

Arterial blood gas changes during cardiac arrest and cardiopulmonary resuscitation combined with passive oxygenation/ventilation: a METI HPS study

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Abstract

Objective: High-fidelity simulators can simulate physiological responses to medical interventions. The dynamics of the partial arterial pressure of oxygen (P_aO_2), partial arterial pressure of carbon dioxide (P_aCO_2), and oxygen pulse saturation (SpO_2) during simulated cardiopulmonary resuscitation (CPR) were observed and compared with the results from the literature.

Methods: Three periods of cardiac arrest were simulated using the METI Human Patient SimulatorTM (Medical Education Technologies, Inc., Sarasota, FL, USA): cardiac arrest, chest compression-only CPR, and chest compression-only CPR with continuous flow insufflation of oxygen (CFIO).

Results: In the first period, the observed values remained constant. In the second period, P_aCO_2 started to rise and peaked at 63.5 mmHg. In the CFIO period, P_aCO_2 slightly fell. P_aO_2 and SpO_2 declined only in the second period, reaching their lowest values of 44 mmHg and 70%, respectively. In the CFIO period, P_aO_2 began to rise and peaked at 614 mmHg. SpO_2 exceeded 94% after 2 minutes of CFIO.

Conclusions: The METI Human Patient SimulatorTM accurately simulated the dynamics of changes in P_aCO_2 . Use of this METI oxygenation model has some limitations because the simulated levels of P_aO_2 and SpO_2 during cardiac arrest correlate poorly with the results from published studies.

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Keywords

Patient-specific modeling, cardiac arrest, blood gas analysis, pulmonary ventilation, cardiopulmonary resuscitation, high-fidelity simulator

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Introduction

According to the current European Resuscitation Council recommendations, standard cardiopulmonary resuscitation (CPR) should be performed with a compression-to-ventilation ratio of 30:2.¹ Ventilation during the early phase of CPR has been questioned and reevaluated.² Adverse consequences from excessive positive-pressure ventilation could result in increased intrathoracic pressure³ and decreased coronary perfusion pressure, thereby worsening patient outcomes.⁴ The fact that both laypersons⁵ and health professionals⁶ are reluctant to perform mouth-to-mouth ventilation has also contributed to the re-evaluation of ventilation in the early phase of CPR. As a result, the current European Resuscitation Council guidelines encourage CPR providers who witness sudden adult collapse to perform continuous chest compression-only CPR without mouth-to-mouth ventilation.⁷ An alternative approach to oxygenation during CPR is continuous flow insufflation of oxygen (CFIO) through a Boussignac tube,⁸⁻¹⁰ nonrebreather facemask (NRB),¹¹ or nasal oxygen tube.¹² Some studies have shown that CFIO is more effective¹¹ or equally as effective as the recommended intermittent positive-pressure ventilation with respect to outcomes after resuscitation^{10,12} and yields better oxygenation⁹ and coronary perfusion pressure during resuscitation efforts.⁸

The METI Human Patient Simulator™ (METI HPS) (Medical Education Technologies, Inc., Sarasota, FL, USA) is a sophisticated mannequin with integrated pathophysiological models that can reproduce different clinical scenarios and simulate physiological responses to medical interventions while giving real-time feedback. The METI HPS is currently used as an educational tool for nurses,¹³ medical students,^{14,15} and medical providers.¹⁶ It has also been used as an experimental tool to simulate bodily responses to extreme environments such as carbon monoxide poisoning during occupational mining accidents.¹⁷ High-fidelity simulators were designed to test medical devices and interventions, especially in emergency situations,^{18,19} before their translation into clinical research or clinical practice, allowing safer clinical human studies and clinical practice. It can be concluded that the METI HPS accurately reproduces real clinical situations. The accuracy of the METI physiological model during oxygen administration and apnea maneuvers was recently investigated.²⁰ The study showed some discrepancies between the obtained data and the results from the literature.

The purpose of this study was to quantitatively observe the dynamics of changes in the METI HPS oxygenation model (changes in arterial blood gas and oxygen pulse saturation) when performing chest compression-only CPR or a combination of chest compression-only CPR and CFIO.

