 mL) ventilations for patients in cardiac arrest. Rescuers should deliver 2 breaths during the brief pause that follows every 30 chest compressions. When an advanced airway is not deployed, rescuers should not synchronize ventilations with compressions. Other airway adjuncts, such as nasopharyngeal airway (NPA) and oropharyngeal airway (OPA), may assist with maintenance of a tight facemask seal. Use of the OPA and NPA may also decrease airway resistance through displacement of the tongue. Cricoid pressure is also addressed in the 2010 guidelines. Although long considered customary in nearly all airway management scenarios, the application of cricoid pressure is not grounded in evidence-based practice. Recent studies associate cricoid pressure with impaired glottic visualization, airway obstruction, and even esophageal perforation.<sup>20,21</sup> The potential for complications and the lack of proven patient benefit resulted in a recommendation to avoid the routine use of cricoid pressure.<sup>16</sup> Like any other skill, BVM ventilation requires ongoing training and skills maintenance. It may be technically difficult for one rescuer to achieve an adequate face mask seal and provide ventilation sufficient to achieve chest rise. AHA guidelines corroborate that the presence of 2 rescuers is key to the execution of excellent BVM technique.<sup>16</sup> Guiding principles of effective BVM ventilation include avoidance of hyperventilation, the delivery of low volumes, and the maintenance of an adequate mouth-to-mask seal.

### ***Supraglottic Airway Device: Selection and Use***

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Providers are faced with many choices with respect to available airway management devices. Although BVM ventilation is often sufficient for the first few minutes of CPR, there is a perceived need to secure the patient's airway. Endotracheal intubation, long considered the optimum means of airway management for patients in arrest, has been supplanted by simpler and more rapid techniques that may have less potential for complication. Maintaining proficiency in endotracheal intubation is a significant barrier for many prehospital providers, and the link between prehospital intubation and survival in out-of-hospital cardiac arrest is not well established. Supraglottic airway devices mitigate some of the concerns and difficulties surrounding endotracheal intubation. In general, less training is required to achieve a baseline level of proficiency. Studies with supraglottic devices, including laryngeal mask airways (LMA) and laryngeal tube airways, indicate that basic-level emergency medical technicians (EMT) can successfully use these devices. Furthermore, time to ventilation is reliably shorter when a supraglottic device is chosen as the initial method for airway management. In the first few minutes of resuscitation, the importance of minimizing interruptions in CPR cannot be overstated. Even when performed by experienced providers, endotracheal intubation may result in unacceptable pauses in chest compressions.<sup>22</sup> Wang and colleagues<sup>22</sup> reported an alarmingly high rate of intubation-associated pauses. The investigators found that "the median total duration (sum) of endotracheal intubation-associated CPR interruptions was 109.5 seconds per patient." The AHA guidelines therefore endorse the use of supraglottic airways as a "reasonable alternative to bag-valve-mask ventilation and endotracheal intubation" in the management of cardiac arrest.<sup>16</sup> A supraglottic airway may confer additional advantages in the more austere out-of-hospital setting. Placement of these devices does not generally require visualization of the glottic opening. Blind insertion methods, such as those used for LMA insertion, obviate neck extension and airway manipulation.

The 2010 guidelines review 2 common types of supraglottic airway: the laryngeal mask and the laryngeal tube.<sup>16</sup> Insufficient exists to recommend one device rather than another. Providers deciding to implement a supraglottic strategy for airway control should be mindful of device-specific considerations. First, insertion of these airways necessitates that the patient have sufficient mouth opening. Trismus, trauma, or supraglottic obstruction interferes with proper device placement. Supraglottic airways are less effective in the ventilation of patients with a fixed decrease in airway compliance. Severe underlying airway obstruction and high airway resistance found in conditions such as cystic fibrosis and severe chronic obstructive pulmonary disease (COPD) may impair ventilation.<sup>23</sup> Gravid patients have decreased lower esophageal sphincter tone and may be at increased risk for aspiration. These considerations should be weighed against the ease of insertion when using a supraglottic device for a patient in cardiac arrest. The relative contraindications for a laryngeal mask airway may not be relevant for failed endotracheal intubation. The supraglottic airways feature prominently in existing algorithms for difficult airway management.<sup>23,24</sup> LMA use should complement other method for airway control. Certain patients may not be ventilated sufficiently with a supraglottic device. Providers directly responsible for airway management should therefore receive training in BVM ventilation and other strategies for airway control such as endotracheal intubation or esophageal tracheal combitube insertion.

