Clinical paper

Association of ventilation with outcomes from out-of-hospital cardiac arrest

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Abstract

Aim of study: To determine the association between bioimpedence-detected ventilation and out-of-hospital cardiac arrest (OHCA) outcomes.

Methods: This is a retrospective, observational study of 560 OHCA patients from the Dallas-Fort Worth site enrolled in the Resuscitation Outcomes Consortium Trial of Continuous or Interrupted Chest Compressions During CPR from 4/2012 to 7/2015. We measured bioimpedance ventilation (lung inflation) waveforms in the pause between chest compression segments (Physio-Control LIFEPAK 12 and 15, Redmond, WA) recorded through defibrillation pads. We included cases ≥ 18 years with presumed cardiac cause of arrest assigned to interrupted 30:2 chest compressions with bag-valve-mask ventilation and ≥ 2 min of recorded cardiopulmonary resuscitation. We compared outcomes in two *a priori* pre-specified groups: patients with ventilation waveforms in $<50\%$ of pauses (Group 1) versus those with waveforms in $\geq 50\%$ of pauses (Group 2).

Results: Mean duration of 30:2 CPR was 13 ± 7 min with a total of 7762 pauses in chest compressions. Group 1 (N = 424) had a median 11 pauses and 3 ventilations per patient vs. Group 2 (N = 136) with a median 12 pauses and 8 ventilations per patient, which was associated with improved return of spontaneous circulation (ROSC) at any time (35% vs. 23%, $p < 0.005$), prehospital ROSC (19.8% vs. 8.7%, $p < 0.0009$), emergency department ROSC (33% vs. 21%, $p < 0.005$), and survival to hospital discharge (10.3% vs. 4.0%, $p = 0.008$).

Conclusions: This novel study shows that ventilation with lung inflation occurs infrequently during 30:2 CPR. Ventilation in $\geq 50\%$ of pauses was associated with significantly improved rates of ROSC and survival.

Keywords: Heart arrest, Cardiopulmonary resuscitation, Ventilation detection, Outcomes, Bioimpedance

Introduction

Cardiac arrest is the third leading cause of death in the United States with over 400,000 out-of-hospital cardiac arrests (OHCA) occurring annually.[1,2] Starting resuscitation efforts as quickly as possible is essential to maintain perfusion and decrease the duration of ischemia in vital organs. Multiple, large-scale observational and experimental resuscitation studies have informed national and international guidelines on best practices of delivering quality cardiopulmonary resuscitation (CPR). These studies have focused on measuring important characteristics of chest compressions such as rate, depth, and fraction (the proportion of time spent doing chest compressions).[3–6] The results have improved OHCA patient survival-to-hospital discharge rates from 8.2% in 2006 to 12% in 2014, but more improvement is needed.[1] In contrast to chest compressions,

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ventilation metrics have not been included in the analysis of these trials because there has been no widely available way to measure ventilation in the out-of-hospital setting. The role of ventilation in outcome from cardiac arrest is not well understood because ventilation is difficult to measure during the initial and most important stages of resuscitation. Thus, it is unknown whether the quality of ventilations or which ventilation metric is a factor in improving patient outcomes.

In the prehospital setting, emergency medical services (EMS) providers are trained to deliver CPR consisting of chest compressions and ventilations. Studies have shown that chest wall rise and fall from chest compression generates insufficient tidal volume for adequate gas exchange and oxygenation.[7–9] Therefore, providers usually give some form of artificial ventilation to promote oxygenation during resuscitation efforts. During initial resuscitation, rescuers commonly use a bag-valve-mask (BVM) device for ventilation. Traditionally, medical providers are taught to observe chest wall rise and fall as an indication of successful lung inflation. The tidal volume associated with detectable chest wall movement is between 300–500 mL.[10] Capnography can be used to determine if ventilation is present, but capnography is usually measured only after placement of an advanced airway, which occurs later in resuscitation.

In the 1960s, NASA used thoracic bioimpedance to monitor respiratory and cardiac parameters in astronauts during early space launches.[11] Bioimpedance has also been used to measure tidal volume.[12–14] Recently, thoracic bioimpedance recordings have been shown to measure tidal volume and ventilation frequency during CPR.[15–17] When a person inhales and exhales, the chest wall expands and contracts, and thoracic electrical resistance oscillates, which is detected by changes in thoracic bioimpedance. During CPR, thoracic bioimpedance is captured through the defibrillator pads placed on the chest and recorded by the defibrillator. After resuscitation has concluded, software can download and display the thoracic bioimpedance recordings on a computer where it can then be analyzed. The advantage of using thoracic bioimpedance to measure ventilation is that the recording starts when the defibrillator chest pads are placed, which usually occurs during the first few minutes of CPR.

