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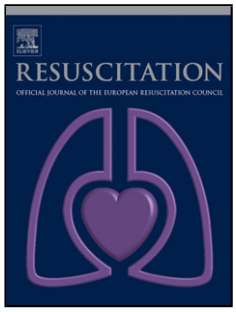

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Chest compressions during ventilation in out-of- hospital cardiac arrest cause reversed airflow.

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Abstract

Aim: During cardiopulmonary resuscitation, once the patient is intubated, compressions and ventilations are performed simultaneously. Chest compressions during the inspiratory phase of ventilation may force air out of the lungs, causing so-called "reversed airflow", which may lead to ineffective ventilation. The purpose of this study is to determine the occurrence of this phenomenon and to quantify the volume of reversed airflow.

Methods: Observational study. During manual ventilation of intubated patients receiving chest compressions, the pressure gradient over the endotracheal tube was measured using two air-filled catheters connected to a custom-made portable device. Chest compression data were measured using an accelerometer on a Zoll E- series defibrillator. All data are reported as mean (standard deviation; range).

Results: Twenty-five patients and a total of 368 ventilations were studied, on average 15 (6; 10-30) per patient. The mean tidal volume, minute volume and ventilation rate were respectively 690 ml (160; 240-1260), 10.5 L/min (4.8; 4.4-22.1) and 18/min (6; 6-35). Reversed airflow was observed in 21/25 patients (84%) and in 65% of all ventilations, with on average two episodes per ventilation. Fifty-five percent of the chest compressions during the inspiratory phase of the ventilation generated reversed airflow. The mean volume of the reversed airflow was 96 ml per episode (52; 12-364).

Conclusion: Chest compressions during ventilation in intubated patients generated reversed airflow in most patients. There was wide variation in the number of episodes and volume of the reversed airflow between patients. The effect of this phenomenon on the efficacy of ventilation during resuscitation and on outcome needs further investigation.

Key words: compressions, CPR; feedback, resuscitation; reversed airflow; tracheal pressure measurements; ventilation

1. Introduction

Cardiopulmonary resuscitation (CPR) consists of chest compressions and ventilations to sustain blood flow and oxygenation until spontaneous circulation is restored.[1] The optimal ventilation strategy during advanced cardiac life support in intubated patients is unkown.[2] The guidelines of the European Resuscitation Council (ERC) recommend the use of Intermittent Positive Pressure Ventilation (IPPV) with a ventilation rate of 10 breaths per minute and a tidal volume of 6-7 ml/kg.[3] Once the endotracheal tube is placed, chest compressions and ventilations are performed simultaneously and asynchronously without pausing chest compressions in order to shorten hands-off time and to improve the effectiveness of resuscitation.[4,5] This technique, however, causes multiple interactions between compressions and ventilations. Chest compressions increase the peak inspiratory pressures. In ventilations without chest compressions peak inspiratory pressures are 17 to 24 cm H_2O on average, whereas they are 43 to 61 cm H_2O when chest compressions are superimposed on the ventilations.[6,7] Chest compressions cause atelectasis of the alveoli and generate extravascular lung water.[8,9] In pigs there is a significant decrease of dynamic lung compliance after 30 minutes of cardiopulmonary resuscitation and in humans, lung compliance decreases shortly after starting chest compressions.[6,8] Chest compressions superimposed on ventilations may have a positive effect on haemodynamics due to improvement of the thoracic pump mechanism, provided that venous return is not compromised by excessive ventilation rates[10,11,12] Little is known about the effect of chest compressions on the ventilation flow during IPPV. Ventilations and chest compressions are opposite forces and chest compressions during the inspiratory phase of the ventilation may drive air out of the lungs, so-called “reversed airflow” (RF), which could lead to ineffective ventilation. The purpose of the current study was to investigate the effect of chest compressions on ventilation by determining the occurrence and the volume of RF.

2. Materials and methods

2.1 Study population

The current study is part of a larger project evaluating the use of airway pressures during resuscitation, started in 2010. In this single centre observational study, tracheal pressures are measured using a custom-made device in adult patients (≥18 years) suffering out-of-hospital cardiac arrest (OHCA), resuscitated and intubated in the pre-hospital setting by the Mobile Intensive Care Unit of Ghent University Hospital (staffed by an emergency medical technician, an emergency nurse and an emergency physician) according to the ERC guidelines. We

previously reported applications of this technique to discriminate oesophageal intubation from tracheal intubation, to measure ventilation rates during prehospital care and to detect and quantify gasping.[13,14,15,16] In this retrospective subanalysis, twenty-five consecutive patients eligible for analysis were selected from our database. Patients were eligible for analysis if data on airway pressures and chest compression depth and rate of at least one minute was available and if no positive end-expiratory pressure (PEEP) was applied. Ghent University Hospital is a tertiary referral hospital (annual census of about 35 000 patients in the emergency department) located in the city of Ghent (population approximately 300 000). The Ethics committee of Ghent University Hospital approved the study granting deferred consent (B67020083371).