### ***Tracheal Intubation: Revisiting the Gold Standard (Polishing the Touchstone?)***

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Endotracheal intubation is the gold standard in advanced airway management. Benefits traditionally associated with endotracheal intubation include effective ventilation

and protection from aspiration. Despite its long-standing place in emergency airway management, there is a paucity of data that link endotracheal intubation to improved survival and neurologic recovery from cardiac arrest. Clinically significant patient outcomes are consistently matched with the delivery of excellent basic life support to include uninterrupted and effective compressions. The performance of endotracheal intubation requires a considerable amount of initial provider training in addition to ongoing skills maintenance. The challenge of visualizing a glottic opening and placing a tube through the cords has been shown to interrupt the delivery of potentially life-saving cardiac compressions. As previously noted, these pauses are detrimental to achieving needed CPP associated with successful ROSC. Current AHA guidelines caution against interruption of CPR, and providers are encouraged to synchronize their intubation efforts with ongoing resuscitation. Further complicating the performance of endotracheal intubation is its unacceptably high complication rate. Hypoxemia, oropharyngeal trauma, and misplacement can result from failed or prolonged intubation attempts. The decision to tracheally intubate a patient in cardiac arrest is therefore contingent on several factors including level of provider training and experience. Frequent experience or frequent retraining is recommended for health care professionals authorized and trained to perform endotracheal intubation.<sup>16</sup>

Endotracheal intubation is also seldom performed compared with other procedures undertaken by prehospital providers. In a large study involving more than 40 EMS agencies, more than 30% of patients intubated in the prehospital setting required more than 1 attempt.<sup>25</sup> Multiple attempts at endotracheal intubation can be associated with airway trauma, aspiration, hypoxemia, and other serious complications.<sup>26,27</sup> Endotracheal intubation is a difficult procedure that requires a significant amount of time, and it is potentially associated with major complications, so it should be deemphasized in the management of patients in cardiac arrest.

The timing of endotracheal intubation may be critically important to the question of survival. In the first few moments of cardiac arrest, patient survival is most clearly linked to the preservation of coronary perfusion and minimally interrupted CPR.<sup>10</sup> Endotracheal intubation cannot therefore supplant the delivery of excellent and effective basic life support. AHA guidelines do not articulate or recommend a specific time interval for endotracheal intubation. No studies exist to directly address the relationship of advanced airway timing to improved survival. Immediate tracheal intubation is deemphasized.<sup>16</sup> In some cases, ventilation with a BVM or supraglottic airway may function as definitive airway control. Certain clinical situations may mandate endotracheal intubation. When protective airway reflexes are absent and frequent suctioning is required to maintain patency, the introduction of an endotracheal tube is key to ongoing airway management. Some patients cannot be ventilated with a BVM or supraglottic airway; these individuals require tracheal intubation for definitive control. For cardiac arrest, the AHA guidelines remind providers to limit intubation attempts to less than 10 seconds.<sup>16</sup>

Providers should be meticulous in the confirmation and subsequent anchoring of the endotracheal tube. Patient movement and transfer from ambulance stretcher to emergency department bed carry a risk of dislodgement.<sup>28</sup> Confirmation of placement is at once clinical and objective. Providers ideally should visualize the tube through the glottic opening, auscultate lung sounds, and use end-tidal carbon dioxide. A multimodal approach is desirable because any single strategy fails to detect the potentially lethal complication of tube misplacement or dislodgement: “Improper placement of endotracheal tubes into the esophagus...can remain undetected despite physical examination, chest radiography, and pulse oximetry methods.”<sup>28</sup> The setting of cardiac arrest is time dependent. The imperative to rapidly secure the airway, coupled with a patient

