A novel capnogram analysis to guide ventilation during continuous chest compressions resuscitation. From clinical to experimental observations

Amaud Lesimple  
CNRS, INSERM 1083, MITOVASC, University of Angers, Angers

Caroline Fritz  
Anesthesia-Intensive Care department, European Hospital Georges Pompidou APHP, Paris

Alice Hutin  
SAMU of Paris, Necker Hospital, Paris

Emmanuel Charbonney  
Hospital Center of University of Montréal, Montreal Qc H2X 0C1

Dominique Savary  
Vent’Lab, Angers University Hospital, University of Angers, Angers

Stéphane Delisle  
FCCM University of Montréal, Department of Family and Emergency Medicine, Montréal, Québec

Paul Ouellet  
Vitalité Health Network, North West Zone, Edmundston

Gilles Bronchti  
Anatomy department, University of Québec at Trois-Rivières, Trois-Rivières

Fanny Lidouren  
Ecole Nationale Vétérinaire d’Alfort, IMRB, AfterROSC Network, F-94700, Maisons-Alfort

Thomas Piraino  
St. Michael’s Hospital Toronto, Ontario

François Beloncle  
Vent’Lab, Angers University Hospital, University of Angers, Angers

Nathan Prouvez  
Vent’Lab, Angers University Hospital, University of Angers, Angers

Alexandre Broc  
Vent’Lab, Angers University Hospital, University of Angers, Angers

Alain Mercat  
Medical ICU, Angers University Hospital, University of Angers, Angers

Laurent Brochard
Keenan Research Centre for Biomedical Science, Li Ka Shing Knowledge Institute, St. Michael’s Hospital, Toronto

**Renaud Tissier**
Ecole Nationale Vétérinaire d’Alfort, IMRB, AfterROSC Network, F-94700, Maisons-Alfort

**Jean-Christophe Richard**
Vent’Lab, Angers University Hospital, University of Angers, Angers

**Cardiac Arrest and Ventilation International Association for Research Group CAVIAR**
Anatomy department, University of Québec at Trois-Rivières, Trois-Rivières

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**Research Article**

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Abstract

Background: Cardio-Pulmonary Resuscitation (CPR) decreases lung volume below the functional residual capacity and can generate intrathoracic airway closure. Conversely, large insufflations can induce thoracic distension and jeopardize circulation. The capnogram (CO$_2$ signal) obtained during continuous chest compressions can reflect intrathoracic airway closure and we hypothesized here that it can also indicate thoracic distension.

Objectives: to test whether a specific capnogram may identify thoracic distension during CPR and assess its impact on gas exchange and hemodynamics.

Methods:

1. In out-of-hospital cardiac arrest patients, we identified on capnograms three patterns: intrathoracic airway closure, thoracic distension or regular pattern. An algorithm was designed to identify them automatically.

2. To link CO$_2$ patterns with ventilation, we conducted three experiments:

i) Reproducing the CO$_2$ patterns in human cadavers.

ii) Assessing the influence of tidal volume and respiratory mechanics on thoracic distension using a mechanical lung model.

iii) Exploring the impact of thoracic distension patterns on different circulation parameters during CPR on a pig model.

Measurements and main results:

Clinical data: 202 patients were included. Intrathoracic airway closure was present in 35%, thoracic distension in 22% and regular pattern in 43%.

Experiments:

i) Higher insufflated volumes reproduced thoracic distension CO$_2$ patterns in 5 cadavers.

ii) In the mechanical lung model, thoracic distension patterns were associated with higher volumes and longer time constants.

iii) In six pigs during CPR with various tidal volumes, a CO$_2$ pattern of thoracic distension, but not tidal volume per se, was associated with a significant decrease in blood pressure and cerebral perfusion.

Conclusions: During CPR, intrathoracic airway closure, thoracic distension or regular pattern can be identified by capnogram analysis. A thoracic distension pattern on the capnogram may indicate a
negative impact of ventilation on blood pressure and cerebral perfusion during CPR, not predicted by tidal volume per se.