Materials and methods

A patient was simulated using the version 6 METI HPS in the Simulation Center of the Medical Faculty, University of Maribor (Maribor, Slovenia). This full-scale, high-fidelity simulator uses a hybrid (mathematical and mechanical) self-regulating lung model with a real physical system to model pulmonary gas exchange and lung mechanics of a simulated patient. Briefly, uptake and excretion of oxygen, carbon dioxide, nitrous oxide, and a volatile anesthetic were physically created based on the measured concentrations in the bellows of the simulated lung and in a software model representing uptake, distribution, storage, consumption, and/or production in the body. Lung perfusion was also accounted for in this model by modeling the cardiovascular subsystem of the patient being simulated.²⁰ Unlike this hybrid lung model, all other simulator models, such as cardiovascular (blood pressure) and systemic uptake and distribution (arterial blood gas) models, are mathematical models.

Simulation of cardiac arrest and chest compressions were performed as follows. Initially, cardiac arrest was simulated for 10 minutes (cardiac arrest period). The cardiac rhythm of the METI HPS was set as asystole to simulate cardiac arrest during this period. After the first period, resuscitation efforts (chest compression-only CPR period) were simulated for the next 10 minutes. This required minor changes in the software of the METI HPS to simulate the second period. The tidal volume was set to the lowest possible value of 200 mL with a breathing frequency of 40/minute (highest possible) to simulate passive ventilation/oxygenation achieved by chest compression-only CPR. Our goal was to set the breathing frequency as close as possible to the chest compression rate during CPR. Because of the software limitation, the highest possible value was chosen.

The respiratory quotient was set at 0.8 (oxygen consumption of 250 mL/min and carbon dioxide production of 200 mL/min) for a standard adult (body weight of 70 kg) and remained constant during the simulation. Cardiac output was set at 25% of the normal value with a heart rate of 100/minute (the cardiac rhythm was set at sinus rhythm) to simulate the cardiac output achieved by chest compression-only CPR. These values of tidal volume²¹ and cardiac output achieved during chest compressions have already been confirmed in previously published studies.^{22,23} After the initial 20 minutes, the third 10-minute period (CFIO period) of chest compression-only CPR with CFIO followed the first and second periods. During the third period, oxygen was applied to the simulator via a nasal cannula (NC), NRB, or combination of the two to perform CFIO. The flow of oxygen was set at 15 L/minute in the third period. The partial arterial pressure of oxygen (P_aO_2) and partial arterial pressure of carbon dioxide (P_aCO_2) were continuously measured by the METI HPS respiratory gas analyzer module during the simulation and recorded in a data log file for each procedure. The oxygen pulse saturation (SpO_2) was simulated with the data from the model and was also continuously recorded in a data log file. The obtained data were compared with the results from the literature.

This study was conducted in a simulation center using only a METI HPS high-fidelity simulator. No human or animal subject was included in the study. Therefore, approval from an ethics committee was not needed.

Results

The dynamics of the changes in P_aCO_2 are presented in Figure 1. For the first 10 minutes, P_aCO_2 remained similarly constant (42 mmHg). After the initial 10 minutes (chest compression-only CPR),

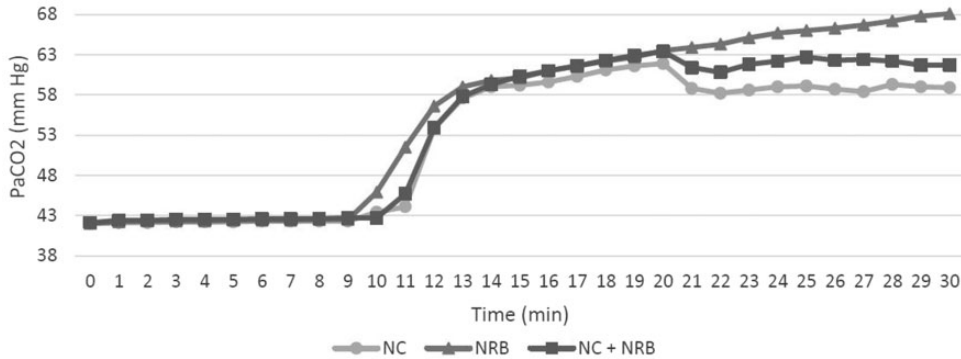


Figure 1. Dynamics of changes in P_aCO_2 .

Abbreviations: P_aCO_2 , partial arterial pressure of carbon dioxide; mmHg, millimeters of mercury; min, minute; NC, nasal cannula; NRB, nonrebreather facemask.