presenting in extremis, poses many challenges to both prehospital and hospital-based providers. An ideal strategy for tube confirmation should therefore require little time, a minimum amount of training, and exhibit high reliability. Despite several readily available methods and devices to assist providers with confirmation of placement, the single most reliable indicator of successful tracheal intubation remains detection of exhaled carbon dioxide.<sup>29,30</sup> Colorimetric detectors are simple devices that are easy to deploy. The devices answer the question of correct placement within the space of several PPVs. When managing the airway of a patient in full arrest, providers must pay meticulous attention to tube confirmation because the decreased exchange of carbon dioxide may lead to false-negative readings.<sup>28,29</sup> Although the presence of end-tidal carbon dioxide is specific for successful tube placement, no single confirmation modality approaches perfect sensitivity or 100% reliability. Because resuscitation is a dynamic event, it is prudent to continuously monitor carbon dioxide throughout treatment and transport. Waveform capnometry might not be readily available in all settings of emergency health care. However, waveform capnometry permits real-time assessment of tube position and permits early detection of tube dislodgement or ineffective air exchange.<sup>30</sup> As stated in the 2010 AHA guidelines, “providers should always use both clinical assessment and devices to confirm endotracheal tube location immediately after placement and throughout the resuscitation.”<sup>16</sup>

## VENTILATORY MANAGEMENT STRATEGIES

### *Avoidance of Hyperoxia*

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Successful resuscitation of the victim of cardiac arrest must rely on a team approach to airway management, as well as meaningful and thoughtful goals of oxygenation and ventilation. Much of the research has been focused on adequate perfusion techniques, showing the negative aspects of airway control regarding outcome. Oxygenation strategies must be considered during airway and ventilation management throughout resuscitation and ROSC. Most, if not all, attempts at reviving victims of cardiac arrest involve the use of oxygen delivery systems by health care providers. Therefore, oxygen should be considered a resuscitation drug and used as such. Most medical providers consider oxygen an important but benign aspect of emergency care. So how much oxygen is too much? Many experts think that oxygenation strategies are less important during resuscitation attempts, but are they? Is too little oxygen or too much oxygen deleterious to patient outcomes? Historically, oxygen has been a mainstay of emergency skills teaching since the early studies of CPR. The recommendation now is to assist ventilations during cardiac arrest with 100% forced inspiratory oxygen ( $\text{Fio}_2$ ), 5 times that of room air. Could this aggressive oxygenation with 100%  $\text{Fio}_2$  be causing further harm in an already compromised circulation? This goal of treating patients using hyperoxia should be carefully examined. There has been extensive research into examining the effects of high levels of oxygen during prolonged ventilation of patients in the intensive care unit. The theory is that such supranormal levels of oxygen are likely resulting in the production of oxygen free radicals (OFR). These OFRs are capable of interrupting cellular signaling pathways in addition to causing direct damage to the cells. In a recent multicenter cohort study, hyperoxic patients admitted to the intensive care unit (ICU) after being successfully resuscitated from cardiac arrest showed increased in-hospital mortality.<sup>31</sup> In this study, patients who were deemed to have arterial hyperoxia (defined as partial arterial oxygen tension [ $\text{PaO}_2$ ]  $\geq 300$  mm Hg) were associated with a higher mortality than hypoxic patients ( $\text{PaO}_2 < 60$  mm Hg).<sup>31</sup> In support of these observations, studies of cardiac arrest using hyperoxia have shown a worsened

oxidative stress and, hence, impaired neurologic outcomes.<sup>32–34</sup> In addition to OFR-mediated oxidative stress, studies suggest that hyperoxia may compromise coronary perfusion and cardiac output through coronary and peripheral vasoconstriction, thus leading to worsened ischemia-reperfusion injury as well as fueling the inflammatory response.<sup>35–37</sup> Based on these results, the idea of oxygenating and ventilating cardiac arrest patients with 100% F<sub>O</sub>₂ should perhaps be revisited. However, this issue is complex. Many experts argue that there is a major difference between early resuscitation and late resuscitation and/or ROSC. So is it necessary to titrate oxygen therapy throughout the phases of resuscitation? A study of experimental cardiopulmonary arrest suggested salutary effects of titrating oxygen delivery to arterial oxygen saturation in the early postresuscitation period.<sup>32</sup> In addition to oxygenation issues, ventilation after ROSC involves a careful and thoughtful plan to optimize patient outcomes.