The objectives of this study were to characterize bioimpedance ventilation waveforms during chest compression pauses in 30:2 CPR, to determine the incidence of ventilation during 30:2 CPR, and to assess the association of ventilation (lung inflation) with outcomes from OHCA.

Methods

Setting and design

This is a retrospective, secondary, observational study using defibrillator files and patient care data from the Dallas-Fort Worth (DFW) site of the 30:2 arm of the Resuscitation Outcomes Consortium (ROC) Trial of Continuous or Interrupted Chest Compressions during CPR (CCC) clinical trial.[18] The University of Texas Southwestern Medical Center Institutional Review Board approved this study. The ROC CCC study has a database of over 23,000 cardiac arrest patients

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┌─────────────────────────────────┐          ┌─────────────────────────────────┐
│ 2,911 Patients were enrolled in │ Excluded │ 1,492 Patients were assigned to │
│ the ROC CCC Trial at the        │ ───────► │ the continuous chest compression│
│ Dallas-Fort Worth Site          │          │ arm of the study                │
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│ 1,419 Patients were assigned to │ Excluded │ 252 Patients had a defibrillator│
│ the 30:2 arm of the CCC study   │ ───────► │ other than a LP12 or LP15       │
│                                 │          │ applied during CPR              │
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│ 1,167 Patients had a LP12 or    │ Excluded │ 340 Patients had continuous     │
│ LP15 applied during 30:2 CPR    │ ───────► │ chest compressions or did not   │
│                                 │          │ have obvious 30:2 CPR           │
│                                 │          │                                 │
│                                 │          │ 116 Defibrillator files without │
│                                 │          │ a recorded impedance signal     │
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│ 711 Patients had obvious 30:2   │ Excluded │ 151 Patients had less than two  │
│ CPR                             │ ───────► │ minutes of recorded CPR         │
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│ 560 Patients comprise the       │
│ analyzable cohort               │
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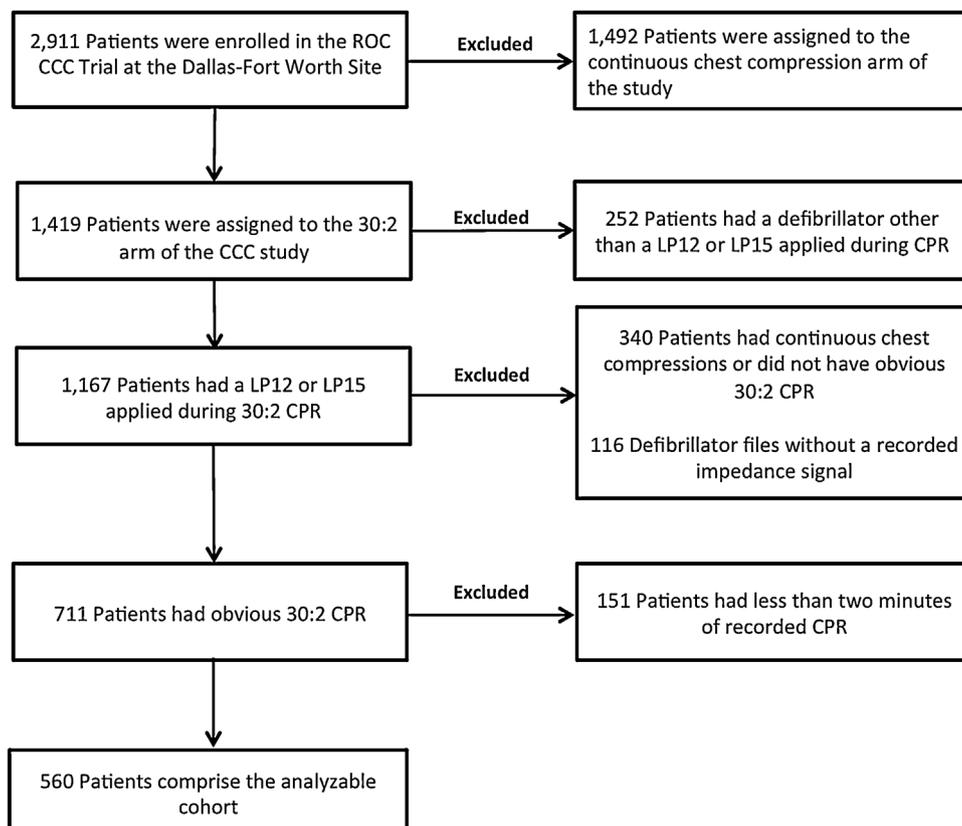
Fig. 1 – Study cohort and exclusions. ROC indicates Resuscitation Outcomes Consortium; CCC, Trial of Continuous or Interrupted Chest Compressions during CPR; CPR, cardiopulmonary resuscitation; LP, LIFEPAK.

that includes defibrillator files. The database also contains data abstracted from electrocardiogram files, EMS patient care reports, and hospital medical records, including survival outcomes.