Table 1. Differences in partial pressure of carbon dioxide (ΔpCO_2).

Study period	Cardiac arrest		Chest compression-only CPR		CFIO period		
	5th	8th	9th	10th	2nd	4th	6th
ΔpCO_2 from compared studies (mmHg)	+0.0*	+2.2 [†]	+22.0*	+11.8 [‡]	+25.0 [¶]	+27.0 [¶]	+15.0 [¶]
ΔpCO_2 from METI HPS study (mmHg)	+0.2	+0.3	+17.0	+19.9	+16.2	+17.0	+16.7

Abbreviations: ΔpCO_2 , difference in partial pressure of carbon dioxide from baseline level; CPR, cardiopulmonary resuscitation; CFIO, continuous flow insufflation of oxygen; mmHg, millimeters of mercury; METI HPS, Medical Education Technologies Inc. Human Patient Simulator. *Study by Idris et al.²⁴; [†]Study by Steen et al.⁸; [‡]Study by Chandra et al.²⁵; [¶]Study by Hayes et al.¹² Values of ΔpCO_2 from the present METI HPS study were taken from the nasal cannula oxygen application, which was also used by Hayes et al.¹² in the CFIO period.

P_aCO_2 began to rise and reached its highest value of 63.5 mmHg at the 20-minute mark. At the beginning of the CFIO period, P_aCO_2 began to slightly decline, reaching 58.9 mmHg using the NC and 61.7 mmHg using the NRB at the end of the CFIO period. P_aCO_2 continued to rise using the NC + NRB after the 20-minute mark and reached its highest level of 68.1 mmHg at the end of the experiment.

The differences in pCO_2 from the baseline levels at different time points of the present METI HPS study and various studies from the literature are presented in Table 1.

The changes in P_aO_2 are shown in Figure 2. For the first 10 minutes, P_aO_2 remained constant (121 mmHg). From the 10-minute mark, it began to decline, reaching its lowest value of 44 mmHg at the 20-minute mark. In the CFIO period, P_aO_2 began to rise and reached its highest value of 395 mmHg using the NC, 477 mmHg using the NRB, and 614 mmHg using the NC + NRB at the end of the experiment.

The differences in pO_2 from the baseline levels at the different time points of the present METI HPS study and various studies from the literature are presented in Table 2.

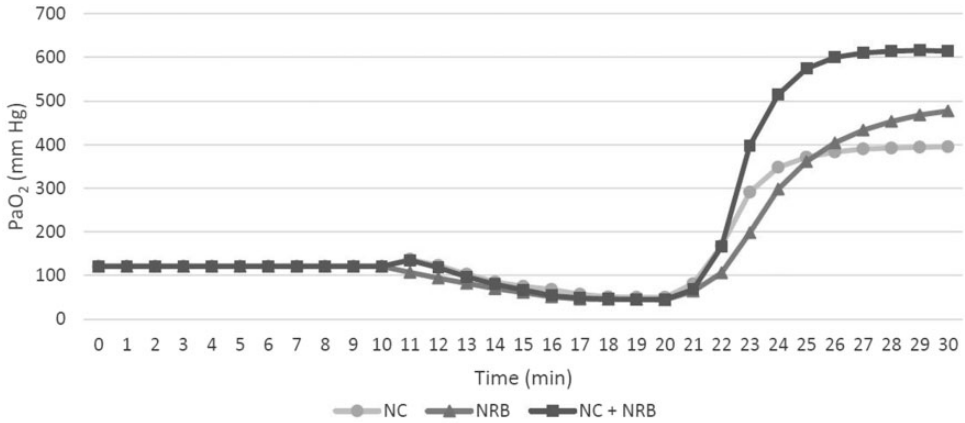


Figure 2. Dynamics of changes in PaO₂.

Abbreviations: PaO₂, partial arterial pressure of oxygen; mmHg, millimeters of mercury; min, minute; NC, nasal cannula; NRB, nonrebreather facemask.

Table 2. Differences in partial pressure of oxygen (ΔpO_2).

