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REVIEW ARTICLE

Emergency Medical Services

A review of ventilation in adult out-of-hospital cardiac arrest

Matthew R. Neth MD[1] | Ahamed Idris MD[2] | Jason McMullan MD, MS[3] | Justin L. Benoit MD, MS[3] | Mohamud R. Daya MD, MS[1]

[1]Department of Emergency Medicine, Oregon Health and Science University, Portland, Oregon

[2]Department of Emergency Medicine, UT Southwestern, Dallas, Texas

[3]Department of Emergency Medicine, University of Cincinnati College of Medicine, Cincinnati, Ohio

Correspondence
Matthew R. Neth, MD, Department of Emergency Medicine, Oregon Health & Science University, Mail Code: CDW-EM, 3181 SW Sam Jackson Park Road, Portland, OR 97239, USA.
Email: neth@ohsu.edu

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Abstract

Out-of-hospital cardiac arrest continues to be a devastating condition despite advances in resuscitation care. Ensuring effective gas exchange must be weighed against the negative impact hyperventilation can have on cardiac physiology and survival. The goals of this narrative review are to evaluate the available evidence regarding the role of ventilation in out-of-hospital cardiac arrest resuscitation and to provide recommendations for future directions. Ensuring successful airway patency is fundamental for effective ventilation. The airway management approach should be based on professional skill level and the situation faced by rescuers. Evidence has explored the influence of different ventilation rates, tidal volumes, and strategies during out-of-hospital cardiac arrest; however, other modifiable factors affecting out-of-hospital cardiac arrest ventilation have limited supporting data. Researchers have begun to explore the impact of ventilation in adult out-of-hospital cardiac arrest outcomes, further stressing its importance in cardiac arrest resuscitation management. Capnography and thoracic impedance signals are used to measure ventilation rate, although these strategies have limitations. Existing technology fails to reliably measure real-time clinical ventilation data, thereby limiting the ability to investigate optimal ventilation management. An essential step in advancing cardiac arrest care will be to develop techniques to accurately and reliably measure ventilation parameters. These devices should allow for immediate feedback for out-of-hospital practitioners, in a similar way to chest compression feedback. Once developed, new strategies can be established to guide out-of-hospital personnel on optimal ventilation practices.

KEYWORDS

cardiopulmonary resuscitation, emergency medical technician, minute ventilation, out-of-hospital, out-of-hospital cardiac arrest, paramedic, prehospital, respiration, respiratory rate, tidal volume, ventilation, ventilation rate

1 | INTRODUCTION

Approximately 360,000 adults suffer an emergency medical services (EMS)-treated out-of-hospital cardiac arrest in the United States each year.[1] Cardiac arrest continues to be a devastating condition for most individuals with a current overall survival rate of around 10%.[2] Outcomes have improved since the introduction of the cardiac arrest chain of survival in the early 1990s; however, there are still many

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areas of resuscitation science to be explored, which could potentially improve patient outcomes.[3,4] One of the most important links in the chain is early high-quality cardiopulmonary resuscitation (CPR), with an emphasis on chest compressions. However, guidelines provide rescuers with minimal direction on optimal ventilation management during resuscitation.

Ventilation may play a crucial role in cardiac arrest resuscitation by allowing for adequate oxygen administration and meaningful carbon dioxide elimination, thereby improving cellular metabolism. Modifiable factors affecting out-of-hospital ventilation include rate, tidal volume, ventilation delivery time, airway pressure, and timing with chest compressions. The goals of this narrative review are to evaluate the available evidence regarding the role of ventilation in out-of-hospital cardiac arrest resuscitation and to provide recommendations for future directions.

1.1 | Current guidelines

Although effective ventilation management may be an essential aspect for all types of out-of-hospital cardiac arrest, research provides limited insight into the optimal ventilation parameters. Despite these limitations, recommendations are provided for ventilation rate, ventilation delivery time, tidal volume, and timing with chest compressions.[5-8] Guidelines do not address other ventilation components (eg, airway pressures). Furthermore, these recommendations are uniform for all types of out-of-hospital cardiac arrest, although different etiologies may require unique ventilation strategies.

The American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care supports the use of bag-valve-mask ventilation and advanced airways (ie, supraglottic airway or endotracheal intubation) to assist with oxygenation and ventilation during out-of-hospital cardiac arrest.[5,8] Airway management strategy is typically based on professional skill level and the situation faced by rescuers.[5,8] Before placement of an advanced airway, interrupted ventilation cycles of 2 breaths every 30 compressions or asynchronous ventilation with continuous compressions at a rate of 10 breaths/min (ie, 1 breath every 6 seconds) are the currently recommended ventilation strategies. After advanced airway placement, guidelines recommend asynchronous ventilation at a rate of 10 breaths/min with continuous compressions.[5-8]

Guidelines recommend a tidal volume around 600 mL for adults, or enough to produce visible chest rise, with each ventilation delivered over 1 second. Supplemental oxygen use is recommended if available and should be supplied at a minimum flow rate of 10–12 L/min or an oxygen concentration of >40%.[5,7]

2 | HOW DOES VENTILATION AFFECT THE PHYSIOLOGY OF CARDIAC ARREST?

