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Clinical paper

Comparison of volume-controlled, pressure-controlled, and chest compression-induced ventilation during cardiopulmonary resuscitation with an automated mechanical chest compression device: A randomized clinical pilot study



Kristina Fuest^a, Florian Dorfhuber^a, Marco Lorenz^{b,a}, Falk von Dincklage^b, Rudolf Mörgeli^b, Karl Friedrich Kuhn^b, Bettina Jungwirth^{a,c}, Karl-Georg Kanz^d, Manfred Blobner^{a,c}, Stefan J Schaller^{a,b,*}

^a Technical University of Munich, School of Medicine, Klinikum rechts der Isar, Department of Anesthesiology and Intensive Care, Ismaninger Str. 22, Munich, Germany

^b Charité – Universitätsmedizin Berlin, Corporate Member of Freie Universität Berlin and Humboldt-Universität zu Berlin, Department of Anesthesiology and Operative Intensive Care Medicine, Charitéplatz 1, Berlin, Germany

^c University of Ulm, School of Medicine, Department of Anesthesiology and Intensive Care Medicine, Germany

^d Technical University of Munich, School of Medicine, Klinikum rechts der Isar, Department of Trauma Surgery, Ismaninger Str. 22, Munich, Germany

Abstract

Aim of the study: Automated mechanical chest compression devices (AMCCDs) can help performing high-quality cardiopulmonary resuscitation (CPR). Guidelines for CPR are lacking information about the optimal ventilation mode during CPR using AMCCDs. Aim of this pilot study was to compare three common ventilation modes during CPR using AMCCD.

Methods: In this randomized controlled trial, we included patients with an out-of-hospital cardiac arrest arriving at the resuscitation room receiving chest compressions via AMCCD with an expected continuation of at least 15 min. Patients were randomly assigned to three groups: biphasic positive airway pressure with assisted spontaneous ventilation (BIPAP) with assisted spontaneous breathing, continuous positive airway pressure (CPAP) and volume-controlled ventilation (VCV). Outcomes were tidal volume, respiratory minute volume, and end-tidal CO₂ during the study period. Groups were compared using generalized linear models. Data is given as median and interquartile ranges.

Results: Of 53 screened patients, 30 were randomized. The tidal volume was significantly ($p < 0.05$) lower in patients of the CPAP group (68 [64–83] ml) compared with those of the BIPAP (349 [137–500] ml), while the respiratory minute volume differed between the CPAP group (6.2 [5.3–8.1] l/min) and both the BIPAP (7.1 [6.7–10.2] l/min) and VCV group (7.2 [3.7–8.4] l/min).

Conclusions: All ventilation modes achieved an adequate respiratory minute volume during CPR with an AMCCD. However, BIPAP seems to be superior due to the higher tidal volume. Therefore, we recommend starting mechanical ventilation when using AMCCD with BIPAP ventilation to avoid risks related to dead space ventilation.

Keywords: Cardiopulmonary resuscitation, CPR, Automated mechanical chest compression device, Ventilation mode

* Corresponding author at: Charité – Universitätsmedizin Berlin, CC07 – KAI, Charitéplatz 1, 10117 Berlin, Germany.

E-mail address: stefan.schaller@charite.de (S.J Schaller).

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Background

The survival rate of cardiopulmonary resuscitation (CPR) is affected by its quality.^{1,2} Sufficiently applied CPR uses a ratio of 30 chest compressions, with a depth of 5 cm and a frequency of 100–120 compressions per minute, to 2 breaths.³ In particular, the European Resuscitation Council Guidelines for Resuscitation emphasize the importance of minimally interrupted high-quality chest compressions during CPR³ and as well as the importance of early chest compressions.⁴

Automated mechanical chest compression devices (AMCCDs) are designed to meet the conditions of a constant and reliable CPR performance.⁵ Therefore, current guidelines recommend AMCCDs as a reasonable alternative in situations where manual chest compressions are impractical or endanger the providers' safety.³

However, there is a lack of prospective studies or information on optimal ventilation strategies during CPR using AMCCDs. Moreover, studies investigating the effects of AMCCDs do not sufficiently address ventilation strategies and quality during the application of assist devices. Accordingly, only expert-based recommendations regarding ventilation during CPR using AMCCDs are currently available.⁶

We compared three different ventilation strategies (biphasic positive airway pressure with assisted spontaneous breathing, chest compression-induced ventilation with continuous positive airway pressure and volume-controlled ventilation) during CPR using AMCCDs, analysing ventilatory parameters as endpoints of this randomized clinical pilot study.