Study period	Cardiac arrest		Chest compression-only CPR				CFIO period				
	7th	8th	2nd	4th	6th	8th	10th	2nd	4th	6th	10th
ΔpO_2 from compared studies (mmHg)	-4.0 [¶]	-3.7 [†]	-7.3 [‡]	-13.1 [‡]	-24.6 [‡]	-51.6 [‡]	-66.9 [‡]	+11.0 [¶]	+17.0 [¶]	+21.0 [¶]	+306.0 [†]
ΔpO_2 from METI HPS study (mmHg)	+0.0	+0.0	+2.0	-36.0	-53.0	-70.0	-71.0	+47.0	+227.0	+262.0	+274.0

Abbreviations: ΔpO_2 , difference in partial pressure of oxygen from baseline level; CPR, cardiopulmonary resuscitation; CFIO, continuous flow insufflation of oxygen; mmHg, millimeters of mercury; METI HPS, Medical Education Technologies Inc. Human Patient Simulator. [†]Study by Steen et al.⁸; [‡]Study by Chandra et al.²⁵; [¶]Study by Hayes et al.¹² Values of ΔpO_2 from the present METI HPS study were taken from the nasal cannula oxygen application, which was also used by Hayes et al.¹² in the CFIO period.

The changes in S_pO₂ are presented in Figure 3. After constant levels in the initial period, S_pO₂ remained unchanged until the 14- and 15-minute marks, when the S_pO₂ value declined to <94% and <90%, respectively. The S_pO₂ continued to decline during the chest compression-only CPR period and reached its lowest value of 70% at the end of this period. Already 2 minutes after starting CFIO, the S_pO₂ increased to >94%; it then increased to the maximal value of 100% after 4 minutes of CFIO regardless of the

method of oxygen application and remained at this level until the end of the experiment.

The dynamics of the mean arterial pressure are presented in Figure 4. The changes in the mean arterial pressure were similar in all three scenarios. The pressure fell after 2 minutes of cardiac arrest to the lowest level of 17 mmHg and started to rise only after 2 minutes of chest compressions to the maximal level of approximately 50 mmHg; it then remained constant until the end of the experiment.

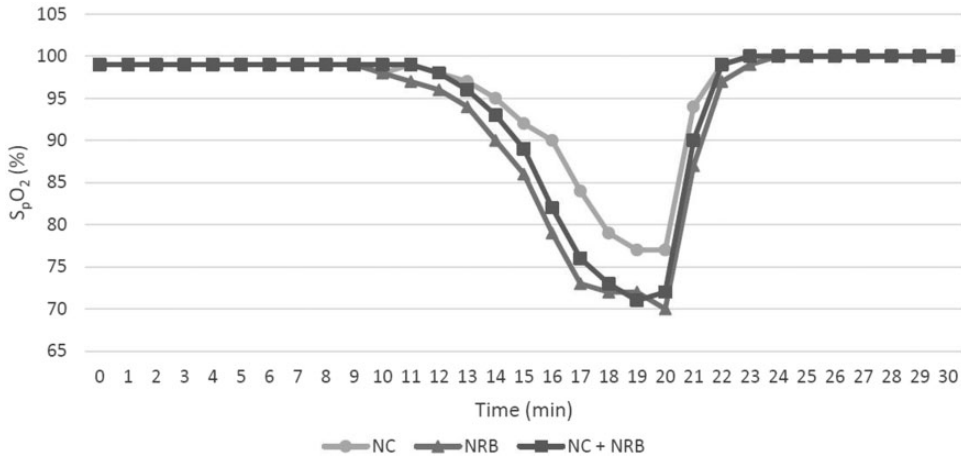


Figure 3. Dynamics of changes in SpO₂.
Abbreviations: SpO₂, oxygen pulse saturation; min, minute; NC, nasal cannula; NRB, nonre-breather facemask.

