Impact of ventilation strategies during chest compression. An experimental study with clinical observations

Ricardo L. Cordioli,1,2,3 Assiam Lyazidi,1,4,5 Nathalie Rey,6 Jean-Max Granier,1 Dominique Savary,7 Laurent Brochard,8,9,10 and Jean-Christophe M. Richard7,10

1University Hospital of Geneva, Intensive Care Unit, Geneva, Switzerland; 2Intensive Care Unit, Hospital Israelita Albert Einstein, São Paulo, Brazil; 3Intensive Care Unit, Hospital Alemão Oswaldo Cruz, São Paulo, Brazil; 4Laboratoire Rayonnement-Matière et Instrumentation, Département de Physique, Université Hassan Ier, Settat, Morocco; 5Institut Supérieur des Sciences de la Santé, Université Hassan Ier, Settat, Morocco; 6Department of Anesthesiology and Intensive Care Unit, Rouen, France; 7Emergency and Intensive Care Department, General Hospital of Annecy, Annecy, France; 8Keenan Research Centre, St. Michael’s Hospital, Toronto, Ontario, Canada; 9Interdepartmental Division of Critical Care Medicine, University of Toronto, Toronto, Ontario, Canada; 10INSERM UMR 955, Creteil, France

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Cordioli RL, Lyazidi A, Rey N, Granier J, Savary D, Brochard L, Richard JM. Impact of ventilation strategies during chest compression. An experimental study with clinical observations. J Appl Physiol 120: 196–203, 2016. First published November 19, 2015; doi:10.1152/japplphysiol.00632.2015.—The optimal ventilation strategy during cardiopulmonary resuscitation (CPR) is unknown. Chest compression (CC) generates circulation, while during decompression, thoracic recoil generates negative pressure and venous return. Continuous flow insufflation of oxygen (CFI) allows noninterrupted CC and generates positive airway pressure (Paw). The main objective of this study was to assess the effects of positive Paw compared with the current recommended ventilation strategy on intrathoracic pressure (PIT) variations, ventilation, and lung volume. In a mechanical model, allowing compression of the thorax below an equilibrium volume mimicking functional residual capacity (FRC), CC alone or with manual bag ventilation were compared with two levels of Paw with CFI. Lung volume change below FRC at the end of decompression and PTR, as well as estimated alveolar ventilation, were measured during the bench study. Recordings were obtained in five cardiac arrest patients to confirm the bench findings. Lung volume was continuously below FRC, and as a consequence PTR remained negative during decompression in all situations, including with positive Paw. Compared with manual bag or CC alone, CFI with positive Paw limited the fall in lung volume and resulted in larger positive and negative PTR variations. Positive Paw with CFI significantly augmented ventilation induced by CC. Recordings in patients confirmed a major loss of lung volume below FRC during CPR, even with positive Paw. Compared with manual bag ventilation, positive Paw associated with CFI limits the loss in lung volume, enhances CC-induced positive PTR, maintains negative PTR during decompression, and generates more alveolar ventilation.

cardiopulmonary resuscitation; ventilation; chest compression; continuous flow insufflation; lung volume

THE IMPORTANCE of high-quality chest compression (CC) during cardiopulmonary resuscitation (CPR) has been repeatedly highlighted. The optimal ventilation to be delivered has been debated and its place in international guidelines has been modified to better acknowledge the risks of hyperventilation or of interrupting compressions (4, 5, 35a, 25, 29, 36). The last recommendations for advanced cardiac life support from the American Heart Association, European Resuscitation Council Guidelines for Resuscitation, and International Liaison Committee on Resuscitation (13, 14, 30) emphasize that ventilation becomes crucial when CPR is prolonged. Ventilation during CPR is expected to allow adequate carbon dioxide removal and provide sufficient arterial oxygen content, while minimizing the risk of impairing circulation. Oxygenation is key at the very beginning of CPR, whereas CO2 clearance becomes more important at a later phase.