Several mechanisms to explain blood flow during cardiac arrest have been described.[9] A widely accepted theory involves the thorax acting as a pump in a dynamic circuit of oscillating intrathoracic pressures.[9-12] On chest compression release, the decline in intrathoracic pressure causes blood to flow back into the heart and pulmonary circulation. Blood flows from the pulmonary circulation to the left side of the heart and out to the systemic circulation during the active chest compression downforce due to an increase in intrathoracic pressure.[13] Contrary to the chest pump (ie, thoracic pump) theory described above, the cardiac pump theory hypothesizes that direct compression of the ventricles generates forward blood flow.[9,11,12]

When a positive pressure breath is delivered, this leads to a decrease in venous return and subsequent decline in cardiac output.[14-16] Therefore, ventilation may play a pivotal role by affecting the intrathoracic and intravascular pressures.[17,18]

2.1 | Importance of ventilation

Satisfactory levels of oxygen are available during the first few minutes of cardiac arrest before the initiation of chest compressions, particularly in witnessed ventricular fibrillation/tachycardia arrests (ie, non-asphyxia precipitated arrests).[19-22] However, as the resuscitation progresses, ventilation becomes more important as oxygen is depleted and carbon dioxide (CO_2) levels begin to rise.[23-25] Studies have shown that without sufficient ventilation, hypoxemia, hypercapnia, and acidemia can result.[25-30] Managing ventilation in low blood flow states like out-of-hospital cardiac arrest becomes a delicate balance between ensuring adequate oxygenation and removal of carbon dioxide while avoiding the potential adverse hemodynamic effects of ventilation (eg, diminished coronary perfusion and venous return) and avoiding prolonged interruptions of effective chest compressions.[22,31-33]

Ventilation is essential for CO_2 removal. In low blood flow states, less CO_2 is delivered to the lungs for elimination, which results in the accumulation of CO_2 in the body.[25] Rising CO_2 levels can lead to a decrease in serum pH and a decreased affinity between oxygen and hemoglobin. The resultant hypercarbic, hypoxemic, and acidotic state leads to ineffective cardiac contractility, reduced systemic vascular resistance, and an increase in pulmonary vascular resistance.[34-38] This suboptimal metabolic state decreases the threshold for ventricular dysrhythmia development and is associated with ineffective cardiac defibrillation.[25,39-42]

2.2 | Impact of ventilation on pulmonary and systemic hemodynamics

Ventilation impacts the hemodynamics of both the pulmonary and systemic systems.[25] Poorly aerated lungs lead to increased pulmonary vascular resistance and a subsequent decline in pulmonary blood flow.[43,44] Decreases in alveolar oxygen levels contribute to pulmonary artery vasoconstriction as the body attempts to shunt blood to better-oxygenated areas of the lungs.[43,44] This increased resistance to pulmonary blood flow may inhibit blood flow from the right heart to the left heart, decreasing the effectiveness of chest compressions.

Hyperventilation can have negative impacts on cardiac physiology and survival.[22,31,32] The increased intrathoracic pressure caused by excessive positive pressure ventilation leads to decreased venous return and subsequent decreased cardiac output.[14-16] Downstream effects include a reduction in coronary and cerebral perfusion pressures, which are essential components of improved out-of-hospital cardiac arrest outcomes.[45]

Factors leading to increased intrathoracic pressure include excessive ventilation rate, increased tidal volume, and decreased expiration time (ie, auto-PEEP). Although excessive positive pressure ventilation leads to decreased venous return and cardiac output, managing out-of-hospital cardiac arrest without ventilation can lead to increased pulmonary vascular collapse, atelectasis, and shunting.[25,43,44]

2.3 | Effect of ventilation on the development of atelectasis during CPR

During cardiac arrest resuscitation, atelectasis likely occurs due to direct lung injury and pulmonary congestion caused by the persistent chest compression force and is worsened by ineffective ventilation.[46-48] A porcine model showed that animals receiving no ventilation had about twice the amount of alveolar atelectasis formation than those receiving positive pressure ventilation.[47] The resultant loss of alveolar volume contributes to ineffective gas exchange leading to hypoxemia, hypercapnia, and acidosis and ultimately worse outcomes.[48,49]

2.4 | Ventilation provided by chest compressions and intrathoracic airway collapse