### ***Ventilation During Induced Hypothermia***

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In the past decade, there has been a renewed interest in hypothermic treatment of patients suffering from traumatic brain injury, stroke, and cardiac arrest. However, there has been a paucity of evidence to support the use of routine hypothermia in patients suffering from head injury or stroke. In contrast, there has been a significant amount of data to suggest that routine use of induced hypothermia in patients who have been resuscitated from cardiac arrest may improve neurologic outcome. The protective effects of induced hypothermia are thought to be secondary to decreased cerebral carbon dioxide production, decreased oxygen consumption, immunomodulation, and overall diminished cerebral edema and epileptic foci.<sup>38–40</sup> Despite a renewed interest in hypothermic techniques for cardiac arrest resuscitation and recovery, little has been studied with respect to ventilator therapy and lung mechanics. Clinical scientists have had to rely on ventilator outcome data from similar populations such as critically ill septic patients, patients having cardiac bypass, and patients with neurotrauma. The literature is lacking with respect to changes in lung physiology and ventilator management when caring for the patient in the ICU after arrest.

During the initial moments after ROSC, many tasks must be completed with respect to airway management. A goal-directed strategy must be implemented to ensure optimal oxygenation and ventilation. If a supraglottic airway device has been used in the field, it must be replaced by an endotracheal tube so that mechanical ventilation can be commenced. The patient will likely be cooled in the first minutes to hours after ROSC. To achieve the goals of induced hypothermia and improved neurologic outcome, the patient should be sedated if the neurologic and hemodynamic states warrant this.

Before the induction of hypothermia, a baseline panel of respiratory blood gases should be examined. Evidence shows that the hypothermic state slows metabolism and therefore likely alters normal gas exchange and cellular use. Hence, goals for mechanical ventilation include optimizing blood gas levels, preventing hemodynamic instability, and resting the patient. It can be assumed that all patients that are resuscitated and in a reperfusion state are at risk for hemodynamic instability, acute lung injury, acute respiratory distress syndrome (ARDS), and other lung-related disorders such as pneumonia, atelectasis, and pulmonary edema. Patients after arrest are at particularly high risk for acute lung injury and ARDS, especially those transported from the field. Many of these patients have associated lung comorbidities such as COPD and history of smoking in addition to their coronary disease. Furthermore, a large percentage of patients managed in the field have aspirated various amounts of gastric contents before the placement of a definitive secured airway. In addition

to these pulmonary embarrassments, all resuscitated patients have the secondary insult of ischemia-reperfusion injury to tissue beds including the lung. As a result, attention must be focused on preventing further lung injury in these cooled, mechanically ventilated patients.

There are data suggesting that mild hypothermia may alter lung mechanics as well as respiratory gas production and use. Hypothermic reduction in carbon dioxide production may be masked by simultaneous alterations in lung compliance, resistance, and gas exchange.<sup>41,42</sup> To test this notion of altered lung mechanics during induced hypothermia, Aslami and colleagues<sup>43</sup> studied the effects of mild induced hypothermia on lung parameters in patients admitted to the ICU after resuscitation from cardiac arrest. Parameters included tidal volume, positive end-expiratory pressure (PEEP), plateau pressure, respiratory rate, end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>), and Fio<sub>2</sub>. In addition, static compliance and dead-space ventilation were recorded. The study cohort was mechanically ventilated in a pressure-controlled mode with an inspiratory/expiratory time of 1:2. Paco<sub>2</sub> was decreased during the hypothermic period with unchanged minute ventilation, whereas Pao<sub>2</sub>/Fio<sub>2</sub> ratio increased without altering PEEP levels. During rewarming, Paco<sub>2</sub> was unchanged; however, ETCO<sub>2</sub> increased with the same minute ventilation. Dead-space ventilation was unchanged and decreased during hypothermia and rewarming, respectively. Conversely, respiratory static compliance was unaltered throughout both hypothermic and rewarming phases. Based on the results of these studies, it is likely that hypothermia decreases carbon dioxide production in the mechanically ventilated postarrest population. Furthermore, lower tidal volume, as used in patients with acute lung injury (ALI), may prove to be beneficial in this patient population by resting the ischemia-reperfused lung tissue in hypocapnia. Results suggest that tidal volumes as low as 4 mL/kg may be beneficial in these hypocapneic, hypothermic patients to prevent further lung injury. Although lung compliance has been shown to be altered in most critically ill ventilator-dependent patients, this may not be the case during temporary induction of hypothermia in postarrest ICU management. In addition to the potential of unaltered lung compliance in most patients, the suspected decrease in oxygen demand and use during induced hypothermia may allow reduction in Fio<sub>2</sub> as well as PEEP levels throughout the postarrest recovery phase. Based on the limited literature and anecdotal experience, ventilator management in the postarrest hypothermic patient should be targeted to resting the lungs, which can be accomplished through either a volume or pressure mode of ventilation, targeting smaller tidal volumes and plateau pressures. In addition, it may be prudent to use minimal to moderate PEEP in this patient population depending on the individual's oxygenation status. All patients undergoing a hypothermic postarrest protocol should be mechanically ventilated, using sedation and muscle relaxation. Muscle relaxants allow for controlled respirations, maintaining a narrow window of targeted blood gas values, in addition to preventing the shivering response, which could potentially alter metabolic goals.