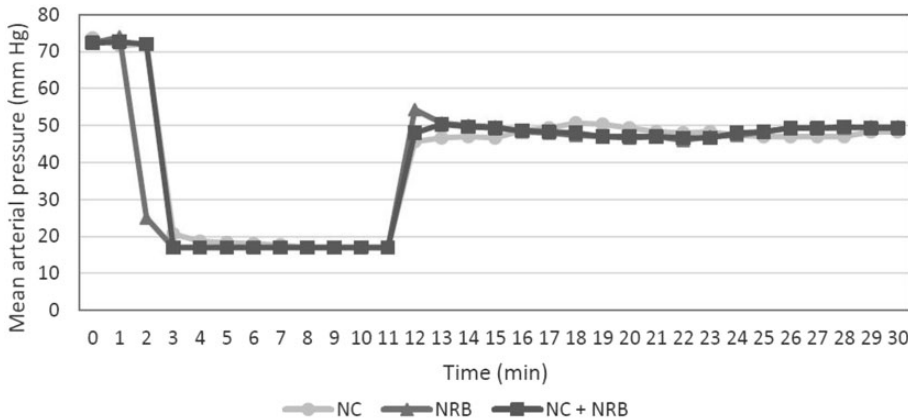


Figure 4. Dynamics of changes in MAP.
Abbreviations: MAP, mean arterial pressure; mmHg, millimeters of mercury; min, minute; NC, nasal cannula; NRB, nonrebreather facemask.

Discussion

The dynamics of changes in arterial blood gas parameters and oxygen pulse saturation during cardiac arrest and resuscitation efforts followed by chest compression-only CPR combined with CFIO were simulated using the METI HPS, and the data were compared with the results reported in the literature.

In the present study, PaCO₂ did not change during the cardiac arrest period. This observation is in accordance with those reported by Steen et al.⁸ and Idris et al.,²⁴ who found no significant difference in PaCO₂ between baseline and after 8 minutes of cardiac arrest and between baseline and after 5 minutes of cardiac arrest, respectively. PaCO₂ started to rise

when chest compressions were performed. This could have been the result of the increased delivery of carbon dioxide from peripheral tissue into the cardiocirculatory system during chest compressions. The pattern of changes in $P_a\text{CO}_2$ during the chest compression-only CPR period of the present study is in accordance with previously published animal studies.^{25,26} However, Chandra et al.²⁵ and Berg et al.²⁷ reported lower values of $P_a\text{CO}_2$ in animal studies (38.8 ± 6.4 mmHg after 10 min and 41 ± 12 mmHg after 4 min of chest compression-only CPR). This difference may be due to the lower baseline (prearrest) values of $P_a\text{CO}_2$ in canines (27 ± 1.5 mmHg)²⁵ and shorter period of cardiac arrest (2 min)^{25,27} compared with our study. Dorph et al.²⁶ observed significantly higher levels of carbon dioxide (92.5 mmHg after 9 minutes of chest compression-only CPR) in pigs with obstructed airway, which prevented passive ventilation and resulted in higher values of $P_a\text{CO}_2$.

During the CFIO period, we observed two different patterns of $P_a\text{CO}_2$ changes. CFIO using an NC or a combination of an NC and NRB resulted in slightly decreased values of $P_a\text{CO}_2$ compared with the previous period. Similar values of $P_a\text{CO}_2$ were reported by Hayes et al.¹² (57 ± 9 mmHg after 6 min of CPR with CFIO). It could be presumed that CFIO together with chest compressions produces passive ventilation and thus eliminates carbon dioxide. Branditz et al.²⁸ showed that external cardiac chest compressions combined with CFIO generate adequate ventilation, while CFIO generates positive pressure in the lungs. In contrast, when oxygen was applied using the NRB in the present study, an additional rise of $P_a\text{CO}_2$ was observed, with the highest value being achieved at the end of the CFIO period. It seems that the increased values of $P_a\text{CO}_2$ in our study could be attributed to insufficient ventilation. This finding is supported by an

animal study by Idris et al.,²⁴ who detected similar values of $P_a\text{CO}_2$ (62 ± 16 mmHg) in the nonventilated group of domestic swine during chest compressions. An inadequate facemask seal, which is a common problem observed with the METI HPS, is considered to be a factor that contributed to insufficient ventilation in our study. The inadequate seal may have led to less pressure in the lungs and thus less effective passive ventilation, resulting in the accumulation of carbon dioxide in the lungs.