Methods

This randomised controlled monocentric pilot trial was performed between November 1st, 2017 and December 12th, 2019 at the Technical University of Munich, Germany. The study was approved by the ethical committee of the Medical Faculty of the Technical University of Munich (159/17), which approved deferred consent for surviving patients and waived consent for expired patients. The study is registered at Clinicaltrials.gov (NCT03347175).

Patients were enrolled by the attending anaesthesiologist. Eligible patients had an ongoing out-of-hospital cardiac arrest when arriving at the resuscitation room, an endotracheal tube in place and were receiving chest compressions by the LUCAS[®] Device (Physio Control, Redmond USA). This AMCCD is routinely applied for transport of patients under CPR by the out-of-hospital emergency teams in Bavaria^{7,8}, and provides 100 ± 2 compressions per minute with a depth of 40–53 mm, depending on the suction cup's position. Patients were excluded if the attending anaesthesiologist expected to not continue with the resuscitation for at least 15 min.

Following arrival and transfer to the resuscitation room, the patient was positioned on the computer tomography table while continuing CPR with the AMCCD of the out-of-hospital emergency team. After connection to the monitoring and ventilator of the resuscitation room, the patient was ventilated according to the study protocol and the study period began. In parallel, a full-body CT was performed, and blood values were analysed. Additional peripheral or central venous access ports were placed, and an artery was cannulated.

The study intervention analysed the type of ventilation during the first 15 min following arrival at the resuscitation room. All patients were ventilated with an Oxylog3000[®] (Drägerwerk AG & Co. KGaA, Lübeck, Germany). Patients in the “BIPAP” group were ventilated using “biphasic positive airway pressure with assisted spontaneous breathing (BIPAP-ASB)”. The inspiratory pressure was set to 26 cm H₂O, spontaneous breathing was assisted with 10 cm H₂O and a steep ramp, the positive end-expiratory pressure (PEEP) to 5 cm H₂O, and the respiratory rate to 12/min. The ventilation mode for patients in the “CPAP” group was a passive inhalation and exhalation through the chest compression combined with continuous positive airway pressure (CPAP), i.e., a “chest compression-induced ventilation with continuous positive airway pressure”. The ventilator settings for this mode were a PEEP of 20 cm H₂O and no ASB. Patients in the “VCV” group were ventilated using “volume-controlled ventilation”. The tidal volume was set to 6 and 8 ml/kg of assumed ideal body weight, the PEEP to 5 cm H₂O, and the respiratory rate to 12/min. All groups shared the maximum inspiratory pressure of 40 cm H₂O. The treatment team was allowed to change the ventilation settings for safety reasons at any time during the 15-minute study period, if the patient's ventilation was deemed insufficient.

The outcome of the study was the quality of ventilation during the first 15 min period after admission to the emergency room, connection to the study ventilator, and start of the allocated ventilation protocol. The primary endpoint was the tidal volume, and the secondary endpoints were respiratory minute volume and end-tidal CO₂. Tidal volume and respiratory minute volume were chosen as quality markers of ventilation, and end-tidal CO₂ as a quality marker of the gas exchange. These values were collected by the software of the ventilator. Several exploratory outcome parameters were additionally assessed, such as the effective respiratory rate, the effective PEEP, and the effective maximum inspiratory pressure.

The return of spontaneous circulation (ROSC) was attained if the patient had spontaneous circulation at least once during resuscitation. Since establishment of arterial access under CPR is usually difficult and cannot be reliably distinguished from venous access, pH and pCO₂, but not pO₂, were evaluated once at the end of the study period.

Continuous variables were collected automatically by a computer connected via RS-232 to the ventilator and running VentView[®] Software (Drägerwerk AG & Co. KGaA, Lübeck, Germany). The software saves all the ventilator data (such as pressure levels and ventilation mode) in intervals of ten seconds. Blood gas samples were analysed on a Rapid-Point 500[®] (Siemens Healthcare GmbH, Erlangen, Germany) point-of-care analyser. Patient characteristics were collected from the charts of the out-of-hospital emergency teams and the anaesthesia documentation of the in-hospital emergency team. Patient history was obtained by reviewing information included in the prehospital report, patients' relatives or our hospital system.

Randomisation was performed 1:1:1 using the algorithm provided on randomization.com see <http://www.jerrydallal.com/random/randomcite.htm>. No block-randomisation was done. The assignments were prepared by SJS and kept in sealed opaque envelopes.