**Introduction**

In the management of cardiac arrest, it is recommended to perform high quality chest compressions (1). The optimal ventilation strategy during Cardio-Pulmonary Resuscitation (CPR) remains to be determined (2). CO₂ monitoring is recommended in clinical practice by International guidelines (1, 3). However, the application of continuous chest compressions during CPR influences CO₂ waveform and complicates its interpretation (4, 5). We previously showed that the actual recommended rate and depth of chest compressions are such that CPR tends to operate below the functional residual capacity (FRC) (6). During each chest decompression, the natural recoil of the chest creates a negative intrathoracic pressure with a beneficial circulatory effect. We also showed that the reduction of lung volume due to continuous chest compressions can result in “intrathoracic airway closure” that influences the capnogram waveform (7). We recently identified in out-of-hospital cardiac arrest patients, another capnogram pattern referred to as “thoracic distension”, in which oscillations are not present at the beginning of expiration but appear after a few chest compressions have been generated, while lung volume decreases. We hypothesized that relatively large insufflations place lung volume above functional residual capacity, therefore losing the natural inward / inspiratory recoil of the chest and transiently affecting the circulatory effect of decompression by limiting negative recoil pressure, until returning below FRC. The significance of this “thoracic distension” CO₂ pattern, as representing a potentially harmful condition for circulation, was investigated in the present study.

The objectives of this study were: i) to design an algorithm permitting to classify and assess the occurrence of the different CO₂ patterns observed during CPR in series of out-of-hospital cardiac arrest patients; ii) to reproduce the CO₂ pattern associated with thoracic distension on different experimental models; iii) to evaluate the impact of a thoracic distension capnogram pattern on ventilation and circulation in pigs during CPR performed with continuous chest compressions.

**Methods**

**CO₂ patterns detection**

*Capnogram classification: the three patterns.*

Capnograms were analyzed as illustrated on figure 1 (panels A, B and C) using a simple classification algorithm. For each ventilatory cycle, the expiratory part of the CO₂ signal obtained during continuous chest compressions was labelled into one of the three patterns defined as follows:

1) **intrathoracic airway closure**: oscillations due to chest compressions and decompressions are small or absent. Lung volume reduction far below the FRC and complete or partial intrathoracic airway closure
explain this capnogram.

ii) thoracic distension: oscillations due to chest compressions and decompressions are limited or absent at the beginning of the expiration phase, and resume after a few chest compressions. Increase of lung volume above FRC explains this capnogram.

iii) regular pattern: oscillations due to chest compressions and decompressions are clearly visible during the entire duration of the expiration phase. This pattern corresponds to the situation when neither thoracic distension nor intrathoracic airway closure is identified.

The different steps of the classification algorithm are detailed in the supplementary methods (additional file 1).

**Distension ratio, definition and calculation.**

To quantify the thoracic distension pattern, a distension ratio was defined based on the analysis of the area under the CO$_2$ curve (see figure 1 panel D). In case of thoracic distension, one, two or sometimes more CO$_2$ oscillations disappear at the beginning of expiration despite the delivery of continuous chest compressions. To explain this specific CO$_2$ feature, we hypothesized that the thorax is transitorily above FRC due to the insufflation, preventing a negative recoil pressure during decompression (that only occurs below the FRC) and as result preventing chest compression-induced CO$_2$ oscillations. To assess the level of thoracic distension, we computed the ratio between two areas under the capnogram curve; 1. the area 1 under the CO$_2$ curve (AUC1) from the beginning of expiration to the first local minimum (the first local minima having an amplitude two times lower than the mean amplitude of all peaks are discarded) and 2. the area 2 under the CO2 curve (AUC2) of the first “normal” oscillation corresponding to an efficient compression decompression phase around FRC (see figure 1 panel D). The distension ratio corresponds to the ratio AUC1 / AUC2. To evaluate the incidence of thoracic distension, a distension ratio of 2 was arbitrarily defined as a cut-off value; considering that the loss of oscillations includes at least two inefficient chest decompressions (distension ratio >= 2).