Patient population

Our study included 560 patients with OHCA enrolled in the ROC CCC study from April 2012 to July 2015 at the DFW ROC site with files available from the LIFEPAK 12 (LP12) and LIFEPAK 15 (LP15) defibrillators (Physio-Control, Redmond, WA, USA) (Fig. 1). Inclusion criteria were non-traumatic cardiac arrest patients at least 18 years old who had defibrillator recordings with a minimum of two minutes of 30:2 CPR (patients received chest compressions interrupted by ventilation in a ratio of 30 chest compressions to 2 ventilations) by EMS providers. Of the 1419 patients assigned to the 30:2 CPR arm, we excluded 340 patients who actually received continuous chest compressions or who did not have obvious 30:2 CPR.[18] Also excluded were 116 cases that lacked a recorded impedance signal and 151 cases with less than two minutes of recorded CPR. Moreover, we excluded 140 files recorded with the Philips MRx defibrillator (Andover, MA) and 112 files recorded with the ZOLL defibrillator (Chelmsford, MA) because the numbers were low and we have not yet studied the software used for recording bioimpedance in those devices.

Measurement

Ventilation waveform analysis

Two physicians (MPC, YL) initially reviewed 200 defibrillator files from patients with OHCA who received 30:2 CPR and attempted ventilation with a BVM device. They developed criteria specifying ventilation bioimpedance waveform characteristics associated with lung inflation. The reviewers inspected each recording for the presence of typical, low frequency lung inflation waveforms during 3 s–15 s pauses in chest compressions and excluded pauses marked for such events as attempted defibrillation, administration of medication, and rhythm checks (Fig. 2).

Prior studies show that >250 mL is a reasonable minimum tidal volume that can result in gas exchange.[19–21] The threshold of 250 mL is an approximation of the minimum amount of tidal volume needed to overcome anatomic and physiologic dead space and produce clinically meaningful gas exchange.