The lungs collapse during chest compressions and subsequently expand due to the negative intrathoracic pressure generated by chest wall recoil upon compression release. This act of passive ventilation creates pressure changes within the thorax, resulting in the movement of gas within the airway. However, the tidal volumes generated by compression-only CPR are insufficient to support sufficient gas exchange.[27,29,50-52] Chest compressions without positive pressure ventilation produce average tidal volumes of 156 mL in intubated and paralyzed adult patients.[52] In paralyzed adult patients without a patent airway, tidal volumes are considerably lower and may approach zero or even be negative.[52,53] Adult out-of-hospital cardiac arrest patients receiving compression-only CPR with the use of a mechanical compression device showed median tidal volumes of 41.5 mL.[50] A recent study evaluating guideline-compliant compression depth (median of 2.2 in.) in adult out-of-hospital cardiac arrest patients observed a median tidal volume of 7.5 mL during compressions, with the highest observed value being 45.8 mL.[51] The overwhelming majority of recorded tidal volumes were <20 mL.[51]

Intrathoracic airway collapse (occurring when the end-expiratory lung volume falls below airway closing capacity), the loss of intercostal muscle tone, and the downforce generated by chest compressions contribute to these insignificant volumes.[54] Although a small amount of positive end-expiratory pressure (PEEP) may counteract this phenomenon and generate tidal volume during chest compressions, the optimal amount of PEEP needed to offset airway collapse while not causing detrimental hemodynamic effects is unknown.[52-55]

3 | HOW IS VENTILATION CURRENTLY MEASURED IN THE OUT-OF-HOSPITAL SETTING?

Due to limitations with current technology, ventilation is difficult to measure in the out-of-hospital setting. The most commonly measured ventilation characteristic is the rate, which can be assessed in real-time by an observer or in monitor software analyzed after a resuscitation attempt. However, other ventilation parameters are difficult to measure in the out-of-hospital setting. Common strategies for assessing ventilation in out-of-hospital cardiac arrest resuscitation attempts are addressed below.

3.1 | Capnography

Capnography plots the concentration of CO_2 throughout the respiratory cycle. End-tidal CO_2 ($EtCO_2$) refers to the concentration of CO_2 at the end of exhalation and is usually the value displayed on cardiac monitors. The displayed $EtCO_2$ value represents the highest measurement in a ventilation cycle, and under a normal physiologic state is between 35 and 45 mm Hg.[55] Capnography has many different applications in the management of out-of-hospital cardiac arrest resuscitation, including endotracheal tube confirmation, assessing for endotracheal tube dislodgement, ensuring chest compression quality, detection of return of spontaneous circulation, aiding in termination of resuscitation decisions, and optimizing ventilation.[55-61] Pulmonary blood flow is the primary determinant of $EtCO_2$ values in low blood flow states (ie, cardiac arrest).[58,61]

Capnographic fluctuations during out-of-hospital cardiac arrest may not allow for reliable guidance of ventilation during resuscitation attempts. Figure 1 illustrates chest compression waveform artifacts that can be seen during resuscitation and compares it with a capnogram without artifacts in Figure 2. $EtCO_2$ readings are also affected by changes in minute ventilation, chest compressions, cardiac arrest etiology (eg, unwitnessed arrest, pulmonary embolism, asphyxia precipitated arrest), cardiac arrest duration, and the administration of bolus medications (eg, sodium bicarbonate and epinephrine).[27,57,58,61-66]

Chest compression-generated tidal volumes (dependent on intrathoracic airway patency, patient physiology, and equipment error) can cause artifacts (ie, oscillations) in $EtCO_2$ waveforms, which may hinder accurate interpretation of CPR quality and ventilation.[67-71] One strategy to assist with the interpretation of $EtCO_2$ waveforms with oscillations is to use the maximum $EtCO_2$ recorded between uninterrupted ventilations, as this likely reflects accurate alveolar CO_2.[68] However, this strategy may be unreliable during a fluctuating