**Clinical observations**

Capnograms were obtained from patients enrolled in the French RENAU network registry for Out of Hospital Cardiac Arrest (OHCA) (authorization number CNIL 046461). Patients who were receiving manual continuous chest compressions after intubation according to international recommendations (1) with available capnograms were enrolled in the study. Of the patients included in the present study (n=202), capnograms of 89 patients were already reported in a previous study (7). Patients were ventilated with a transport ventilator (Monnal T60, Air Liquide Medical Systems Antony, France) using a bilevel pressure mode called CPV, with standardized ventilator settings: respiratory rate (RR) 10 breaths/min; inspiratory time 1 second and expiratory time 5 seconds (I/E=1/5); inspired oxygen fraction (FiO2) 100%; inspiratory pressure 20 cmH$_2$O; positive end expiratory pressure (PEEP) 5 cmH$_2$O. Soon after intubation, CO$_2$ signal was recorded at airway opening from LifePak monitor/defibrillator (LIFEPACK 15,
Physio-Control, Redmond, WA 98052, USA) with a sidestream sensor placed between the Y-piece and the endotracheal tube. Data were prospectively collected without any interference with care. The study complied with the Declaration of Helsinki and was approved by the ethics committee of the University Hospital of Clermont-Ferrand, France (IRB no. 5891) with waiver of consent.

**Human cadavers with simulation of CO₂ production.**

To validate observations obtained from clinical data, the different conditions (i.e., intrathoracic airway closure, thoracic distension and regular pattern) were reproduced with Thiel embalmed human cadavers with simulation of CO₂ production in the Anatomy Laboratory of the Université Québec à Trois Rivières (UQTR) in Canada with five bodies - authorization number CER-14-201-08-06-17). Those cadavers were validated as a robust model to study ventilation during CPR (8). The study was approved by the ethics committee of the University of Quebec at Trois-Rivieres (SCELERA-19-01-PR02). Methods used to ventilate the cadavers and to simulate CO₂ production have already been described; airway pressure, flow and esophageal pressure were recorded (8, 9) (see supplementary methods - additional file 1). The Airway Opening Pressure (AOP) was determined in each cadaver as previously reported (10). Regular pattern and thoracic distension were obtained with a PEEP set above AOP while intrathoracic airway closure was obtained with a PEEP set below AOP. Using pressure-controlled ventilation, we adapted different inspiratory pressures (20, 30, 40 cmH₂O) to generate a high range of tidal volumes. Ventilation cycles were classified according to the same algorithm used for the clinical study as intrathoracic airway closure, thoracic distension or regular pattern based only on the CO₂ pattern.

**Mechanical bench with simulation of CO₂ production.**

The objective of the bench study was to reproduce CO₂ patterns identified as thoracic distension and better understand the influence of respiratory mechanics and volume on this pattern. A thoracic lung model called POUTAC was used to reproduce the mechanical properties of the respiratory system during CPR as described previously (6) (see supplementary methods - additional file 1). The model is designed to allow ventilation either above (as permitted by all lung models) or below FRC (a unique situation specific to CPR). To study the potential impact of thoracic distension on the CO₂ signal during CPR, manual chest compressions were applied continuously on the POUTAC using different compliance (C= 20 – 40 – 60 ml/cmH₂O) and resistance (R = 5 – 10 cmH₂O/L/s), and capnograms were recorded. For each set of resistance and compliance (time constant as RxC), different tidal volumes were delivered to the POUTAC ranging from 0.3L to 1L. For each configuration (of RxC and tidal volume), capnogram was analyzed and classified into either thoracic distension or regular pattern as defined with the classification used in both the clinical and the cadaver study.