To determine the relationship between tidal volume and bioimpedance waveform amplitude, we studied seven healthy volunteers. The volunteers breathed fixed tidal volumes (250 mL, 300 mL, 400 mL, 600 mL, and 800 mL) given by a Maquet SERVO-I ventilator (Getinge US Sales, Wayne, NJ, USA) through a mouthpiece while a LP12 defibrillator recorded thoracic bioimpedance through electrode pads placed on the chest.
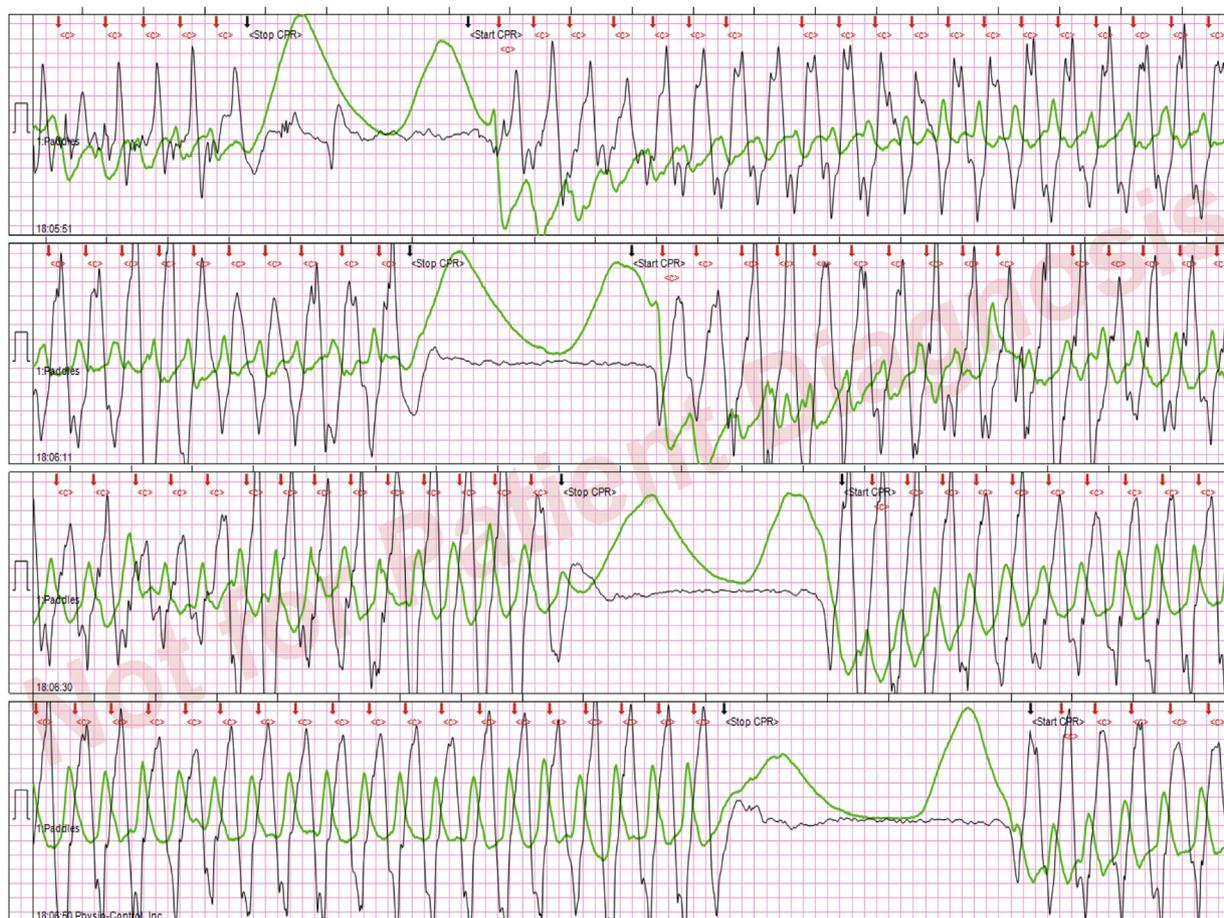

Fig. 2 – The figure is an 80 s segment of a LIFEPAK 12 defibrillator electronic file recorded during 30:2 CPR on a patient in our study. The black line shows the electrical channel, the green line shows thoracic bioimpedance. The green waveforms marked with red arrows show chest compressions; the low frequency waveforms during interruptions in chest compressions show ventilations.

The detailed criteria characterizing acceptable bioimpedance lung inflation waveforms were reported previously.[21] The independent review of the initial 200 files for ventilation incidence had an inter-rater agreement that was excellent ($\kappa > 0.8$) with $\kappa = 0.89$.

After the initial review of 200 patient files, we estimated that 592 patients would be needed for the study to have an 80% power to detect a survival-to-hospital discharge rate of 6% in those who had ventilation waveforms in $< 50\%$ of pauses in chest compressions vs. 9% in those with waveforms in $\geq 50\%$ of pauses at a two-sided alpha level of 0.05.

EMS rescuers downloaded electronic defibrillator files for enrolled patients who received CPR. We collected and analyzed LP 12 and LP 15 defibrillator files recorded from 711 patients in the 30:2 arm of the CCC study. The reviewers (MPC, YL) independently reviewed the files for lung inflation waveforms and found 151 files that had less than two minutes of 30:2 CPR data (Fig. 1). From the remaining 560 files, the inter-rater agreement for the presence of lung inflation waveforms during pauses in chest compressions had $\kappa = 0.94$ ($p < 0.005$).[21] We included ventilation data from the start of chest compressions until placement of an advanced airway.

Statistical analysis

We pre-specified two groups *a priori* for comparison: patients with ventilation waveforms in $<50\%$ of chest compression pauses (Group 1) vs. patients with ventilation waveforms in $\geq50\%$ of pauses (Group 2). The primary outcome was survival to hospital discharge. The secondary outcomes were return of spontaneous circulation (ROSC) at any time, ROSC on arrival at the emergency department (ED ROSC), survival to hospital admission, and neurological outcome (modified Rankin score).

Dose-response relationships between the number of ventilations per pause (VPP) and each of the outcomes (ED ROSC, hospital admission, survival to discharge and mRs of 3 or less) were estimated by natural cubic splines with a single knot point at VPP value of 1 using the library "splines" in the R statistical software package.