Continuous flow insufflation (CFI) of oxygen is acknowledged as a promising alternative technique. Delivered with small levels of positive pressure, CFI has shown beneficial effects in animal studies and some advantages in clinical trials (3, 7, 8, 18, 35, 37). It seems essential, however, to assess the real changes in intrathoracic pressure (PIT) when using CFI because positive pressure could impact negatively on venous return (22). Venous return is mainly generated by intrathoracic negative pressure resulting from the recoil forces of the thorax during decompression. The amplitude of negative PIT is mostly influenced by the return of lung volume to functional residual capacity (FRC) during decompression. To investigate the PIT and thoracic lung volumes associated with positive airway pressure (Paw) during CPR, we reasoned than a realistic thoracic lung model allowing the start of CC from FRC was needed. This in vitro study aimed at accurately assessing the effects of positive Paw and CFI compared with the current recommended ventilation strategies (13, 14, 30) on PIT variations, lung volume, and ventilation. Furthermore, recordings from mechanically ventilated patients during CPR were used to confirm our in vitro findings.

METHODS

Thoracic Lung Model

An original mechanical thoracic lung model was designed to reproduce the mechanical properties of the thoracic compartment during CPR, and was specifically created to allow CC (Poumon test pour l’arrêt cardiaque, license in process).

This thoracic lung model was made of a monocompartment bellow with an internal volume mimicking the FRC at the equilibrium state (static condition) (Fig. 1). A spring and two 60-ml syringes filled with an adjustable amount of compressible air acted in opposite directions, simulating the thoracic elastic properties above and below FRC, and also allowing adjustment of the mechanical proprieties of the thoracic

Address for reprint requests and other correspondence: R. L. Cordioli, Dept. of Adult Intensive Care, Hospital Israelita Albert Einstein, Ave. Albert Einstein, 627, Morumbi, São Paulo - SP, 05652-900, Brazil (e-mail: rlcorvidoli@gmail.com or ricardolc@einstein.br).

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model. The model permitted generation of CC starting from FRC, thus simulating a nonbreathing patient with cardiac arrest, and reproducing the elastic recoil properties of the thorax during the phases of decompression. A compressible conducting connector, creating a Starling resistor, was added to reproduce airway collapse that may affect the airways when Paw becomes much lower than the surrounding pressure, especially at low lung volumes.

A respiratory system with a resistance of 10 cm H₂O-liter⁻¹·s⁻¹, a compliance of 15 ml/cm of H₂O, and an FRC of 1,600 ml was used. Compliance values have been reported in various ranges in patients during cardiac arrest (12, 31). We used a low value of compliance in our lung model for the following reasons. The shape of the pressure volume curve of the respiratory system indicates that compliance rapidly falls below FRC. Considering the lung volume loss that we observed, we reasoned that the expected compliance during chest compression would be markedly lower than the one measured at FRC, as reported in the literature in patients after cardiac arrest. We calculated that a value of 15 ml/cmH₂O during CPR could be realistic if one starts from a value of 40 ml/cmH₂O at FRC. In addition, lung compliance tends to decline as CPR is prolonged (12), reinforcing the idea that compliance during CPR may become extremely reduced.

To standardize CC, a mechanical device (LUCAS 2, Jolife AB/Physio-Control, Lund, Sweden) was used. By compressing the model in a reproducible way, this setup offered the opportunity to evaluate different ventilation strategies under similar conditions of CC. Compression induced a positive $P_{TR}$ inside the thoracic model, while passive decompression generated a negative $P_{TR}$ due to the elastic properties of the thoracic lung model. During CC, the dynamic changes of the bellow, mimicking the thoracic lung system relative to initial FRC in static condition were recorded on millimeter grid graph paper (Fig. 2).