The study could not be conducted as a fully blinded study, as the treatment team had to set the ventilator according to the group allocation. Due to the setting of the study and the expected low recruitment rate of two per month, we were unable to guarantee a second blinded team to separate management of the intervention from patient care. That the patients were blinded is implied. The team

responsible for data plausibility checks, evaluation and analyses was not informed about the interventional group, although it cannot be excluded that the treatment allocation could be derived from the collected data.

The target of 10 patients for each group was set without sample size calculations, as there is no data yet available to serve for this purpose.

The primary analysis was a per-protocol analysis assigning all patients to the ventilation mode they had actually received. Thus, only values collected during the assigned treatment were analysed.

Statistical analysis was conducted using R version 3.6.2. Repeated measurement generalised linear models were used for time series. An analysis of variance was performed as a post-hoc analysis. Further post-hoc analyses and analyses of discontinuously available data were performed with Kruskal-Wallis-tests, Mann-Whitney-U-tests, and Chi-square-tests, as appropriate. The hierarchical approach was used to correct for multiple comparisons within one variable. Due to the feasibility nature of a pilot study, we did not correct for the multiple testing of similar variables.

All blood sample values reported as “high” or “low” by the point-of-care analyser were assigned the upper or lower bound of their respective range, according to the concept of informatively missing values described by Lachin.⁹ The analyses of end-tidal CO₂, pH and pCO₂ were adjusted for their respective confounders, including sodium bicarbonate infusions, timing of the blood sampling, and the source of the sample (venous or arterial).

Results

We randomized 30 of 53 screened patients and allocated ten patients to each ventilation mode (Fig. 1). Patients were predominantly male (80%), with a median age of 67 [56–77] years (Table 1). Two patients were not ventilated according to the assigned mode: one patient assigned to CPAP was ventilated with BIPAP-ASB mode, while another was assigned to BIPAP-ASB and was ventilated with VCV.

Ventilation parameters are displayed in Fig. 2, values and results of the statistical analyses are presented in Table 2. The tidal volumes differed significantly between treatment groups (intervention: $p = 0.031$) with significantly higher tidal volumes with BIPAP-ASB as compared with CPAP. During the 15-min-periods, the tidal volumes increased while the respiratory minute volumes decreased (time: $p = 0.006$ and $p = 0.019$, respectively). Different trajectories of respiratory minute volume (time \times intervention $p = 0.002$) are due to the exclusive decrease in patients in the CPAP group ($p < 0.05$ for each post-hoc analysis). Analysis of end-tidal CO₂ levels was adjusted for intravenous infusion of sodium bicarbonate solution (8.4 g in 100 ml) administered to 17 of 30 patients during the 15-min study period. Overall, there was an increase in end-tidal CO₂ (time: $p = 0.014$) based on different trajectories in the treatment groups (time \times intervention: $p = 0.020$).

In the exploratory analysis, the variables PEEP and respiratory rate confirmed the set ventilator values according to the treatment groups. The maximum inspiratory pressure was significantly different among groups ($p = 0.035$), decreasing over time (time: $p < 0.001$), but also with varying trajectories among groups (time \times intervention: $p = 0.020$). This was primarily based on the decreasing trajectories within the BIPAP and the CPAP groups, in contrast to the increasing trajectory of the VCV group ($p < 0.05$ for

the post-hoc comparisons of the trajectories of BIPAP vs. VCV and of CPAP vs. VCV, respectively).

During the study period, the ventilation settings of three patients with VCV were adapted without changing the mode, while the ventilation mode of three further patients were changed. Two patients initially ventilated with CPAP were changed to BIPAP (7th min) and VCV (6th min), respectively. The third patient was changed from VCV to CPAP (13th min). At the end of the study period, a fourth patient's CPAP ventilation was switched to BIPAP-ASB ventilation.

The adjusted last measured pH and pCO₂ values showed a significantly higher pH and a significantly decreased pCO₂ in the BIPAP group compared to the CPAP and VCV groups ($p = 0.022$ and $p = 0.042$, respectively, see Table 2).

Discussion

This pilot trial indicates that BIPAP-ASB provided significantly higher tidal volumes, resulting in increasing minute volumes and end-tidal CO₂ values, higher pH, and lower pCO₂ values in patients under CPR with an AMCCDs when compared with chest compression-induced ventilation supported by CPAP. While maximum inspiratory pressure and PEEP did not differ significantly between BIPAP-ASB and VCV ventilated patients, higher pH and lower pCO₂ in the BIPAP group support the notion that the trend to higher tidal volumes with BIPAP-ASB may contribute to superior ventilation over time. Furthermore, the decrease in effective PEEP and maximal inspiratory pressure in the BIPAP group with unchanged settings during the trajectory may indicate a less traumatic ventilation.