**Animal study**

*Ethical statement*
This study was approved by the ethics committee for animal research Cometh - 016 (project 2018062813205311). The procedure for the care and sacrifices of study animals was in accordance with the European Community Standards on the Care and Use of Laboratory Animals. A reporting checklist regarding animal preparation and study design is provided in the additional file 2, in compliance with the ARRIVE guidelines.

**Experimental protocol**

We tested 7 female pigs weighing 28±1 kg. A first animal was tested over a large range of tidal volumes (from 6 ml/kg to 20 ml/kg) to illustrate what can be expected in terms of circulation impact and capnogram patterns.

Six animals were enrolled in the main study. Ventricular fibrillation was induced by a pacing wire inserted in the right ventricular through the femoral vein catheter. Fibrillation was left untreated during 4 min (no-flow period). Then continuous mechanical chest compression was started at a rate of 100 per minute and a depth of 5 cm with ventilation as recommended (100 % oxygen fraction, respiratory rate 10/min, I/E 1/5, tidal volume 6 ml/kg). CPR was organized into three periods associated to a specific tidal volume (period T0 to T5 => 5 minutes at 6 ml/kg - period T5 to T10 => 5 minutes at 12 ml/kg - period T10 to T15 => 5 minutes at 6 ml/kg). Blood gases were measured at each tidal volume change. Animals were sacrificed at the end of the protocol (i.e., low-flow period of 15 minutes) with a lethal dose of pentobarbital (60 mg.kg⁻¹). Details of animal preparation are available in the supplementary methods (additional file 1).

**Capnogram analysis and thoracic distension.**

Thoracic distension was defined based on the “distension ratio” calculated as a continuous variable as illustrated in figure 1 (panel D). This ratio was computed and averaged for each tidal volume period. Correlations between the “distension ratio”, tidal volume, time and hemodynamic parameters were performed.

**Statistical analysis**

Statistical analysis was performed with Python Software (Python version 3.9.5, Wilmington - USA). Data are summarized as mean (± SD) for continuous variables and count (%) for categorical variables. Comparisons between groups were assessed with ANOVA test. Concerning bench experiments, results were averaged over three ventilation cycles for every condition. For the pig experimentation, correlation was assessed using a random effects linear model with each pig’s id as the random effect. All statistical tests were two-sided and results with p <0.05 were considered statistically significant.

**Results**

**Clinical observations.**
Capnography was obtained in 202 patients soon after intubation during continuous chest compressions and all were included in the study. Patients’ characteristics and outcomes are described in table 1. Return of spontaneous circulation (ROSC) and rates of survival at hospital admission were 20.5% and 12.9% respectively.

From the 202 patients included in the study, 35% of patients showed airway closure, 22% thoracic distension pattern and 43% regular pattern (see figure 1).

**Human ThielCadavers**

The characteristics of the cadavers are given in the supplementary table 1 (additional file 2). Figure 2 shows an illustration of the three CO₂ patterns obtained with the Thiel cadavers. Thoracic distension based on capnogram was associated with higher tidal volumes compared with intrathoracic airway closure (p = 0.008) or regular pattern (p = 0.005) (after ANOVA). Mean tidal volume was 130 ± 136 ml for intrathoracic airway closure, 453 ± 222 ml for thoracic distension and 141 ± 82 ml for regular pattern.

**Bench study**

Table 2 shows that thoracic distension was favored by high tidal volumes and high time constants (R x C). The larger the insufflated volume or the longer the time constant, the more likely thoracic distension was present. Thoracic distension was identified on capnograms with a frequency of 0%, 0%, 33%, 33%, 66%, 83%, 83% and 100% for insufflated volumes of respectively 300 ml, 400 ml, 500 ml, 600 ml, 700 ml, 800 ml, 900 ml and 1000 ml. Thoracic distension was detected on capnograms with a frequency of 13%, 50%, 38%, 50%, 75% and 75% for RC values of respectively 0.10 s, 0.2 s, 0.25, 0.40 s, 0.5 and 0.80 s.