**Measurements and Evaluation Criteria**

A pressure transducer (MP45 $±$ 100 cm H₂O; Validyne, Northridge, CA) connected into the thoracic model was used to measure $P_{TR}$. The mean positive intrathoracic pressure ($P_{TR\text{ (positive)}}$), generated by chest compression, and the mean negative intrathoracic pressure ($P_{TR\text{ (negative)}}$), generated by chest decompression, were analyzed for each CC. Dynamic thoracic volume change was calculated as the difference between the FRC and the lung position at the end of decompression (Fig. 2). Using the compliance of our model, we calculated the relation between the position change of the model registered in the millimeter paper and the dynamic thoracic volume variation.

Airflow was measured with a pneumotachograph (maximum flow 8 liters/s; Fleisch n°3, Lausanne, Switzerland) placed at the external extremity of the endotracheal tube connected to the lung model.

Mobilized volume by each CC cycle was calculated by integrating flow-time trace. The positive flow related to the application of CFI was subtracted while integrating the flow-time curve to calculate the real mobilized volume. The volume mobilized was often small and presumably lower than physiological dead space, and therefore unlikely to generate efficient ventilation. To estimate what would be the minute alveolar ventilation due to CC during CPR, an estimated physiological dead space value was thus subtracted from the volume mobilized. A realistic value of 200 ml was used (15, 16).

Pressures and flow were recorded online and subsequently analyzed with AcqKnowledge software (AcqKnowledge 3.7.3, Biopac systems, Goleta, CA). For each condition, 1 min (representing 100 CC) of recording was analyzed after stabilization of thoracic position on the millimeter grid paper.

**Bench Protocol**

CC was performed with LUCAS 2 set at a rate of 100/min with an equal compression/decompression time. The device promoted a compression depth of 4 to 5 cm from the starting position. CC was never interrupted during the different CPR strategies according to the international recommendations.

Four different ventilation strategies were tested during CC to simulate CPR. 1) CC alone without assisted ventilation or CFI. 2) CC with manual bag ventilation at a rate of 10/min performed by three experienced physicians with no attempt to synchronize with CC, during 2 min, following the international recommendation of CPR for an intubated patient (the average result obtained from these three physicians was used). 3) CC with CFI of oxygen using a specific endotracheal tube designed for CPR (Boussignac CPR system, Vygon, Ecouen, France) this endotracheal tube contains capillaries molded into the tubing wall allowing high velocity gas injection, generating turbulences creating a positive pressure at the tip of the tube (24) (also referred to as a “virtual valve”). Oxygen flow rate was set to obtain at the airway opening a pressure of 5 cm H₂O. This pressure was measured before starting CC. 4) CC with CFI of oxygen set with an initial airway opening pressure of 10 cm H₂O.

**Clinical Observations**

In patients with out-of-hospital cardiac arrest receiving CPR with an automatic noninvasive cardiac support pump (AutoPulse Zoll, Chelmsford, MA) and ventilated with a turbine based ventilator (Monnal T60 Air Liquide Medical Systems, Antony France), airway pressures, flow generated by CC, and mechanical insufflations were recorded via the ventilator and subsequently analyzed with the AcqKnowledge software. Lung volumes during CC were calculated by integrating flow-time traces when reaching an equilibrium lung vol-
ume after CC interruption for ECG analysis. The Monnal T60 ventilator allows reliable measurement of flow and pressure in the settings of CPR, as assessed independently on the bench test during CPR (data not shown). The ventilator was set in volume controlled mode with default settings of tidal volume (450 ml) and respiratory rate (10/min), compatible with international recommendations. The positive end-expiratory pressure (PEEP) was set by the physician from 0 to 5 cm H2O. These recordings were performed in the hospital of Annecy as part of the French registry of cardiac arrest (Réseau Nord Alpin des Urgences RENAU, http://www.renau.org) after obtaining approval from the local Research Ethics Committee (No. 5891).

Statistical Analysis

Statistical analysis was performed with Sigma Plot 12.0 statistical package software (Systat Software, San Jose, CA). The Shapiro-Wilk goodness-of-fit model showed a parametrical distribution for all variables, and data are reported as means. Standard deviation was minimal in the bench tests because of standardization of CC and is not represented. We used a multivariate analysis of variance to compare the different CPR strategies. When the overall comparison was significant, two-by-two post hoc comparisons were made using a Tukey’s test. A P value less than 0.05 was considered statistically significant.