The benefit of mechanical ventilation during chest compression was disputed for many years due to two major side-effects. First, ventilation of patients lacking a secure airway device was inherently ineffective, with the additional risk of inflating the stomach, hampering ventilation of the basal lung segments, reducing blood flow to the right heart, and increasing the risk of regurgitation, aspiration and, in the worst case, oesophageal or gastric rupture.^{10,11} Second, when patients did have a secure airway, mechanical ventilation increased the risk of pulmonary barotrauma due to the high inspiratory pressures with simultaneous chest compression and inspiration.¹² Accordingly, corresponding animal studies (most of them in pigs) focussed on either passive, assisted, or synchronised ventilation modes.

The idea of avoiding mechanical ventilation with any kind of intermittent positive pressure during chest compression was based on passive inhalation and exhalation through the chest compression. Since the so-called chest compression-induced ventilation led to hypercapnia and hypoxia,¹³ it was successfully combined with either CPAP using 100% oxygen¹⁴ or high-flow oxygen insufflation¹⁵ in pig models. In our study in humans, however, we were not able to confirm sufficient CO₂ elimination when CPAP is added to chest compression-induced ventilation. The low end-tidal partial pressure of CO₂ and the high pCO₂ values in blood samples of patients in the CPAP group is indicative of hypoventilation, most likely due to the low tidal volumes and the associated higher fraction of dead space ventilation.

Although the high-flow approach using the Boussignac-tube additionally reduces dead space ventilation by separating inhalation and exhalation flows improving oxygenation in CPR with AMCCD,^{16,17} this technology requires large oxygen supplies increasing the risk of explosion due to a constant oxygen flow of 15 l/min when used in an emergency ambulance.

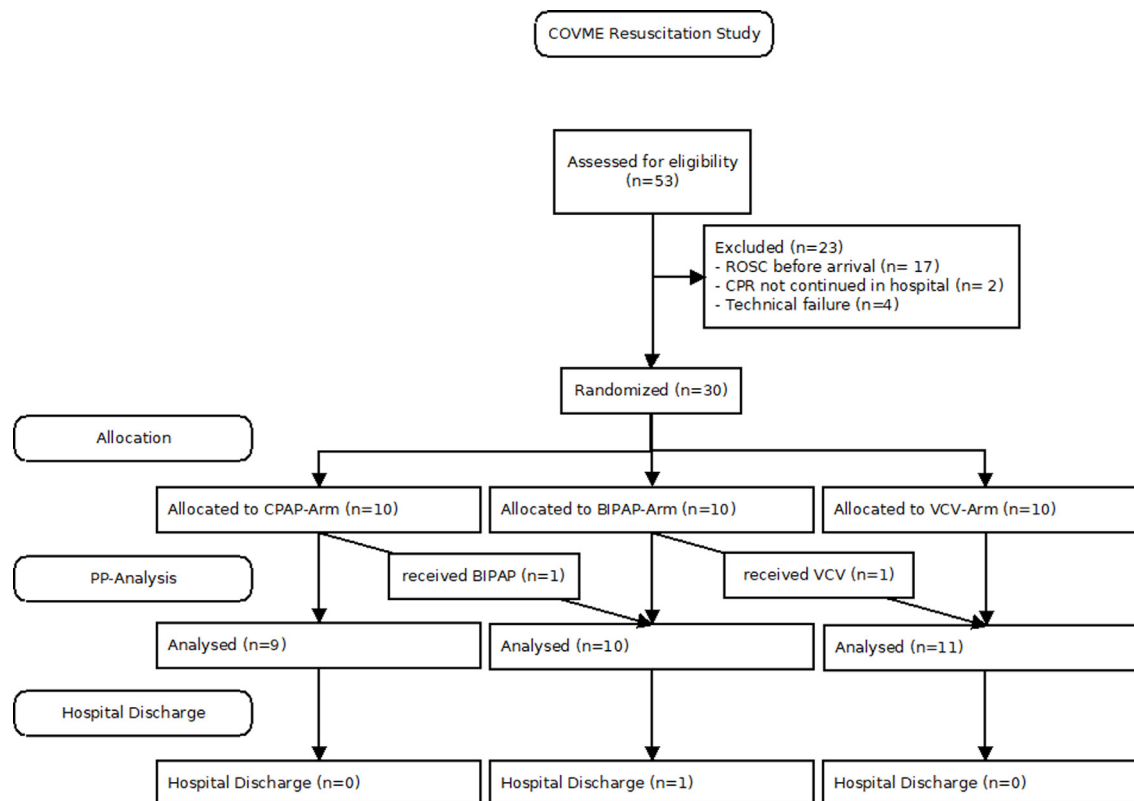


Fig. 1 – CONSORT Diagram. BIPAP, biphasic positive airway pressure; CPAP, continuous positive airway pressure; CPR, cardiopulmonary resuscitation; ROSC, return of spontaneous circulation; VCV, volume-controlled ventilation.