All statistical analyses were performed with commercially available statistical packages (SAS, version 9.1.3, Cary, NC; R, version 2.5.1, Vienna, Austria; Stata, version 11, College Station, TX). Descriptive statistics were calculated for witnessed status, initial cardiac rhythm, chest compression rate and fraction over the first 6 min of CPR, ventilation quality metrics, prehospital ROSC, ED ROSC, ROSC at any time, survival-to-hospital discharge, and favorable neurological outcome, defined as a modified Rankin (mRs) score ≤ 3. Summary results are presented as mean ($\pm$SD) or median with interquartile range (IQR). Those with available data were categorized into two pre-specified groups and were compared using Fisher's exact test, with significance level of 0.05. We also used a multiple logistic regression model to calculate unadjusted and adjusted odds ratios (OR) and 95% confidence intervals (CI) of the association between those with ventilation waveforms in $<50\%$ of pauses vs. those with ventilation waveforms in $\geq50\%$ of pauses for both survival and ROSC. The model contained potential confounding variables identified *a priori* including age, sex, bystander-witnessed cardiac arrest, EMS-witnessed cardiac arrest, attempted bystander CPR, public location, and first known EMS rhythm.

Results

We found that the relationship between tidal volume and bioimpedance amplitude was consistent with previous studies (Fig. 3).[12,17] In
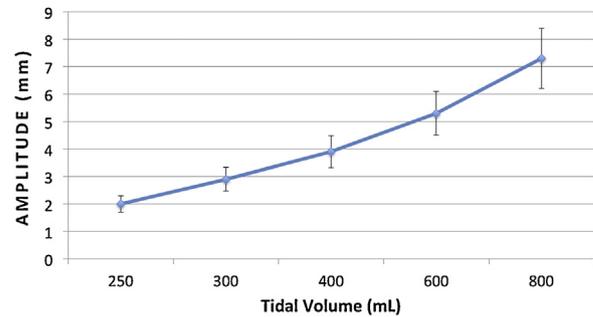
Fig. 3 – Mean ($\pm$SD) bioimpedance amplitude (mm) (y-axis) measured with a LIFEPAK 12 defibrillator vs. tidal volume (mL) (x-axis) of breaths given with a Maquet SERVO-I ventilator in seven volunteers.

particular, the mean ($\pm$SD) bioimpedance amplitude (mm) at 250 mL tidal volume was 2.0 ± 0.6 mm [95% CI 1.8–2.2 mm] and for 300 mL, the amplitude was 2.9 ± 0.7 mm (95% CI 2.7–3.1 mm). Based on these measurements, we set the minimum bioimpedance amplitude value at >2.2 mm ($0.5\,\Omega$) to identify lung inflation waveforms with tidal volumes >250 mL.

The overall mean patient age was 62 years old with a 59% male predominance (Table 1). The mean duration of 30:2 CPR was 13 ± 7 min from the start of chest compressions until placement of an advanced airway. There was a total of 7762 pauses in compressions in the 560 individual patient files. The majority of patients (N = 424, 75.7%) comprised the group (Group 1) with lung inflation waveforms in $<50\%$ of pauses while only 560 (24.3%) had waveforms in $\geq50\%$ of pauses (Group 2) (Table 1). For Group 1 and Group 2, respectively, the median (IQR) number of pauses per patient file was 11 (7, 18) vs. 12 (7, 18) (NS) and the median number of ventilations was 3 (2, 6) vs. 8 (4, 14) per patient file ($p < 0.0001$). Furthermore, for Group 1 vs. Group 2, respectively, the median duration of pauses was 5.3 s (4.5, 6.6) vs. 5.2 s (4.5, 6.6) (NS), median duration of ventilations was 1.8 s (1.6, 2.1) vs. 1.9 s (1.8, 2.1) ($p = 0.022$), and median ventilation waveform amplitude was $1.04\,\Omega$ (0.8, 1.4) vs. $1.07\,\Omega$ (0.8, 1.4) (NS), respectively (Table 1). An amplitude of $1\,\Omega$ is approximately 400 to 500 mL of tidal volume. There were no significant differences between the two groups regarding witnessed status, initial cardiac rhythm, chest compression rate or fraction (Table 1). Those with ventilation waveforms in $\geq50\%$ of pauses had associated improved return of spontaneous circulation (ROSC) at any time (35% vs. 23%, $p = 0.005$), prehospital ROSC (19.8% vs. 8.7%, $p = 0.0009$), ED ROSC (33.1% vs. 21.0%, $p = 0.005$), and survival to hospital discharge (10.3% vs. 4.0%, $p = 0.008$) (Table 1).