We set the tidal volume on the METI HPS at the lowest possible value (200 mL) and the highest possible breathing frequency (40/min) to simulate passive ventilation achieved by chest compressions. Owing to the simulator limitations, we could not set the breathing frequency closer to the recommended rate of chest compressions (i.e., 100–120/min). The tidal volume was similar to that reported by Safar et al.,²¹ who showed that in anesthetized patients with an open airway, rhythmic firm pressure over the lower half of the sternum at a rate of one compression per second generates an average tidal volume of 156 mL. In more than half of the patients, the tidal volume was larger than the estimated dead space, presuming effective passive ventilation occurred in those patients.²¹

Steen et al.⁸ also demonstrated that CFIO during mechanical chest compression-active decompression CPR provided adequate ventilation. The airway pressure induced by CFIO was positive during the entire cycle of CPR, thus increasing the functional residual capacity and decreasing physiological dead space. Saissy et al.¹⁰ found significantly greater elimination of carbon dioxide in the CFIO group because of better lung mechanics in this group and concluded that CFIO through a multichannel open tube was as effective as intermittent positive-pressure ventilation during out-of-hospital arrest. Additionally, animal studies have demonstrated large

minute volumes generated by chest compressions alone in dogs²⁵ or through a combination of precordial compression and gasping in pigs.²⁹

Deakin et al.³⁰ reported that passive ventilation occurring as a result of compression-only CPR in humans appears to be ineffective in generating tidal volumes adequate for gas exchange. They found that in all patients, the passive tidal volume was significantly lower than the patients' estimated dead space.³⁰ This finding could be the consequence of reduced respiratory system compliance because the measurement in that study was made 40 to 50 minutes post-arrest, when the respiratory compliance had already decreased due to pulmonary edema and venous congestion; in contrast, measurements were made a few minutes post-cardiac arrest in the above-mentioned previous studies. Deakin et al.³⁰ also found sustained levels of end-tidal carbon dioxide in most patients during compression-only CPR, suggesting that alveolar gas exchange was occurring despite the low passive tidal volumes measured.

Oxygenation is the aim of emergency ventilation. Therefore, CFIO was introduced as a new approach in resuscitation. Our study showed that P_aO_2 did not change during the cardiac arrest period. The P_aO_2 during this period is expected to decline due to utilization of oxygen in peripheral tissues for ongoing metabolic processes. A decline in P_aO_2 during cardiac arrest was observed by Hayes et al.¹² and Idris et al.,²⁴ who found a decrease in P_aO_2 between baseline and after 7 minutes of cardiac arrest and between baseline and after 5 minutes of cardiac arrest, respectively. In the present study, P_aO_2 started to fall only after the beginning of the chest compression period. The same decline in P_aO_2 was described by Chandra et al.²⁵ with a similar value of P_aO_2 (40.9 ± 7.5 mmHg) 10 minutes after chest compression-only CPR.

After starting CFIO combined with chest compressions, increasing P_aO_2 values were observed. The highest value was noted after 10 minutes of combined application of oxygen (NC + NRB) during chest compressions. This type of combined application of oxygen is commonly used to prevent desaturation during emergency airway management.³¹ Administration of oxygen using an NC is as effective as using an NRB but reaches a lower maximal P_aO_2 value. The PaO_2 values obtained in the present study are higher than those reported by Hayes et al.¹² and are in accordance with those in the animal study conducted by Steen et al.,⁸ who showed significantly higher average PaO_2 values in the CFIO group during 30 minutes of mechanical CPR than in the intermittent positive-pressure ventilation group.

Steen et al.⁸ used an endotracheal Boussignac tube, which was developed for oxygen administration in the distal trachea through five or eight capillaries molded into the tubing wall and an opening in the main lumen 2 cm above the distal end of the tube. Although the P_aO_2 values in both studies were similar, oxygen delivery using a Boussignac tube could be more effective in real-life situations than that using an NC or NRB, for which distal delivery of oxygen depends on a patent airway.

In the present study, the S_pO_2 did not change during the cardiac arrest period. In contrast, Steen et al.⁸ noted a significant fall of S_pO_2 (to $86\% \pm 2\%$) after an 8-minute-long cardiac arrest period. A delayed fall in S_pO_2 was observed in our experiment starting in the chest compression-only CPR period. This finding is in accordance with that by Lejus et al.,²⁰ who described a delayed decrease in S_pO_2 during apnea on the METI HPS in comparison with a clinical situation. Both the shape and values of the S_pO_2 curve (desaturating part of curve) in the present study are similar to those reported by Chandra et al.,²⁵ who examined