RESULTS

Bench Study

Whatever the situation tested, CC always resulted in high positive PIT during compression and negative PIT during decompression, as well as end-compression and end-decompression positions of lung volume well below FRC. CC mobilized different gas volume according to the ventilation strategy adopted.

CC alone. Among the four strategies studied, CC alone produced the lowest mean negative PIT (−55 mmHg) and mean positive PIT (+13 mmHg) (Fig. 3), and produced the most marked reduction in dynamic lung volume at the end of decompression (−352 ml) (Fig. 4). CC alone mobilized an average gas volume of 100 ml at each CC, lower than the estimated dead space, and therefore without generating any significant estimated alveolar ventilation.

CC and manual bag ventilation. Compared with CC alone, the addition of manual bag insufflations generated a mean negative PIT (−44 mmHg) and a mean positive PIT (+ 19 mmHg) significantly higher (Fig. 3) than CC alone and a
dynamic lung volume reduction (~304 ml) significantly lower than CC alone (Fig. 4). The average tidal volume promoted by each bag mask insufflation performed during the CPR done by the three physicians was 680 ± 43 ml. Considering a physiological dead space of 200 ml, we estimated that 480 ml (~43 ml) of alveolar ventilation occurred during each manual bag insufflation. The CC not combined with a manual bag insufflation mobilized volumes lower than the estimated dead space volume (Fig. 4).

CC during CFI of 5 and 10 cm H₂O. The presence of CFI combined with CC significantly modified the PIT behavior, the thoracic volume reduction, and the estimated alveolar ventilation compared with the other two CPR techniques tested (Figs. 3 and 4).

During CFI of 5 cm H₂O the P_{IT}^\text{negative} (−39 mmHg) and the P_{IT}^\text{positive} (+39 mmHg) were significantly higher than the results observed during CC alone or CC with manual bag ventilation (Figs. 3 and 4), and the dynamic lung volume reduction was statistically lower (~240 ml). The estimated alveolar ventilation was 7.5 liters/min.

During CFI of 10 cm H₂O, the P_{IT}^\text{negative}, the P_{IT}^\text{positive}, and the reduction in thoracic volume were ~32 mmHg, +45 mmHg, −224 ml, respectively. P_{IT} remained negative during decompression even using CFI of 10 cm H₂O (Fig. 4). The increment in initial Paw from 5 to 10 cm H₂O further increased the difference with the other CPR strategies tested (Figs. 3 and 4). The estimated alveolar ventilation further increased at 13.4 liters/min.

Clinical Observations

Brief periods (up to 5 min) of Paw and flow vs. time recordings were obtained during CPR in five patients with out-hospital nontraumatic cardiorespiratory arrest of varying etiologies and mechanically ventilated (age 67 ± 4 yr). The brief CC interruptions performed to check the electrocardiographic rhythm allowed us to observe the relative changes in lung volume related to CC. Tracings confirmed a systematic increase in lung volume when CC ceased, indicating that a significant drop in dynamic lung volume had occurred during CC (Fig. 5). On average, the drop in lung volume during CC was estimated at 335 ± 37 ml below the lung volume without CC for a PEEP level of 3 ± 1 cm H₂O. In two patients, a
change in PEEP level from 5 or 3 cm H₂O to zero was performed by the physician and allowed observation of the effects of PEEP on the inspiratory flow during CC (Fig. 6). Consistent with our in vitro findings, flow generated by CC became markedly limited when PEEP was removed, suggesting that airway collapse was partially prevented by PEEP. Total minute ventilation including volume mobilized by CC and volume delivered by the ventilator was 8.6 ± 3.2 liters/min. Compliance of the respiratory system measured in five patients after CC interruption was 40 ± 11 ml/cm H₂O (from 25 to 55 ml/cm H₂O).