### ***Optimal Ventilator Support***

Ventilatory management in patients with ROSC is critical to ensure metabolic recovery. During the initial stages of ROSC, there is considerable damage to the internal milieu of tissue beds and cells. Reperfusion injury results in catastrophic alterations in cellular signalling cascades and metabolic machinery. OFRs, inflammatory cytokines, and acids are an integral part of this process and clinical scientists must do whatever is necessary to prevent further cellular injury. Goals are therefore directed at maintaining the organism's physiology in the most homeostatic state possible, to allow recovery of viable tissues and cells, although this is easier said than done.

Studies have shown that optimal ventilatory support in critically ill patients should target strategies that minimize ALI and the potential for ARDS, as stated earlier. Traditional approaches used tidal volumes of 10 to 12 mL/kg and excessively high PEEPs. To minimize risks of barotrauma and volutrauma (risks for ALI and ARDS), tidal volume ( $V_t$ ) must be set to lower levels (6–8 mL/kg) and best PEEP must be targeted, which is defined as the lowest PEEP possible to ensure optimal oxygenation and hemodynamic stability. High levels of PEEP result in several undesirable effects. High intrinsic PEEP or auto-PEEP leads to increased intrathoracic pressures, interrupting venous return, diminishing cardiac output, and ultimately compromising both coronary and global perfusion. In addition, high intrathoracic pressures may cause an increase in intracranial pressure (ICP), which can be deleterious in an already compromised recovering cerebrum.

Once the patient is stabilized, mechanical ventilator settings optimized, and hypothermic targets reached, a chest radiograph should be ordered to determine correct endotracheal tube placement and whether any lung disorder is present. Blood gas measurements should be followed closely in the first few hours of mechanical ventilation to ensure optimal respiratory physiology. ALI is suggested if the  $\text{PaO}_2/\text{FiO}_2$  is less than or equal to 300 mm Hg. If  $\text{PaO}_2/\text{FiO}_2$  is less than 200, this indicates ARDS. Strategies to improve pulmonary derangements during hypothermic protocol include titrated  $\text{FiO}_2$  and PEEP levels based on dynamic lung physiology. Recommended guidelines suggest titrating the minimum  $\text{FiO}_2$  to maintain arterial oxygen saturations at greater than or equal to 94%, with the goal of ensuring adequate oxygen delivery while preventing hyperoxia.<sup>44</sup> Studies of animal models of ROSC have shown that ventilating with  $\text{FiO}_2$  of 100% in the first 15 to 60 minutes after ROSC worsens brain lipid peroxidation, metabolic dysfunction, neuron degeneration, and short-term functional outcome compared with ventilation with room air or titrating  $\text{FiO}_2$  to arterial saturations between 94% and 96%.<sup>33,34,45–48</sup>