Improvements in ventilator technologies, especially relating to pressure sensors, suggested that assisted or controlled ventilation techniques would be possible under AMCCD without barotrauma of the lung.¹⁸ A more advanced ventilation protocol called “chest compression synchronized ventilation” provides an instant inspiratory pressure at the beginning of the chest compressions measuring the concomitant increase of the airway pressure.¹⁹ Both technologies, however, ventilate the patients at a rate of 100/min, synchronous with the chest compressions with small tidal volumes and major death space fractions.

Normoventilation is more feasible when ventilators are used either in volume or pressure-controlled mode. In this pilot study, tidal volumes provided by both modes were significantly higher than by the chest compression-induced ventilation supported by CPAP. The lower variance within the VCV group is due to the mode and given by its setting. BIPAP mode, however, takes into account the different body habitus without requiring weight and height, which is often unknown in an out-of-hospital resuscitation situation. Importantly, for both techniques, maximum inspiratory pressure limits were set to 40 cm H₂O but effective values remained significantly lower. In

Table 1 – Baseline characteristics. *Except for the initial end-tidal CO₂ (p = 0.03), no significant differences (p > 0.05) were observed for any characteristic among the groups. Scaled variables are presented as median [interquartile range], dichotomous variables are presented as n (%).

Characteristics*	BIPAP	CPAP	VCV
Male sex n (%)	9 (90%)	7 (70%)	8 (80%)
Age (years)	71 [51–78]	66 [57–80]	65 [57–75]
Creatinine (mg/dl)	1.5 [1.3–1.7]	1.6 [0.8–1.8]	1.5 [1.3–1.7]
Troponin (ng/l)	72 [52–142]	76 [75–292]	85 [54–189]
CRP (mg/l)	8 [4–16]	16 [2–21]	3 [1–11]
Glucose [mg/dl]	296 [249–321]	309 [229–432]	264 [191–324]
Procalcitonin (ng/ml)	0.1 [0.1–0.1]	0.1 [0.1–0.3]	0.1 [0.1–0.1]
Initial pH	6.93 [6.82–7.28]	6.71 [6.58–6.89]	6.73 [6.63–6.95]
Initial paCO ₂ (mmHg)	28 [23–34]	12 [2–20]	16 [8–36]
Initial end-tidal CO ₂ (mmHg)	29 [16–36]	12 [2–19]	28 [15–40]

BIPAP, biphasic positive airway pressure; CPAP, continuous positive airway pressure; CRP, C-reactive protein; PEEP, positive end-expiratory pressure; VCV, volume-controlled ventilation.