**Pig model**

Intrathoracic airway closure was not observed in the animals enrolled in the experiment. Pigs’ characteristics are given in the supplementary table 2 (additional file 2).

**Test Animal.**

Figure 3 illustrates in one animal the increasing variations induced by ventilation of aortic blood pressure, right atrial pressure, intracranial pressure, coronary and cerebral perfusion pressure as Vt increased. The capnogram depicted a change of the CO₂ pattern from regular pattern to thoracic distension as Vt increased.

**Experiment in six animals.**

The “distension ratio”, expressing the level of thoracic distension based on the capnogram, (figure 4, panels A-B and figure 5) was significantly and inversely correlated with cerebral perfusion pressure (p = 0.002), with mean blood pressure (p=0.006), systolic blood pressure (p=0.007), and diastolic blood
pressure (p=0.009). There was no significant effect on coronary perfusion pressure and carotid blood flow.

The different hemodynamic parameters recorded were not significantly impacted by tidal volume per se. There was no significant correlation between Vt and any recorded circulation parameter, as shown on figure 4 (panels C and D - coronary and cerebral perfusion pressure) and supplementary figure 1 (additional file 2 - mean, systolic and diastolic blood pressure as well as carotid blood flow).

A time effect was present on the different hemodynamic parameters recorded except for coronary perfusion pressure, cerebral perfusion pressure and diastolic blood pressure (supplementary figures 2 and 3 - additional file 2).

**Discussion**

The main results of the present translational study could be summarized as follows:

1. In the present series, intrathoracic airway closure, thoracic distension and regular pattern concerned respectively 35%, 22% and 43% of 202 OHCA patients after intubation.

2. The capnogram indicating thoracic distension was associated with higher tidal volumes on Thiel cadavers. Capnogram indicating thoracic distension on a CPR bench model was also more likely to occur with higher insufflated volumes or longer time constants (RxC).

3. In the animal experiment, the distension ratio calculated from the capnogram to quantify thoracic distension was inversely correlated with cerebral perfusion and arterial blood pressure while no correlation was found with tidal volume.

**Theoretical optimal thoracic volume for effective chest compressions**

The application of continuous chest compressions during CPR complicates CO2 waveform interpretation and generates specific CO2 patterns (4, 5, 6, 7). Both compression and decompression are needed to generate and sustain effective circulation. The increase of intrathoracic pressure during compression has been shown to generate circulation, thus introducing the concept of thoracic pump theory (11). Venous return is facilitated by natural recoil of the chest creating a negative intrathoracic pressure if lung is placed below the functional residual capacity (FRC) when decompression starts. CPR close to the FRC with effective venous return could be identified by the regular CO2 pattern with fully oscillating capnogram. Interestingly, non-oscillating capnograms reported by Grieco et al. (7) reflect intrathoracic airway closure that affects ventilation and occurs when thorax is pushed far below the FRC along the course of CPR.

*Thoracic distension* pattern of the capnogram
We hypothesized that the specific capnogram called “thoracic distension” may indicate the risk associated with excessive ventilation inflating the thorax above FRC. It may jeopardize circulation (venous return) by limiting negative intrathoracic pressure during decompression (12, 13). Expired CO2 oscillations which result from the combination of compression and decompression may transiently disappear when the time during which thoracic volume above FRC is prolonged, indicating this risk (see figure 6 and additional file 2).

This is also markedly visible in the pig model (test animal), where we observed that the stepwise increase of Vt from 6 to 20 ml/kg magnified coronary and cerebral circulation oscillations related to ventilation and modified capnogram from regular to thoracic distension in parallel (figure 3).