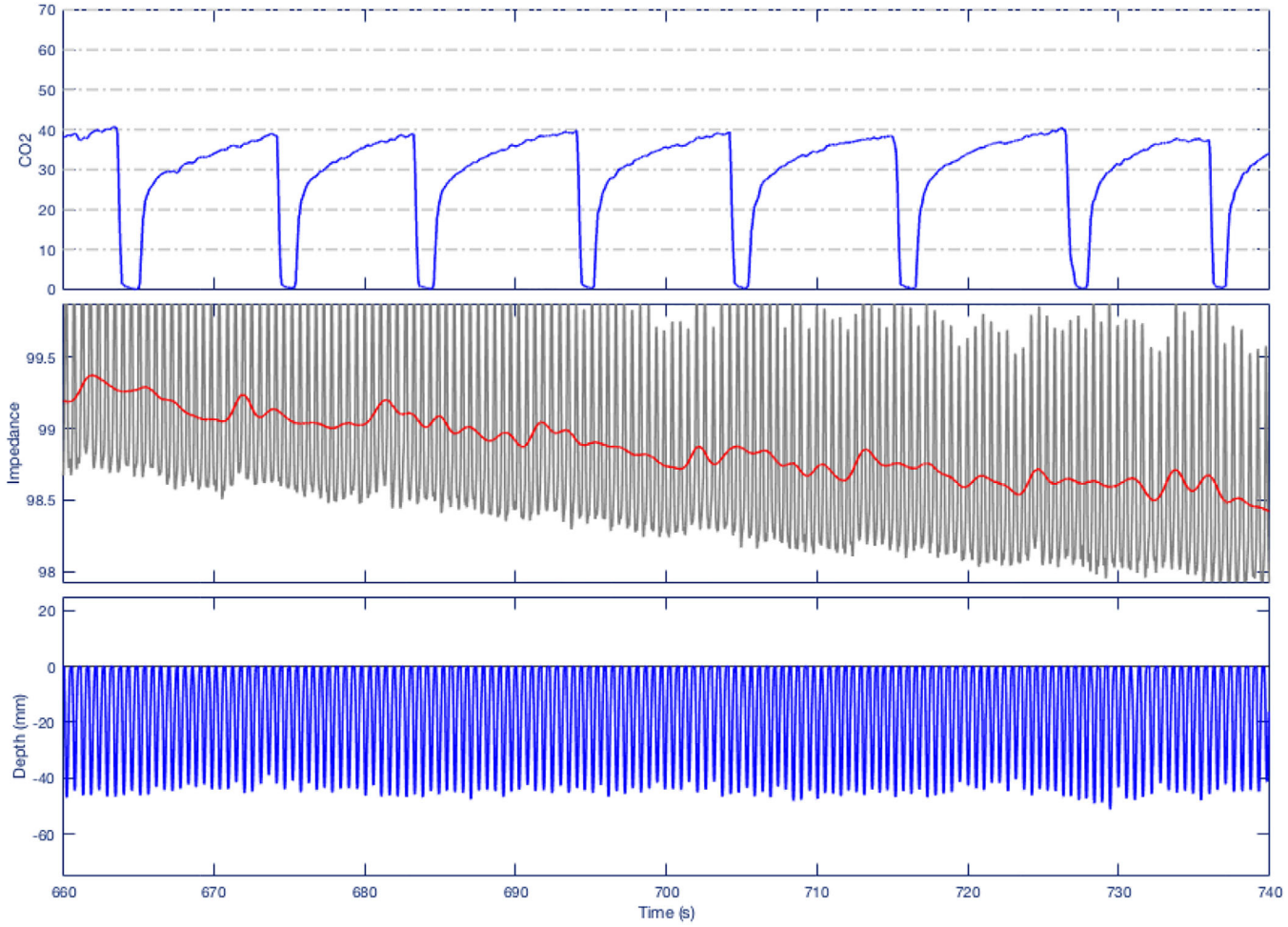

FIGURE 1 Capnogram with $EtCO_2$ waveform chest compression artifact

resuscitation. New strategies are being explored, which may allow for reliable real-time ventilation feedback (similar to compression feedback).[71,72] Algorithms are being developed to aid in the interpretation of waveforms and provide rescuers with more reliable ventilation rate feedback.[70-74]

3.2 | Thoracic impedance signal

Thoracic impedance signal has also been proposed as a method to measure and provide reliable ventilation feedback during CPR.[72,75-78] Thoracic impedance signals use data recorded from defibrillator pads to assess ventilation rate through changes induced by chest wall movement (ie, lung volume).[78,79] Similar to capnography, the thoracic impedance signal is often affected by chest compressions making ventilation rate interpretation challenging.[72,75,76,78] Techniques to calculate tidal volume data are being explored, although they do not appear accurate at this time.[76,77] Additionally, the majority of thoracic impedance signal data is assessed after the resuscitation, with an inability to provide real-time ventilation rate feedback to rescuers using existing technology.[71,72,75]

4 | HOW ARE VENTILATIONS CURRENTLY DELIVERED?

Ensuring effective airway patency is vital to successful ventilation management in out-of-hospital cardiac arrest. While many strategies for airway management exist, the majority of resuscitation attempts are managed initially with basic airway skills (ie, upper airway positioning and bag-valve-mask ventilation).[80] Patient anatomy, external factors (eg, blood, secretions, positioning), and poor technique contribute to the difficulties of performing effective bag-valve-mask ventilation. Previous studies have shown that a 2-person bag-valve-mask technique is more effective than 1-person bagging and is recommended assuming an adequate number of rescuers are available.[81,82] Additionally, because bag-valve-mask ventilation does not create a secure passage of air into the lung, gastric insufflation and aspiration can develop.[83,84]