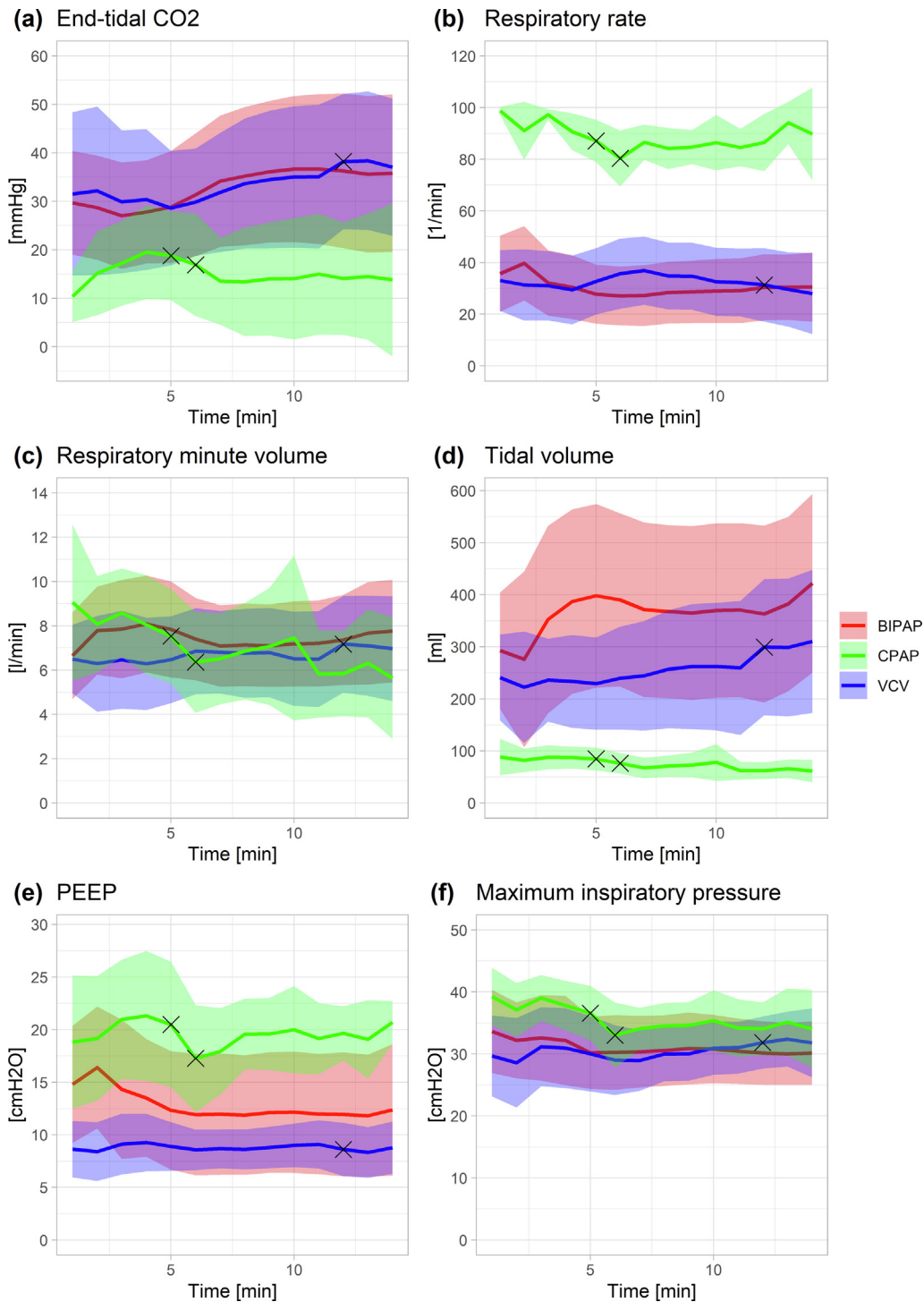


Fig. 2 – Ventilation parameters during the 15-min-period in the three different ventilation modes. The trajectories are presented as means with 95% confidence interval: BIPAP is shown as red, VCV as blue, and CPAP as yellow. See Table 2 for values. Individual values were censored (X) if the ventilation mode was changed during the 15-minute-period. BIPAP, biphasic positive airway pressure; CPAP, continuous positive airway pressure; PEEP, positive end-expiratory pressure; VCV, volume-controlled ventilation.

Table 2 – Primary and secondary outcomes. Continuous variables of the per protocol groups are presented as medians [interquartile range], dichotomous variables are presented as n (%).

	BIPAP (n = 10)	CPAP (n = 9)	VCV (n = 11)	p-value (intervention)	p-value (time)	p-value (time × intervention)
Primary Outcome						
Tidal volume (ml)	349 [137–500]	68 [64–83]	196 [159–278]	0.031*	0.006	0.073
Secondary Outcomes						
Respiratory minute volume (l/min)	7.1 [6.7–10.2]	6.2 [5.3–8.1]	7.2 [3.7–8.4]	0.411	0.019	0.002* §
End-tidal CO ₂ (mmHg) ¹	33 [24–34]	16 [5.5–26]	31 [18–39]	0.462	0.014	0.020
Exploratory Outcomes						
Ventilation						
Respiratory rate (/min)	27 [15–39]	87 [85–92]	27 [17–44]	<0.001* §	<0.001	0.176
PEEP (mbar)	12 [7–14]	22 [19–25]	7 [6–11]	0.006	0.273	0.002* § §
Maximum inspiratory pressure (mbar)	32 [24–39]	39 [33–40]	29 [25–36]	0.035	<0.001	<0.001§ §
Blood gas sample						
pCO ₂ (mmHg)	58 [42–125]	103 [60–143]	96 [90–127]	0.042	n. a.	n. a.
pH	6.94 [6.82–7.23]	6.72 [6.58–6.86]	6.76 [6.65–7.03]	0.022* §	n. a.	n. a.
General						
Ventilation Mode changed (n)	0 (0%)	3 (33%)	1 (9%)	0.892	n. a.	n. a.
ROSC [#] (n (%))	2 (20%)	2 (22%)	1 (9%)	0.866	n. a.	n. a.