**Is the CO2 pattern associated with thoracic distension more informative than the Vt to detect any impact on circulation?**

Thoracic distension CO2 pattern was reproduced on cadaver, bench and porcine models. This phenomenon was associated on average with higher insufflated volumes compared to intrathoracic airway closure or regular patterns. We found in the pig model that thoracic distension assessed by distension ratio was significantly and negatively correlated to mean arterial blood pressure and cerebral perfusion pressure, suggesting its potential negative impact on circulation during resuscitation.

Unlike the capnogram, Vt absolute values were not significantly associated with a negative effect on blood pressure, coronary perfusion and cerebral perfusion. Those results may suggest that the capnogram may be more relevant than Vt per se to predict a circulatory impact induced by ventilation.

The bench study provides a possible explanation for the previous observed result. Indeed, prolonged time constant that characterizes the time required to return to FRC may favor thoracic distension even with low Vt.

**Occurrence of thoracic distension, intrathoracic airway closure and regular capnogram**

In our series of 202 OHCA patients, thoracic distension and intrathoracic airway closure concerned 22% and 35% of patients respectively. Interestingly, very similar capnograms have been reported during CPR, without specifically identifying the phenomenon of thoracic distension (4, 5).

An important methodological point is that capnograms from the present study were captured soon after intubation with a respiratory rate of 10/min and a protective pressure mode of ventilation limiting Vt. One cannot exclude that thoracic distension may be much more frequently observed with manual bag ventilation during which Vt and respiratory rate are poorly controlled thus favoring the risk of hyperventilation. In addition, a moderate level of PEEP was used in our series, which could have minimized the occurrence of intrathoracic airway closure, favored by low airway pressures. As CO2 patterns may evolve during CPR intervention, the classification presented in the current study may have
changed depending on the time of intervention, thus precluding any interpretation of its significance in terms of outcome.

Of note, intrathoracic airway closure was not observed during the animal experiment. It is possible that the pig thorax anatomy may limit the reduction of lung volumes we observe in humans during resuscitation and thus occurrence of intrathoracic airway closure. Besides, pig bronchial tree presents lateral connections that may also limit occurrence of distal airway closure (14).

**Clinical perspectives**

Excessive ventilation during cardiac arrest has already been shown to be associated with poor outcomes (15, 16). Nevertheless, it is definitively challenging to control and monitor Vt delivered during manual bag ventilation (17).

Based on these observations, a capnogram-based ventilation strategy may permit to optimize ventilation during CPR, using real-time identification of capnograms (intrathoracic airway closure, thoracic distension or regular pattern). As previously shown, PEEP increase may be considered in case of intrathoracic airway closure to open the airways, while Vt reduction could be proposed in case of thoracic distension. Further evidence is needed before developing such ventilatory approach on a ventilator but these findings may already be of high additional value for bag valve mask ventilation during which hyperventilation is likely to occur.

**Study limitations**

The capnogram analysis proposed in the present study is limited to continuous chest compression and is not directly applicable to interrupted chest compression strategy.

In the animal study, since each animal was its own control, several time related factors might have also impacted circulation. Further studies comparing animals with different ventilation strategies are needed to confirm our observations.

**Conclusion**

During CPR, intrathoracic airway closure, thoracic distension or regular pattern can be reliably identified by the capnogram analysis. We describe a novel CO2 pattern indicating relative thoracic distension, which may be associated with a negative impact on blood pressure and cerebral perfusion, irrespective of tidal volume per se. This original capnogram classification has the potential to help optimizing ventilation during CPR.

**Abbreviations**

- CPR
- Cardio-Pulmonary Resuscitation
- **FRC**
  - Functional Residual Capacity
- **RR**
  - Respiratory Rate
- **FiO₂**
  - Inspired Fraction of Oxygen
- **PEEP**
  - Positive End Expiratory Pressure
- **ROSC**
  - Return Of Spontaneous Circulation
- **R**
  - resistance
- **C**
  - compliance

### Declarations

### Funding

CF received a research grant from French society of intensive care medicine (SRLF).