After basic airway management, successful placement of an advanced airway (ie, supraglottic airway, endotracheal intubation) occurs ~80% of the time.[80] These devices allow a more direct pathway into the trachea and lungs, and in theory, should supply more effective ventilation and oxygenation. Supraglottic airway devices (eg, esophageal tracheal airway, laryngeal mask airway [LMA], King LT

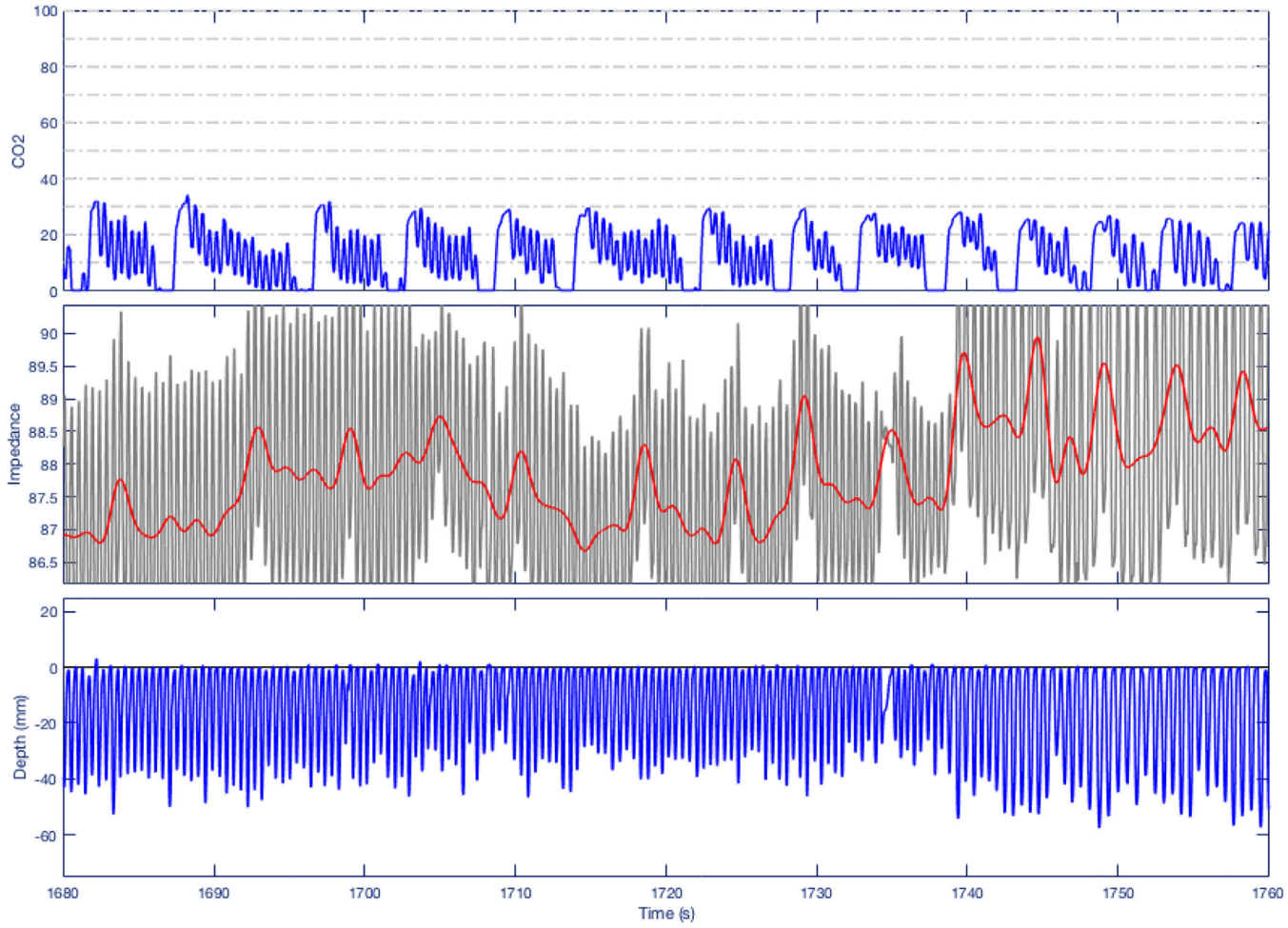

FIGURE 2 Capnogram without chest compression artifact

[Ambu Inc.], and i-gel [Intersurgical Ltd.]) are typically quicker to insert, require less training, and can be placed by less skilled personnel than an endotracheal tube.[85-87] The main limitations of advanced airway placement include a reduction in chest compression fraction (due to longer pause length for airway placement), the potential for a misplaced or dislodged airway (eg, esophageal intubation) leading to ineffective ventilation and oxygenation, and potential injury to surrounding soft tissue structures.[88-93]