Hyperventilation was once thought to be the best method to aid in reversing a combined respiratory and metabolic acidosis that ensued after cardiac arrest. Although theoretically beneficial, we have determined this practice to be detrimental. Hyperventilating the hypothermic patient after arrest can be problematic for several reasons. Increased minute ventilation (respiratory rate and  $V_t$ ) decreases  $\text{PCO}_2$ , thus shifting the oxyhemoglobin dissociation curve to the left, altering the delivery of oxygen to the tissues. In addition, lowered  $\text{PCO}_2$  alters cerebral circulation. Hypocapnia causes cerebral vasoconstriction, thus diminishing cerebral blood flow and oxygenation, exacerbating neurologic ischemic injury. Hence, it is recommended to use conventional ventilator modes, aiming for normocapnia, best PEEP, carefully titrated to lowest  $\text{FiO}_2$  possible to maintain arterial saturations greater than or equal to 94%, and a relaxed sedated patient. There is no compelling evidence to show that any special ventilation method should be used other than those described earlier. Clinicians must be vigilant and prevent hyperventilation, hypoventilation, and associated hypoxemia and hypercarbia, as well as ALI and/or ARDS. In addition to these lung-sparing guidelines, several monitoring techniques are equally important to help guide the clinician caring for the hypothermic patient after arrest.

Once the patient with ROSC has been stabilized in the ICU and is on mechanical ventilation, an arterial catheter should be placed to monitor hemodynamics as well as intermittent arterial blood gas measurements to help guide ventilator management. In addition, continuous arterial pulse oximetry should be used and targeted to 94% to 96%, which should correspond to a  $\text{PaO}_2$  of 80 to 100 mm Hg.  $\text{FiO}_2$  should be titrated down to reach these oxygenation goals. Continuous waveform capnography is now considered the gold standard for monitoring integrity of the intubated patient as

well as to measure  $\text{ETCO}_2$  and maintain a normocapneic state of approximately 35 to 40 mm Hg and  $\text{Paco}_2$  of 40–45 mm Hg. Again, chest radiographs should be used as needed to determine correct endotracheal tube positioning and to follow any developing lung disorders.

Ventilatory management of the patient being resuscitated from cardiac arrest is a delicate balance that must take into account the goals of needing to optimize blood gas levels while resting the patient, maintaining hemodynamic stability. Because such patients have lung mechanics that closely resemble ARDS, the lowest possible PEEP must be used. Hyperventilation must also be avoided. Furthermore, careful titration of  $\text{Fio}_2$  to achieve goal arterial saturations greater than or equal to 94% is recommended.

## SUMMARY

Airway management of the patient in cardiac arrest is of prime importance. Paradigm shifts in the understanding of cardiac arrest physiology have resulted in changes to overall management strategies. Historically, the tracheal intubation was considered central to any successful resuscitation. Tracheal intubation confers advantages compared with noninvasive ventilation, but it can no longer be recommended without reservation. Neurologic recovery and improved survival is closely associated with uninterrupted compressions, high-quality CPR, and basic life support interventions. As opposed to placement of a tube through the glottic opening, the initial goal of emergency airway management is the provision of effective ventilation. Chest rise, as produced with a BVM or supraglottic airway, may be sufficient in the first few minutes of resuscitation. AHA guidelines and current research support the idea that deployment of an advanced airway may be deferred until the completion of 2 compression cycles.<sup>10,16</sup> Although the optimum timing for endotracheal intubation remains unclear, there are no data to support the idea that it must be performed immediately. Providers should focus on minimizing interruptions in compressions and delivering high-quality basic life support. Emergency providers capable of performing endotracheal intubation must do so with the goal of integrating intubation attempts into the resuscitation as opposed to expecting a protracted pause. Endotracheal intubation has complications, and providers should be sufficiently trained and retrained to maintain proficiency. Ideally, advanced airway placement should be confirmed clinically and with the presence of end-tidal carbon dioxide. The addition of continuous waveform capnometry capability permits early recognition of unsuccessful tube placement or dislodgement. Further, the goals of ventilator support of the resuscitated patient include the need to use the lowest possible PEEP while carefully titrating  $\text{Fio}_2$  to avoid hyperoxia.

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