### Authors' contributions

AL, DS, LB, RT and JCR contributed to the study conception and design. CF, AL, AH, EC, PO, SD, FL, YL, NP, AB and JCR performed the experiments, the data collection and the initial data analysis. JCR, AL, RT, CF and LB prepared the first draft of the manuscript. All authors contributed to the data analysis and to the critical revision and approval of the final manuscript. The study was performed in Veterinary school of Maisons-Alfort (France), Anatomy laboratory of University of Quebec at Trois Rivieres (Canada) and University Hospital of Angers (France).

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### References


**Tables**

Table 1

<table>
<thead>
<tr>
<th>Patients characteristics (n=202)</th>
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<tbody>
<tr>
<td>Age (year)</td>
</tr>
<tr>
<td>Sex male (n)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
</tr>
<tr>
<td>Initial rhythm (n)</td>
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<tr>
<td>• Non - shockable</td>
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<td>• Shockable</td>
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<td>Low-flow time (min)</td>
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<td>EtCO₂ at the beginning of ALS (mmHg)</td>
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<td>Maximal EtCO₂ during ALS (mmHg)</td>
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<td>ROSC (n)</td>
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<td>Survival at hospital admission (n)</td>
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Data are presented as means (± SD) for continuous variables and count (%) for categorical variables.

- BMI: body mass index calculated as weight/height²;
- EtCO₂: end tidal CO₂;
- ALS: advanced life support;
- ROSC: return of spontaneous circulation;

Table 2

<table>
<thead>
<tr>
<th>Thoracic distension reproduced on lung model</th>
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The thoracic distension pattern was reproduced on the thoracic lung model called POUTAC. This table displays CO\textsubscript{2} pattern depending on time constant RC (multiplication of resistance and compliance) and the set tidal volume using the classification algorithm described in the clinical study. Each combination of time constant and tidal volume was identified into either regular pattern (called “regular”), or thoracic distension (called “distension”).

**Figures**
The figure illustrates on the top (panels A, B and C) the distribution of capnograms according to the classification. Each panel illustrates a typical capnogram obtained from clinical observations after numerical treatment from raw capnogram data (python, Python Software Foundation, Wilmington, Delaware, USA). X-axis represents inspiratory and expiratory time.

A) **intrathoracic airway closure**: oscillations due to chest compressions and decompressions are small or absent. Lung volume reduction far below the FRC and complete or partial intrathoracic airway closure explain this specific capnogram.

B) **thoracic distension**: oscillations due to chest compressions and decompressions are limited or absent at the beginning of the expiration phase, and resume after a few chest compressions. Increase of lung volume due to large Vt insufflation before returning to FRC explains this specific capnogram.

C) **regular pattern**: oscillations due to chest compressions and decompressions are markedly visible during the entire duration of the expiration phase. The regular pattern corresponds to the situation when neither thoracic distension nor intrathoracic airway closure is identified.
Panel D shows examples of distension ratio (used to quantify thoracic distension) calculated as a continuous variable for the animal experiment.

**D)** Typical capnograms for three values of “distension ratio” are displayed: 5.5 on the left, 3.5 at the center and 1.5 on the right.

AUC1 represents the area under the CO$_2$ curve between the beginning of the expiratory CO$_2$ signal and the first local minimum (the first local minima having an amplitude two times lower than the mean amplitude of all peaks are discarded).

AUC2 represents the area under the CO$_2$ curve of the first “normal” oscillation corresponding to an efficient compression decompression phase around FRC.

The distension ratio corresponds to the ratio $AUC_1 / AUC_2$. It is used as a surrogate marker of the level of thoracic distension.
Figure 2

Reproduction of CO₂ patterns on Thiel cadaver model

Reproduction of CO₂ patterns: illustration in one cadaver.