Until recently, research comparing the placement of a supraglottic airway device versus endotracheal intubation in out-of-hospital cardiac arrest has been limited to observational studies that suggested endotracheal intubation may be a superior technique.[94-104] However, in 2018 two large randomized controlled trials compared out-of-hospital emergency medical technician and paramedic performance of the 2 strategies. The 3000 subject multicenter cluster-crossover randomized trial, Pragmatic Airway Resuscitation Trial (PART), compared the King LT to endotracheal intubation as the initial strategy for advanced airway management in adult out-of-hospital cardiac arrest.[87] Patients managed initially with the King LT showed higher survival at 72 hours compared to those initially managed with endotracheal intubation. Rates of return of spontaneous circulation, hospital survival, a favorable neurologic outcome at discharge, and initial airway attempt success were also higher in the King LT group. The main limitation of the trial is the pragmatic design, which only controlled for the initial advanced airway management strategy, with clinical teams determining all other resuscitation and post-resuscitation care.[87] Similar to PART, the 9289 patient multicenter cluster-randomized clinical trial, AIRWAYS-2, compared the i-gel to endotracheal intubation as the initial device for advanced airway management in adult out-of-hospital cardiac arrest patients.[105] No significant difference was found between the 2 groups for the primary outcome of the modified Rankin Scale score at hospital discharge or 30 days. However, in patients who ultimately received advanced airway management, the i-gel outperformed endotracheal intubation.[105] Additionally, initial ventilation was more likely to be successful in the i-gel group compared with the endotracheal intubation group, and there was no difference in adverse outcomes (ie, aspiration and vomiting) between the groups.[105]

A non-inferiority trial comparing endotracheal intubation to bag-valve-mask ventilation in a physician-driven EMS system was also published in 2018. This 2043-subject study failed to demonstrate non-inferiority or inferiority for survival with a favorable 28-day neurological function of either technique.[106] Prior studies evaluating

the use of a bag-valve-mask device during out-of-hospital cardiac arrest resuscitation demonstrated an association with improved survival and neurologic outcomes as compared to those managed with a supraglottic airway device or endotracheal intubation.[100,107-110] However, these observational studies have been limited by confounding by indication and resuscitation time bias, as supraglottic airways are often placed after other airway management techniques have failed. Additionally, endotracheal intubation may be a surrogate for a prolonged resuscitation.[111,112]

Decisions for airway management strategy and device selection in adult out-of-hospital cardiac arrest should be based on each unique situation and rescuer experience level.[8] EMS professionals must receive adequate training and continuous quality improvement (CQI) to ensure effective airway patency while also ensuring airway management is not interfering with the other aspects of CPR known to improve outcomes.[113,114]

4.1 | Mechanical ventilation

Limited evidence exists evaluating the role of mechanical ventilation in out-of-hospital cardiac arrest resuscitation. Pneumatically powered automatic transport ventilators may be considered for prolonged resuscitations.[115] These devices appear to provide effective gas exchange during CPR, and similar oxygenation and ventilation as compared to manual ventilation (ie, bag-valve-mask ventilation).[116-119] The automatic transport ventilators also offers the advantage of allowing EMS professionals to perform other tasks during the resuscitation, which may be particularly valuable in agencies with limited personnel.[119] EMS professionals need to understand the effect of different ventilation modes, tidal volumes, ventilation rates, inspiratory times, airway pressures, and PEEP have on the patient's physiology during CPR. Those using the automatic transport ventilators should be familiar with the operational advantages and challenges of their specific device.

Oxygen-powered resuscitators may offer a more practical approach to out-of-hospital mechanical ventilation due to their decreased cost, decreased size, and more simplified design. Oxygen-powered, flow-limited resuscitators can be used in either automatic or manual modes. The automatic mode delivers a set oxygen flow rate until a specified airway pressure is achieved, and redelivers oxygen when the device registers lack of flow (typically with every chest compression-decompression).[120] During manual mode, the rescuer manually administers an inspiration with a set oxygen flow rate by pressing a button on the device. The inhalation stops after the button releases or once a set pressure limit is reached. Select devices (eg, Oxylator) provide potentially valuable feedback on airway obstruction (by clicking) and during an ineffective mask seal, or supraglottic airway misplacement (by not delivering the next inspiration).

Per the current American Heart Association guidelines, the use of manually triggered, oxygen-powered, flow-limited resuscitators may be considered in patients without an advanced airway in place.[115] The use of the automatic mode is currently discouraged during CPR due to the potential for increased PEEP, which may lead to compromised hemodynamics.[115] Recent studies have challenged this theory, and have shown good outcomes with the use of the automatic mode.[120-122] Oxygen-powered resuscitators have shown adequate oxygenation and ventilation during CPR.[121-126] These devices may also add the advantage of leading to less gastric insufflation as compared to bag-valve-mask ventilation.[124,125]

The literature does not support any significant benefit of mechanical ventilation over manual ventilation. EMS professionals using these devices should have sufficient training and CQI processes in place to ensure appropriate application and recognition of potential complications.[127] Back-up manual ventilation devices (ie, bag-valve-mask) should be readily accessible in case of mechanical device failure or oxygen source depletion.[115] Device expense, initial EMS personnel training, CQI, high oxygen consumption, and need for a power source (in some automatic transport ventilator devices) limit the use of mechanical ventilation for widespread out-of-hospital CPR application.