The patient survival outcomes were also analyzed using a multiple logistic regression model both unadjusted and adjusted for age, sex, initial cardiac rhythm, witnessed status, bystander CPR, location, and call to arrival time interval. Compared to patients with at least one lung inflation in $<50\%$ of pauses, patients who had at least one lung inflation in $\geq50\%$ of pauses had higher rates of ROSC at the ED (unadjusted OR 2.93, 95% CI 1.56–4.62), hospital admission (unadjusted OR 1.90, 95% CI 1.24–2.92), survival to hospital discharge (unadjusted OR 2.93, 95% CI 1.39–6.18), and modified Rankin score ≤ 3 (unadjusted OR 2.92, 95% CI 1.04–8.20) (Table 2). After adjustment, ORs were similar for ROSC at ED (adjusted OR

Table 1 – Demographics of study participants, chest compression and ventilation quality factors, initial cardiac rhythm, and outcomes by ventilation group: ventilation in less than 50% of pauses vs. ventilation in ≥ 50% of pauses.

	<50% of pauses with ventilation (N = 424)	≥50% of pauses with ventilation (N = 136)	p=
Age years (mean)	62.5	62.6	
Male (%)	251 (59.2)	71 (52.2)	
Witnessed cardiac arrest	172 (40.6)	65 (47.8)	
Initial cardiac rhythm			
VT/VF (%)	93 (21.9)	33 (24.3)	0.56
PEA (%)	105 (24.8)	29 (21.3)	0.49
Asystole (%)	222 (52.4)	73 (53.7)	0.84
No shock advised (%)	4 (0.009)	1 (0.007)	1
Chest compression and ventilation metrics			
Median (IQR) chest compression fraction (%)	76 (67, 82)	74 (63, 80)	0.14
Median (IQR) chest compression rate (Rate/min)	106 (100, 117)	106 (101, 115)	0.75
Median (IQR) number of pauses in chest compressions	11 (7, 18)	12 (7, 18)	0.49
Median (IQR) duration of pauses (s)	5.3 (4.5, 6.6)	5.2 (4.6, 6.6)	0.90
Median (IQR) duration of ventilations (s)	1.8 ((1.6, 2.1)	1.9 (1.8, 2.1)	0.022
Median (IQR) number of ventilations (N)	3 (2, 6)	8 (4, 14)	<0.0001
Median (IQR) ventilation waveform amplitude (Ω)	1.04 (0.8, 1.4)	1.07 (0.8, 1.4)	0.43
Outcomes			
Prehospital ROSC (%)	37 (8.7)	27 (19.8)	0.0009
ED ROSC (%)	89 (21.0)	45 (33.1)	0.0054
Any ROSC (%)	96 (22.6)	48 (35.3)	0.0047
Survival (%)	17 (4.0)	14 (10.3)	0.008
mRs ≤3 (%)	9 (2.1)	7 (5.1)	0.078

Interquartile range (IQR); Ventricular tachycardia (VT); Ventricular fibrillation (VF); Pulseless electrical activity (PEA); Return of spontaneous circulation (ROSC); Emergency department (ED), modified Rankin score (mRs).

Table 2 – Multiple logistic regression analysis of the association between patients who received ≥ one ventilation in at least 50% of pauses and out-of-hospital cardiac arrest outcomes. The multivariable model was adjusted for age, sex, initial cardiac rhythm, witnessed status, bystander CPR, location, and 911 call to emergency medical services arrival time interval.

Outcome	Unadjusted analysis Odds ratio (95% confidence interval)	Adjusted analysis Odds ratio (95% confidence interval)
ROSC at ED	2.68 (1.56–4.62)	2.84 (1.47–5.48)
Survival to hospital Admission	1.90 (1.24–2.92)	1.92 (1.15–3.22)
Survival to discharge	2.93 (1.39–6.18)	2.13 (0.83–5.47)
Favorable neurological Outcome (mRs ≤ 3)	2.92 (1.04–8.20)	4.14 (1.14–15.05)

Return of spontaneous circulation (ROSC); Emergency department (ED); Modified Rankin Score (mRs).

2.84, 95% CI 1.47–5.48) and survival to hospital admission (adjusted OR 1.92, 95% CI 1.15–3.22) (Table 2). Adjustment reduced the OR for survival to hospital discharge to 2.13 (95% CI 0.83–5.47), which was not statistically significant. Adjustment increased the OR for favorable neurological outcome to 4.14 and preserved statistical significance (95% CI 1.14–15.05).