¹ The model was adjusted for infusion of sodium bicarbonate;

* shows a significant difference between BIPAP and CPAP in the post-hoc analysis;

§ shows a significant difference between VCV and CPAP in the post-hoc analysis;

§ shows a significant difference between BIPAP and VCV in the post-hoc analysis;

return of spontaneous circulation was achieved for at least once in a patient; n.a., not applicable; BIPAP, biphasic positive airway pressure; CPAP, continuous positive airway pressure; PEEP, positive end-expiratory pressure; VCV, volume-controlled ventilation.

fact, the effective maximum ventilation pressure in the BIPAP-ASB group decreased over time not only compared to the CPAP group, but also to the VCV group, suggesting that it could reduce the risk of barotrauma.

The higher tidal volumes in patients ventilated with BIPAP-ASB may be also attributed to the assisted spontaneous breathing component with 10 cm H₂O and a steep ramp with a high initial flow (100 l/min), additionally providing inspiratory volumes comparable to the “chest compression synchronized ventilation”.¹⁹

Interestingly, ventilation mode was most often changed in the CPAP group, possibly indicating that anaesthesiologists are uncomfortable with this mode during CPR. The resulting changes within the ventilation parameters (Fig. 2) do not indicate that switching modes was necessary or that it substantially improved ventilation.

The study has limitations, especially if regarded beyond the actual aim of a pilot study. We have consciously refrained from making statements on treatment success or pulmonary barotrauma. Furthermore, insertion of an arterial line was challenging during AMCCD, so that the trajectory of arterial O₂, CO₂ and pH could not be monitored in every case. We also included values from blood samples taken from venous vessels. The measured ventilation parameters are not only influenced by the ventilation modes, but also by the selected settings and not least by the patients. The small benefits of BIPAP-ASB would most likely disappear if VCV had also been augmented with assisted spontaneous breathing. Since not every ventilator can use the applied ventilation modes, the demonstrated ventilation strategies might not be applicable in every situation. Despite randomisation, the small group sizes do not allow for conclusive recommendations on the most appropriate ventilation method. The target of ten patients for each group was set without sample size calculations, as the protocol was conducted as a pilot study. Furthermore, the study was single-blinded, so as to allow patient recruitment 24/7 without an additional blinded study team present.

Conclusion

This pilot study investigated for the first time different ventilation modes in patients during CPR with AMCCDs. All ventilation modes achieved an adequate respiratory minute volume, however, BIPAP-ASB seems to be superior during CPR with AMCCD in achieving an adequate tidal volume. As low tidal volumes increase the risk of death-space ventilation, our analyses suggest that BIPAP-ASB might be the superior alternative at the moment.

CRedit authorship contribution statement

Kristina Fuest: Data curation, Formal analysis, Investigation, Project administration, Writing - original draft, Writing - review & editing. **Florian Dorfhuber:** Data curation, Formal analysis, Investigation, Visualization, Writing - review & editing. **Marco Lorenz:** Investigation, Writing - review & editing. **Falk von Dincklage:** Methodology, Writing - review & editing. **Rudolf Mörgeli:** Methodology, Writing - review & editing. **Karl Friedrich Kuhn:** Methodology, Writing - review & editing. **Bettina Jungwirth:** Methodology, Project administration, Supervision, Writing - review & editing. **Karl-Georg Kanz:** Conceptualization, Methodology, Resources, Writing - review & editing. **Manfred Blobner:** Conceptualization, Data curation, Formal analysis, Methodology, Project administration, Resources, Supervi-

sion, Writing - review & editing. **Stefan J Schaller:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Supervision, Writing - review & editing.

Declaration of Competing Interest

BJ received honoraria for giving lectures from Pulsion Medical Systems SE (Feldkirchen, Germany). MB received research support not related to this manuscript from MSD (Haar, Germany), honoraria for giving lectures from GE Healthcare (Helsinki, Finland) and Grünenthal (Aachen, Germany). SJ Schaller reports grants and non-financial support from ESICM (Brussels, Belgium), Fresenius (Germany), Liberate Medical LLC (Crestwood, USA), Reactive Robotics GmbH (Munich, Germany), STIMIT AG (Nidau, Switzerland) as well as from Technical University of Munich, Germany, from national (e.g. DGAI) and international (e.g. ESICM) medical societies (or their congress organizers) in the field of anaesthesiology and intensive care, all outside the submitted work; SJS holds stocks in small amounts from Alphabeth Inc., Bayer AG, Rhön-Klinikum AG, and Siemens AG. These did not have any influence on this study. The other authors declare that they have no conflict of interest.