From top to bottom recordings of flow at airway opening (Flow), airway pressure (Paw), esophageal pressure (Poeso) and expired CO₂ (CO₂). The tilted line on the Paw tracing represents the Airway Opening Pressure (AOP). The recording is divided into three configurations:

1. Regular pattern: Positive End Expiratory Pressure (PEEP) was set above the AOP to simulate airway patency.

2. Intrathoracic airway closure: PEEP was set below the AOP to simulate airway closure.

3. Thoracic distension: PEEP was set above the AOP to simulate airway patency and peak airway pressure set on the ventilator was increased to generate higher tidal volumes compared to step 1.

Figure 3
Impact of a stepwise increase of tidal volume on airway pressure, circulation and capnograms in a pig during cardiopulmonary resuscitation

From top to bottom, recording tracings of airway pressure, aortic blood pressure, right atrial pressure, intracranial pressure, coronary perfusion pressure (aortic blood pressure minus right atrial pressure), cerebral perfusion pressure (mean arterial pressure minus intracranial pressure) and capnogram during tidal volume (Vt) trial. Vt was increased as follows: 6 - 10 - 15 - 20 ml/kg. Coronary perfusion pressure waveforms should be interpreted cautiously and read only at end of decompression.

Figure 4

Relationship between CO$_2$ pattern analyzed by the distension ratio or tidal volume and coronary and cerebral perfusion in pigs during cardiopulmonary resuscitation
A. Coronary Perfusion Pressure (measured at end-decompression) depending on “distension ratio”. Correlation was assessed using a mixed linear model. The p-value is displayed. Each pig is represented by a different color.

B. Cerebral Perfusion Pressure (mean value throughout chest compression / decompression cycles) depending on “distension ratio”. Correlation was assessed using a mixed linear model. The p-value is displayed. Each pig is represented by a different color.

C. Coronary Perfusion Pressure depending on tidal volume in ml/kg. Correlation was assessed using a mixed linear model. The p-value is displayed. Each pig is represented by a different color.

D. Cerebral Perfusion Pressure depending on tidal volume in ml/kg. Correlation was assessed using a mixed linear model. The p-value is displayed. Each pig is represented by a different color.
Figure 5

Relationship between CO2 pattern analyzed by the distension ratio and mean, systolic, diastolic blood pressure and carotid blood flow in pigs during cardiopulmonary resuscitation

This figure illustrates the relationship between distension ratio and mean blood pressure (panel A: upper left), systolic blood pressure (panel B: upper right), diastolic blood pressure (panel C: lower left) and carotid blood flow (panel D: lower right). Correlation was assessed using a mixed linear model. The p-value is displayed. Each pig is represented by a different color.

Figure 6

Illustration of thoracic distension mechanism based on airway pressure, flow and CO2 analysis
This figure illustrates from top to bottom, airway pressure (Paw), flow at airway opening (Flow) and expired CO2 (CO2) tracings obtained in cadavers (panel A), bench (panel B) and animals (panel C). The left column illustrates thoracic distension while the right column represents regular pattern.

For each situation, the two grey vertical tilted lines define the time for the lung volume to return to FRC (time with thorax above FRC) while the two black vertical tilted lines define the expiration time (time between two insufflations).

Positive flow indicates decompression or insufflation. Negative flow indicates compression or exhalation. Please note the exact time correspondence between flow and CO2 oscillations whatever the situation.

During expiration, in case of thoracic distension (left column), the flow does not return to zero line during a couple of CC indicating that the thorax is still above FRC even during the decompression phase. CO2 oscillations resume only once the flow crosses the zero line, thus indicating the return of lung volume to FRC.

On the contrary, the right column obtained with a smaller Vt illustrates that the flow induced by CC crosses the zero line immediately after insufflation generating CO2 full oscillations. This specific full oscillating CO2 pattern indicates that chest compressions operate close to FRC.

**Supplementary Files**

This is a list of supplementary files associated with this preprint. Click to download.

- Additionalfile1supplementarymethods.docx
- Additionalfile2supplementaryresults.docx