5 | WHAT EVIDENCE IS AVAILABLE REGARDING VENTILATION RATES AND STRATEGY?

Many approaches for ventilation delivery in out-of-hospital cardiac arrest resuscitation have been assessed. Strategies include passive oxygenation with no positive pressure ventilation to various rates and methods of supplying positive pressure ventilation (eg, ventilation with interrupted compressions and asynchronous ventilation with continuous compressions).

Previous research suggests that passive oxygenation offers minimal effective alveolar ventilation and is likely not the optimal strategy for ventilation management, particularly in prolonged and asphyxia precipitated arrest.[27,29,50-52] However, a strategy of passive oxygenation without positive pressure ventilation has shown an association for improved survival during the first few minutes of resuscitation.[128,129] While not fully understood, this may be due to the oxygen reserve present in the body at the moment of cardiac arrest. As researchers identify optimal ventilation strategies to use during cardiac arrest resuscitation, rescuers must continue to understand the effects the lack of ventilation may have on out-of-hospital cardiac arrest physiology.[25,43,44]

Recommendations for the ideal compression-to-ventilation ratio have frequently changed over the years. Before 2000, American Heart Association CPR guidelines suggested a 5:1 ratio for 2-person CPR without an advanced airway.[3,130] Guidelines shifted to recommending the 15:2 ratio before advanced airway placement after the discovery that a 5:1 ratio was associated with a decreased number of total compressions per minute and less favorable perfusion pressures.[130-132] Several different ratios were subsequently explored (eg, 100:2, 50:2, 50:5, 30:2, and 15:1), ultimately changing to the 30:2 compression-to-ventilation ratio recommended today.[5,7,26,133-137]

In an attempt to help limit prolonged chest compression pauses associated with ventilation, researchers have evaluated how

asynchronous ventilation with continuous compressions compared to the traditional interrupted approach.[128,138,139] A cluster-randomized trial allocated EMS practitioners to either continuous compressions (10 breaths/min) or interrupted compressions (with a 30:2 ratio) in the management of non-asphyxia adult out-of-hospital cardiac arrest patients.[139] Advanced airway management was deferred until approximately 6 minutes into the resuscitation.[139] No difference in survival or neurologic function was found between the 2 groups.[139]

Hyperventilation during CPR likely leads to harm. While current guidelines recommend a ventilation rate of 10 breaths/min, the optimal ventilation rate when performing asynchronous ventilation with continuous compressions is not known.[5-7] The porcine model described by Aufderheide showed an association with excessive ventilation rate and poor outcomes.[31,32] Animals ventilated with a rate of 30 per minute resulted in decreased coronary perfusion pressure and survival as compared to those ventilated at a rate of 12 per minute.[31,32] This finding was attributed to increases in intrathoracic pressure (associated with an increased ventilation rate), which is thought to inhibit venous blood flow back to the right heart.[31,32] Hyperventilation also leads to hypocapnia and subsequent cerebral vasoconstriction, further reducing cerebral perfusion and increasing the chance for worse outcomes.[46,140,141] While rescuers need to avoid excessive ventilation, ventilating at lower rates may also lead to poor outcomes. Animals receiving 2 breaths/min in 1 study were found to have significantly lower carotid blood flow and brain-tissue oxygen tension compared to those receiving 10 breaths/min.[142] A recent human study evaluating adult out-of-hospital cardiac arrest patients receiving greater or less than 10 breaths/min found no difference in outcomes between the 2 groups.[143]

5.1 | Effect of varying tidal volumes

Previous research has evaluated the effect varying self-inflatable bag sizes have on tidal volume delivery, peak airway pressures, and rate of gastric insufflation in non-intubated out-of-hospital cardiac arrest patients. Wenzel et al[144] showed that patients ventilated with a 500 mL (pediatric) self-inflating bag received significantly less tidal volume (365 mL vs 779 mL) and developed less peak airway pressure when compared to a 1000 mL (adult) bag. All patients received ~50% FiO_2 via bag-valve-mask; no difference in oxygen saturation was noted.[144] Another study evaluated a 1100 mL (medium-sized) versus a 1500 mL bag, in patients who received room air oxygen with bag-valve-mask ventilation.[145] The medium-sized bag supplied significantly less tidal volume (624 mL vs 738 mL) while having a similar impact on sufficient ventilation (no difference in $PaCO_2$).[145] Patients ventilated with the medium-sized bag had significantly lower PaO_2 than those in the larger bag group; however, supplemental oxygen was not administered.[145] A similar study compared the use of a 700 mL (pediatric) versus a 1500 mL bag on patients receiving bag-valve-mask ventilation without supplemental oxygen.[146] The results were consistent with the previous showing lower recorded tidal volumes (455 mL vs 719 mL) while still preserving effective ventilation.[146] Finally, altering the grip on the self-inflating bag may also affect delivered tidal volumes.[147]

6 | HOW DOES VENTILATION AFFECT OUT-OF-HOSPITAL CARDIAC ARREST OUTCOMES?