Dose-response relationships between the number of ventilations per pause and each of the outcomes (ED ROSC, hospital admission, survival to discharge and mRs of 3 or less) were estimated by natural cubic splines (Fig. 4).

Discussion

This novel study showed that patients who received ventilation in at least half of the pauses in chest compressions during 30:2 CPR for OHCA had associated significantly improved ROSC, survival to hospital discharge, and favorable neurological outcome. Until recently, it has been difficult to measure quality of ventilations given to OHCA patients during early 30:2 CPR. This is the first study that measured bioimpedance ventilation waveforms during OHCA resuscitation and its possible effects on survival outcomes. When
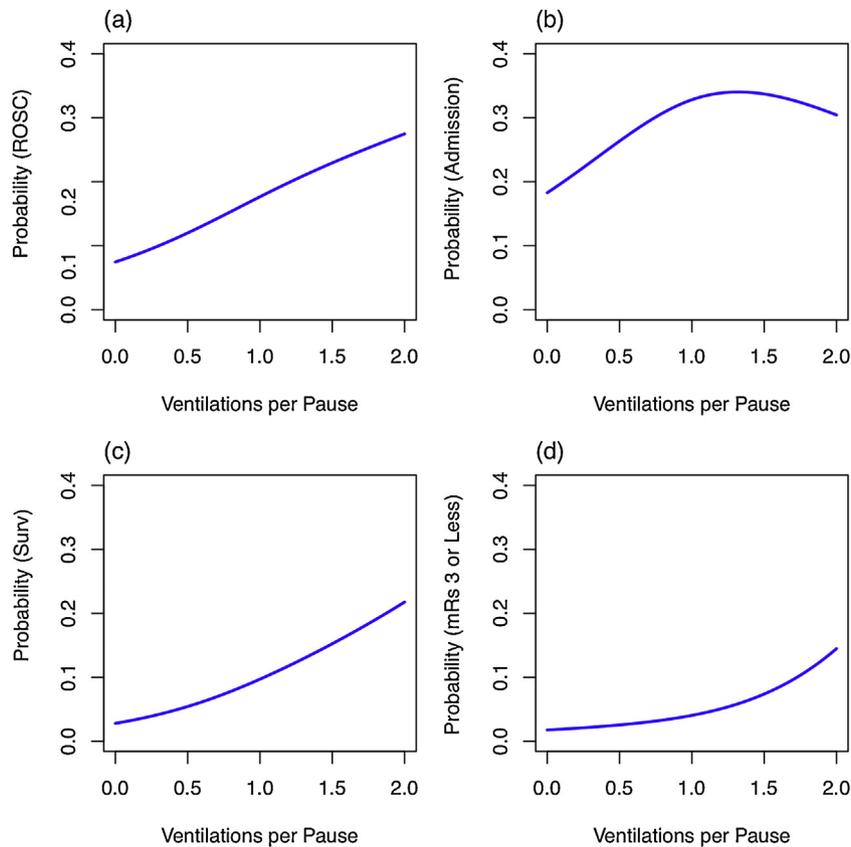
Fig. 4 – Association between number of ventilations per pause and outcomes using natural splines: (a) probability of return of spontaneous circulation (ROSC) on arrival in the emergency department vs. ventilations per pause, (b) probability of hospital admission (admission) vs. ventilations per pause, (c) probability of survival (surv) to hospital discharge vs. ventilations per pause, (d) probability of mRs of 3 or less (favorable neurological outcome) vs. ventilations per pause.

ventilation is attempted with a BVM device in OHCA patients, there may be an assumption that it results in lung inflation most of the time. However, our results unexpectedly showed that less than 25% of patients had lung inflation in more than half of the pauses in chest compressions. A post-hoc analysis demonstrates a dose-response relationship between ventilation and outcomes.

Why did so few patients in our study receive ventilation during CPR? Ventilation with a BVM device is a difficult skill to perform properly and must be practiced to maintain proficiency.[22] The person performing ventilation must extend the neck, or place an oral airway, and/or perform a jaw thrust maneuver in order to maintain an open airway, a tight mask seal on the face must be maintained to prevent air from leaking around the mask, and the rescuer must then simultaneously squeeze the manual ventilator over 1 to 1.5 s. Our study showed no significant difference in the number of pauses between Group 1 and Group 2 patients (11 vs. 12 pauses). However, Group 2 patients received significantly more ventilations than Group 1 patients (8 vs. 3 ventilations). The study suggests that the rescuers in both Groups attempted ventilation about the same number of times per patient, but these attempts frequently did not result in lung inflation in Group 1 patients.