DISCUSSION

The present in vitro study demonstrates that during CPR, ventilation takes place entirely below FRC, resulting in negative P_{RT} during decompression even when positive Paw and CFI is applied. Clinical observations of Paw and volumes during CPR are consistent with these findings. Compared with current recommended CPR strategies (13, 14, 30), positive Paw generated by CFI limits thoracic volume reduction, which could be protective for lung function and can generate some alveolar ventilation. In addition, positive Paw...
and CFI result in an increase in $P_{TT}$ during compression while $P_{TT}$ remains negative during decompression, which is essential for circulation.

**Intrathoracic Pressures**

The influence of $P_{TT}$ changes on hemodynamics was described as the “thoracic pump theory” (10, 33), which explains that $P_{TT}$ plays a major hemodynamic role during CPR. During CC, positive $P_{TT}$ results in ejection of blood in the extrathoracic vessels. Negative $P_{TT}$, caused by passive thoracic recoil during the decompression phase, is responsible for venous return. If $P_{TT}$ is not negative enough during decompression, this can decrease blood flow by impairing venous return during decompression, whereas a low positive $P_{TT}$ during compression decreases the forward blood flow. The amount of negative pressure generated, however, cannot be considered as proportionally increasing venous return and flow. Once pleural pressure is much below atmospheric pressure, the great veins collapse as they enter the thorax and there is flow limitation. Negative pressure, however, can promote atelectasis. This is a potential benefit of CFI plus PEEP. Our results showed that even with high CFI resulting in positive Paw around 10 cmH$_2$O, $P_{TT}$ during the decompression phase of CC remains negative while maximal $P_{TT}$ during the compression phase tends to magnify the intrathoracic, driving pressure during the cycle of CC. According to our model, the effect of reasonable positive pressure associated with CFI seems to carry more benefits than risks regarding the potential hemodynamic efficacy of CPR. The optimal level of positive Paw will depend on respiratory mechanics, but also on the strength and rhythm of CC. Studies in the 1980s suggested that an increase in PEEP would rise the mean $P_{TT}$, promoting a decrease in venous return and in the coronary perfusion pressures during CPR (21).

Animal and clinical studies demonstrated the potentially beneficial hemodynamic effect of the occurrence of a negative $P_{TT}$ promoted by an external device interspersed with positive pressure ventilation (2, 32, 39, 40). However, an important clinical trial (1) involving 8,718 cardiac arrest patients randomized to standard CPR or CFI done with the impedance threshold device that promoted a $P_{TT}$ negative showed no difference regarding survival with satisfactory function, return of spontaneous circulation, survival to hospital admission, and survival to hospital discharge. Animal (27) and clinical (10) studies suggested that a positive pressure ventilation synchronized with sternal compression improved brain perfusion and provided better hemodynamic parameters. The overall CC strategies used in these studies, however, were very different from current recommendations, and these effects need to be reexamined, as allowed by our mechanical model. The rate of CC recommended nowadays, with 80-100 compression-decompression phases, inducing incessant negative $P_{TT}$, explains that $P_{TT}$ will remain negative even with positive Paw applied at the tip of the endotracheal tube with CFI. This statement is supported by our in vitro and clinical observations showing significant lung volume loss during CPR.

**Generation of Ventilation**

The amount of ventilation generated by CC remains a controversial issue because of the difficulties of getting reliable measurements during CPR and also because of the varying conditions of previous animal and clinical experiments. In 1961, Safar et al. (34) demonstrated that manual CC generated a mean tidal volume of 156 ml (ranging between 0 to 390 ml) in anesthetized and curarized adults with an artificial airway, but they did find significant tidal volume generated in intubated cardiac arrest patients. A study in nonintubated dogs by Chandrana et al. (9) demonstrated that the mean tidal volume related to CC varied between 70 to 90 ml. During CPR, the volume mobilized by CC alone is therefore often small and likely below the physiological dead space in these settings. Idris et al. (23) showed in an animal experiment that ventilation related to CC without additional ventilation decreased significantly after 10 min and failed to sustain gas exchange. In contrast, experimental studies have shown that the simple transtracheal administration of oxygen could maintain ventilation during CPR (6, 26).