A majority of EMS personnel ventilate at significantly high rates during CPR, which may have clinical importance.[31,32,78,148-150] In a porcine model, animals ventilated at 30 versus 12 breaths/min showed decreased survival (likely through decreased coronary perfusion pressure).[31,32] Vissers et al[143] published a retrospective analysis of 337 intubated out-of-hospital cardiac arrest patients using $EtCO_2$ and ventilator pressure data to evaluate the impact ventilation rate had on out-of-hospital cardiac arrest outcomes. Groups were stratified based on the 10 breaths/min recommendation of current guidelines.[5-8] The mean ventilation rate was 15.3 breaths/min.[143] The authors concluded a ventilation rate of ≤10 breaths/min was not associated with significantly improved outcomes (eg, return of spontaneous circulation, survival to hospital discharge, and 1-year survival with a favorable neurological outcome) compared to a ventilation rate of >10 breaths/min.[143]

Despite the lack of outcome data, a recent study further stresses the importance of ensuring effective ventilation in the management of out-of-hospital cardiac arrest. Chang et al[79] retrospectively examined 560 adult patients with out-of-hospital cardiac arrest who received bag-valve-mask ventilation by EMS practitioners with a ratio of 30 chest compressions interrupted by 2 ventilations. Defibrillation pads recorded bioimpedance ventilation waveforms (ie, lung inflation) during chest compression pauses, which measured ventilation delivery. Outcomes were assessed in pre-specified groups, in those patients with ventilation waveforms in <50% versus 50% or more of pauses. The researchers report the resuscitation attempts where ventilation was measured in at least 50% of the pauses were associated with significantly increased rates of return of spontaneous circulation and survival, with an increased likelihood of a favorable neurological outcome.[79]

7 | CURRENT CHALLENGES AND FUTURE DIRECTIONS

While auditory and visual prompts have been shown to assist with ideal ventilation delivery rates, existing technology fails to measure and deliver feedback on real-time out-of-hospital ventilation data reliably.[151,152] This hinders the ability to investigate optimal ventilation management in out-of-hospital cardiac arrest. As such, an important next step in advancing cardiac arrest resuscitation care will be to develop a means to measure ventilation parameters.

Techniques using thoracic impedance signals and capnography to measure ventilation rate have been described, although these strategies come with previously noted limitations.[70-72,74,75] New devices are

being developed to measure ventilation rate, tidal volume, inspiratory time, and expiratory time.[153,154] Evaluation for air leakage can also be monitored.[153] Two recently described devices attach between a manual bag resuscitator and the airway device (ie, mask, supraglottic airway, or endotracheal tube), measuring airflow and estimating ventilation parameters.[153,154] Ventilation feedback can be delivered, assisting rescuers in the delivery of high-quality ventilation.[153,154] The use of these devices by out-of-hospital practitioners showed an association for improved ventilation performance based on pre-specified targets.[153,154] However, these studies were performed in a simulated environment with manikins.[153,154]

It will be important for future devices to deliver reliable and accurate ventilation parameters, provide immediate feedback to out-of-hospital personnel, be cost-effective, able to be used with various airway management techniques, and be durable in the out-of-hospital environment. Once developed, researchers should be able to determine the optimal ventilation strategy for all etiologies of out-of-hospital cardiac arrest resuscitation.

8 | CONCLUSIONS

Out-of-hospital cardiac arrest continues to be a devastating condition despite advances in resuscitation care. Although ventilation is important to out-of-hospital cardiac arrest physiology, there is limited evidence to support ideal ventilation management during CPR. Recent studies suggest that ventilation is associated with outcomes from out-of-hospital cardiac arrest. New devices and strategies should be developed to guide out-of-hospital personnel on optimal clinical performance.

AUTHOR CONTRIBUTIONS

Conception of review: MRN and MRD. Drafting the manuscript: MRN and MRD. Critical revision of the article: MRN, AI, JM, JLB, and MRD.

ORCID

Matthew R. Neth MD https://orcid.org/0000-0002-3882-9637

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AUTHOR BIOGRAPHY

Matthew Neth, MD, is Assistant Professor and Director of EMS in the Department of Emergency Medicine at Oregon Health & Science University, Portland, OR.

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