In the last decade, lay rescuer chest-compression-only resuscitation (Hands-Only CPR) has been associated with improved outcomes.[23–25] Possible reasons for such improvement include less

interruption in chest compressions compared with standard 30:2 CPR, increased willingness to perform CPR because mouth-to-mouth ventilation is not part of the protocol, and earlier and more frequent lay rescuer CPR. In addition, with Hands-Only CPR, dispatchers are able to give "just-in-time training" over the telephone to untrained rescuers.[24] Passive ventilation associated with chest compressions probably does not contribute to gas exchange or oxygenation because tidal volume is much less than dead space.[7–9] However, ventilation may be omitted during the first few minutes of CPR because there is sufficient oxygen stored on hemoglobin to last six or more minutes since blood flow during CPR is 20%–25% of baseline, at best. Nevertheless, ventilation should probably be given soon after EMS arrives because it is likely that the patient has not received oxygen for 10–20 min (time intervals for rescuer to recognize an emergency and to call 911 plus EMS response time interval, plus time interval to assemble the airway equipment). This may be another reason Group 2 patients had improved outcomes in our study since they received more effective early ventilations compared with Group 1 patients. Decreasing time to restore specific physiologic functions to the normal steady state is key to mitigate the effects of post-cardiac arrest syndrome (PCAS).[26]

Various aspects of ventilation in OHCA patients have been studied such as the compression to ventilation ratio, ventilation frequency, and methods of ventilation.[27] Oxygenation is important to sustaining vital

organs, and in resuscitation the goal is to artificially provide such support until restoration of function. The methods prehospital providers have for administering ventilation include a manual ventilator with a mask, or with supraglottic airways or an endotracheal tube, or passive ventilation. Studies comparing these ventilation methods have shown mixed results regarding effects on outcomes.[18,28] Most OHCA studies have not attempted to measure lung inflation. While some have measured end-tidal CO_2 (ETCO$_2$), usually the measurement is available only after placement of an advanced airway. In our study, the mean time interval for advanced airway placement was 13 min after the start of CPR. Thus, ETCO$_2$ measurement usually does not include the most important phases of CPR. The advantage of bioimpedance for ventilation measurement is that it is recorded as soon as the defibrillator chest pads are applied. Most EMS protocols call for early placement of a defibrillator during CPR to detect the presence of a shockable rhythm. Our study shows that lung inflation cannot be assumed to have occurred during CPR and that it must be measured to understand fully the impact of ventilation on outcomes.

For patients who need lung protective ventilation strategies in the hospital, ventilator tidal volumes as low as 4 ml/kg have produced adequate gas exchange. This tidal volume approximates the tidal volume threshold used in our study for the average sized patient.[15] It is unknown if there is an optimal tidal volume associated with improved outcomes during CPR for OHCA. Studies show that giving high tidal volumes to OHCA patients may worsen their neurological outcomes. [29]

Our study reported detailed information regarding ventilation during CPR for OHCA including ventilation incidence and frequency, inhalation and exhalation duration, and tidal volume. Future studies with larger sample sizes are needed to evaluate the effect of these ventilation metrics on patient outcomes during out-of-hospital cardiac arrest. This may be another complex piece of the puzzle that warrants further investigation because it could help improve survival rates.

Limitations

This study is a single site study so the results may not be generalizable to other populations. Prior ROC multicenter studies found variation in survival and other outcomes across ROC sites. Other site differences include EMS response interval, quality of CPR, and bystander CPR rates.

The defibrillator files were from only one device manufacturer and our ventilation waveform criteria should be verified in devices from other manufacturers. This study is a secondary observational analysis of data from a clinical trial that addresses a question that was not the purpose of the original trial. Associations between ventilation and outcomes may not represent causal effects.

Conclusions

This novel study demonstrates that ventilation with lung inflation occurs infrequently during 30:2 CPR for out-of-hospital cardiac arrest. Ventilation with measurable lung inflation in $\geq$50% of pauses was associated with significantly increased rates of ROSC and survival, and increased likelihood of favorable neurological outcome. Future studies should evaluate the effect of ventilation metrics in multi-center studies and larger cohorts.

Conflicts of interests

Dr. Idris receives grant support from the US National Institutes of Health (NIH), the American Heart Association, and the US Department of Defense. He serves as an unpaid volunteer on the American Heart Association National Emergency Cardiovascular Care Committee and the HeartSine, Inc. Clinical Advisory Board.

Acknowledgements

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