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Data statement

Data can be requested from the corresponding author upon reasonable request.

REFERENCES

1. Abella BS, Sandbo N, Vassilatos P, et al. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. *Circulation* 2005;111:428–34.
2. Gates S, Smith JL, Ong GJ, Brace SJ, Perkins GD. Effectiveness of the LUCAS device for mechanical chest compression after cardiac arrest: systematic review of experimental, observational and animal studies. *Heart* 2012;98:908–13.
3. Soar J, Nolan JP, Böttiger BW, et al. European Resuscitation Council Guidelines for Resuscitation 2015: Section 3. Adult advanced life support. *Resuscitation* 2015;95:100–47.
4. SOS-KANTO study group. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): an observational study. *Lancet* 2007;369:920–6.
5. Fox J, Fiechter R, Gerstl P, et al. Mechanical versus manual chest compression CPR under ground ambulance transport conditions. *Acute Card Care* 2013;15:1–6.
6. Bernhard M, Hossfeld B, Kumle B, Becker TK, Böttiger B, Birkholz T. Don't forget to ventilate during cardiopulmonary resuscitation with mechanical chest compression devices. *Eur J Anaesthesiol* 2016;33:553–6.
7. Jolife AB. LUCAS 2 Thoraxkompressions-System – Bedienungsanleitung. Lund, Sweden, 2017. URL: https://www.lucas-cpr.com/files/9398026_100901-02_Rev_B_LUCAS2_IFU_DE_LowRes.pdf
8. Jolife AB. LUCAS 3 Thoraxkompressions-System – Bedienungsanleitung. Lund, Sweden, 2018. URL: <https://www.lucas-cpr.com/files/>

- 2948406_101034-02%20Rev%20A%20LUCAS%203%20IFU%20DE_lowres.pdf.
9. Lachin JM. Worst-rank score analysis with informatively missing observations in clinical trials. *Control Clin Trials* 1999;20(5):408–22.
 10. Paal P, Neurauder A, Loedl M, et al. Effects of stomach inflation on haemodynamic and pulmonary function during cardiopulmonary resuscitation in pigs. *Resuscitation* 2009;80(3):365–71.
 11. Wenzel V, Idris AH, Banner MJ, et al. Respiratory system compliance decreases after cardiopulmonary resuscitation and stomach inflation: impact of large and small tidal volumes on calculated peak airway pressure. *Resuscitation* 1998;38(2):113–8.
 12. Buschmann CT, Tsokos M. Frequent and rare complications of resuscitation attempts. *Intens Care Med* 2009;35(3):397–404.
 13. Idris AH, Banner MJ, Wenzel V, Fuerst RS, Becker LB, Melker RJ. Ventilation caused by external chest compression is unable to sustain effective gas exchange during CPR: a comparison with mechanical ventilation. *Resuscitation* 1994;28(2):143–50.
 14. Hevesi ZG, Thrush DN, Downs JB, Smith RA. Cardiopulmonary resuscitation: effect of CPAP on gas exchange during chest compressions. *Anesthesiology* 1999;90:1078–83.
 15. Brochard L, Boussignac G, Adnot S, Bertrand C, Isabey D, Harf A. Efficacy of cardiopulmonary resuscitation using intratracheal insufflation. *Am J Respir Crit Care Med* 1996;154(5):1323–9.
 16. Saïssy J-M, Boussignac G, Cheptel E, et al. Efficacy of Continuous Insufflation of Oxygen Combined with Active Cardiac Compression–Decompression during Out-of-hospital Cardiorespiratory Arrest. *Anesthesiology* 2000;92:1523–30.
 17. Bertrand C, Hemery F, Carli P, et al. Constant flow insufflation of oxygen as the sole mode of ventilation during out-of-hospital cardiac arrest. *Intens Care Med* 2006;32:843–51.
 18. Schaller SJ, Altmann S, Unsworth A, et al. Continuous chest compressions with a simultaneous triggered ventilator in the Munich Emergency Medical Services: a case series. *Ger Med Sci* 2019;17: Doc06.
 19. Kill C, Galbas M, Neuhaus C, et al. Chest Compression Synchronized Ventilation versus Intermittent Positive Pressure Ventilation during Cardiopulmonary Resuscitation in a Pig Model. *PLoS One* 2015;10:e0127759. <https://doi.org/10.1371/journal.pone.0127759>. Available from: <https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0127759>.