Our in vitro and in vivo measurements confirm that volumes mobilized by CC alone are probably smaller than anatomic dead space. The addition of a positive Paw dramatically increased ventilation on the bench, and also resulted in modest minute ventilation in our patients with a positive pressure of 3 to 4 cm H$_2$O set on the ventilator. An original feature of our bench model is the simulated airway collapse, which seems to play a major role during CPR because of the negative $P_{TT}$ during decompression and the lung volume reduction below FRC that may favor collapse of thoracic airways. The degree of collapse may vary between patients and directly affect volume mobilized by CC and thoracic volume loss. Equivalence regarding the level of collapse between in vitro and in vivo conditions is uncertain. Maintaining a modest level of positive airway pressure seems crucial to generate sufficient ventilation, as suggested in different animal models (20, 38). During CPR, two aspects of ventilatory needs must be distinguished. During the low-flow phase of resuscitation, the need for ventilation, i.e., clearance of CO$_2$, is low. Ventilation becomes more important as CPR is prolonged and respiratory mechanics worsen over time. The other component is oxygenation and the delivery of oxygen at the alveolar level. This part is essential to avoid or minimize hypoxemia and hypoxia. Administering a constant flow of oxygen together with a small positive pressure to help maintain open airways as in CFI is likely a useful approach.

**Specific Effects of CFI**

Steen et al. (37) in a pig animal model of CPR compared CFI of oxygen to intermittent positive pressure ventilation while performing mechanical CC. The animals randomized to CFI showed a higher level of return to spontaneous circulation, and the CFI group showed significantly higher levels of oxygenation and coronary perfusion pressure. Brochard et al. (7) in a pig model of CPR showed that CFI alone has no ventilatory effect when animals were simply disconnected from mechanical ventilation and with no CC, but that CFI combined with CC resulted in ventilation associated with higher values of aortic pressure and common carotid blood flows than standard CPR. In a case report, CPR was shown to be efficient during 1 h by using CFI as the unique strategy for ventilation (8). In a clinical trial, after basic CPR resuscitation, 95 patients were randomized to receive manual ventilation or CFI combined with mechanical CC. The percentage of patients who under-
went successful resuscitation were comparable (21% in the manual ventilation and 27% in the CFI group, not statistically significant), but the arterial blood gases performed after admission to the hospital showed significantly higher oxygenation and pH levels and lower partial pressure of arterial carbon dioxide in the CFI group (35). A more recent clinical study (3) randomized 696 out-of-hospital cardiac arrest victims to CPR done with mechanical ventilation or with CFI. The level of detectable pulse saturation and the proportion of patients with saturation above 70% were higher with CFI, which can be a consequence of improved circulation. No difference in outcome was noted regarding return to spontaneous circulation, hospital admission, or ICU discharge in a population with extremely poor prognosis.

Animal and clinical studies suggested that prolonged CPR may impair lung function and promote severe atelectasis (11, 19, 23, 28). CFI with positive pressure ventilation modifies P_{RT} and can attenuate the thoracic volume reduction. This might protect the lung from atelectasis during CPR PR and can attenuate the thoracic volume reduction. This might protect the lung from atelectasis during CPR and can attenuate the thoracic volume reduction. This might protect the lung from atelectasis during CPR and can attenuate the thoracic volume reduction. This might protect the lung from atelectasis during CPR and can attenuate the thoracic volume reduction. This might protect the lung from atelectasis during CPR and can attenuate the thoracic volume reduction. This might protect the lung from atelectasis during CPR and can attenuate the thoracic volume reduction. This might protect the lung from atelectasis during CPR and can attenuate the thoracic volume reduction. 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