the S_pO_2 values in canines (93.9 ± 2.8 at 4 min and 70.4 ± 10.6 at 10 min) after chest compression-only resuscitation. In contrast, significantly lower S_pO_2 values were observed (6 ± 4 at 3 min and 4 ± 1 at 9 min) in pigs resuscitated with only chest compressions, also resulting in a steeper decrease in the desaturation part of the S_pO_2 curve.²⁶ The observed difference in the S_pO_2 values can be explained by the study design. Swine in the previous study had an obstructed airway that prevented potential passive ventilation during chest compression-only CPR, resulting in lower S_pO_2 values. Berg et al.²⁷ noted an earlier fall in S_pO_2 to 77% after 4 minutes in swine subjected to chest compression CPR. After administration of oxygen, S_pO_2 reached a normal value ($>94\%$) after 2 minutes of CFIO in all three oxygen application methods. This finding is similar to that reported by Steen et al.,⁸ who observed constant S_pO_2 values during mechanical CPR in the CFIO group.

Bertrand et al.⁹ reported a higher detectable pulse saturation and higher proportion of patients with a peripheral arterial oxygen saturation of $>70\%$ among patients treated with CFIO. Although the peripheral arterial oxygen saturation is commonly considered to be unreliable in low-flow states, the possibility of detecting it may be increased by improved peripheral circulation and oxygenation.

A significantly higher coronary perfusion pressure when using mechanical CPR combined with CFIO has also been reported,⁸ although this observation did not result in better patient survival. No differences in return of spontaneous circulation,¹⁰ hospital admission, or intensive care unit admission were noted in patients treated with CFIO during CPR versus patients who were mechanically ventilated.⁹ The animal study by Hayes et al.¹² also showed no significant difference in the neurological outcome between the different ventilation protocols. In contrast, among adults with

witnessed out-of-hospital cardiac arrest and ventricular fibrillation/ventricular tachycardia as the initial recorded rhythm, the neurologically intact survival rate was higher for individuals who received CFIO.¹¹ Although we did not analyze different survival outcomes because of the nature of the preset study, findings from the above-mentioned study suggest that patients receiving minimally interrupted cardiac resuscitation are more likely to survive. Advanced airway management can be time-consuming and may disrupt CPR chest compression continuity.

Our study has several limitations. First, instead of manual or mechanical chest compressions, the cardiac output was set at 25% of the normal value and kept constant during the chest compression-only CPR and CFIO periods. The efficiency of chest compressions during resuscitation declines due to fatigue of healthcare professionals,³² resulting in lower cardiac output achieved by resuscitation efforts. In addition, the pathophysiological changes in the myocardium during cardiac arrest lead to a decrease in myocardial compliance, preventing hemodynamically effective chest compressions.³³ These changes probably result in different arterial gas values in clinical practice. Second, the airway of the METI HPS was always patent during the study. The airway in cardiac arrest victims is usually closed and should be opened with the airway adjunct to perform CFIO. The METI HPS has been shown to be the most realistic patient simulator among other simulators with respect to airway anatomy.³⁴ Therefore, the values observed in the present study could differ from those in a study using another high-fidelity simulator. Third, the METI HPS was manipulated to respond to extreme case simulation; therefore, the simulated response may not be physiologically accurate. Owing to these limitations, translation of the results from this study to clinical practice should be

done with caution. Another limitation of the present study is that the simulation was limited to only one session for each intervention without repetition. During the first two periods of the study, minor variance in variables was observed, which led to the assumption that further series of experiments would not contribute to the accuracy of the study. This assumption is supported by Cumin et al.,³⁵ who found minor divergence between time series generated with the METI HPS.

In conclusion, the METI HPS was proven to be a suitable experimental tool to accurately simulate the dynamics of changes in P_aCO_2 . Use of the METI HPS for simulation of oxygenation changes during cardiac arrest has some limitations because the simulated P_aO_2 levels during cardiac arrest correlate poorly with the results from published studies. Our study confirmed the delayed decrease in S_pO_2 already described in the literature. To our knowledge, this is the first study to address the dynamics of arterial blood gas changes during cardiac arrest, resuscitation efforts, and CFIO combined with CPR in a simulated scenario with the METI HPS. Findings from this study show that both transferring the conclusions from METI HPS studies into the clinical environment and testing new equipment on the METI HPS should be done with caution. Further studies are needed to confirm the conclusions of the present study.

Data availability

All data underlying the findings of the study are published in the article.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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