2.2 Materials

During ventilation, the pressure gradient over the endotracheal tube was measured using two air-filled catheters connected to a custom-made portable two-channel recording device (16.5 cm x 8.0 cm x 4.0 cm) (Fig.1). For a detailed description of the materials and methods to measure the tracheal pressures we refer to our previous work.[13] The trachea of all patients was intubated with an endotracheal tube (Lo-Contour™, Mallinckrodt, Hazelwood, MO, USA). After securing the airway, the catheter system was attached to the endotracheal tube and the recording device was activated. Once the intervention completed, the recording was stopped. Rescue personnel were blinded for the data registered by the device as it contained no display or any other instant output. All intubated patients were manual ventilated using an adult 1600 ml self-inflating bag without a positive end-expiratory pressure (PEEP) valve (Laerdal, Stavanger, Norway) After registration, data stored on the recorder were transferred to a personal computer using MSR 5.04 software (MSR, Seuzach, Switzerland).

All patients were monitored with a Zoll E series defibrillator (Zoll Medical Company, Chelmsford, USA). The cardiac rhythm, the compression depth and rate were monitored using self-adhesive defibrillation pads with an accelerometer which was placed on the sternum of the patient (CPR stat-padz multifunction, Zoll Medical Company, Chelmsford, USA). After each resuscitation event, the data from the defibrillator were uploaded to a computer using Code Review 5.5.4 (Zoll Medical Company, Chelmsford, USA). The MSR and Zoll files were exported to a csv file and imported into a spreadsheet application (Excel, Microsoft Corporation, Redmond, USA). A custom-developed Visual Basic for Applications (VBA) program was used to visualise and analyse the ventilation and compression pressure waveforms. For each patient, the ventilation pressure and compression data of the first minute of registration

were synchronised and analysed. The use of both proximal and distal airway pressure signals allowed the calculation of multiple pressure-derived respiratory variables during CPR such as ventilation rate, peak inspiratory pressure, maximum inspiratory flow, inspiratory time, tidal volume and RF volume. The airflow was calculated from the pressure difference between the distal and proximal measuring point in the endotracheal tube, and the resistance to airflow of the endotracheal tube by means of Rohrer's coefficients of linear and nonlinear resistance (Fig 2.A).[17,18] Ventilation volumes were obtained by integration of airflow over in- or expiratory time (Fig. 3A).

An episode of RF was defined as the reversal of the ventilation flow during inspiration caused by a single chest compression. Tidal volumes were calculated as the sum of the inspiratory airflow fragments in case reversed airflow occurred, as illustrated in Fig 3B.

2.3 Statistical analysis

For statistical analysis SPSS 19.0 (Statistical Package for the use of Social Sciences, IBM Company, USA) and a Data Analysis Tool pack in Microsoft Excel (Microsoft, Redmond, WA, USA) were used. The significance level was set at 0.05. All variables are expressed as mean (standard deviation; range). Categorical variables are expressed as percentage of occurrence.

3. Results

Data were analysed from 25 patients (16 male), resuscitated between March 2010 and May 2011. The mean age was 66 (14; 30-87) years. Asystole was the initial rhythm in 18/25 (72%) of the patients, pulseless electrical activity in 4/25 (16%), ventricular fibrillation in 2/25 (8%) and ventricular tachycardia in 1/25 (4%). Eight (32%) patients achieved a sustained return of spontaneous circulation (ROSC) and were admitted. Seven died in the hospital and one was discharged.

RF was observed in twenty-one patients (84%), 95% confidence interval (69%; 98%), with an average volume of 96 ml per episode (52; 12-364), 95% confidence interval (90ml; 102ml). A total of 368 ventilations were studied, on average 15 (6; 10-30) per patient. The mean tidal volume was 690 ml (160; 240-1260). Sixty-five percent of the ventilations were associated with one or more episodes of RF (Fig. 4A). If RF occurred during a particular ventilation, then on average 24% (17; 3-99) of that tidal volume was forced outwards the chest, in one or more episodes. There were on average two episodes (1; 1-4) of RF per ventilation. The mean

inspiration time and the mean expiration time was 1.2 s (0.5; 0.5-3.6) and 0.9 s (0.3; 0.4-2.3) respectively.

On average two (1; 0-5) chest compressions were superimposed on the inspiratory phase of the ventilation. Fifty-five percent of these chest compressions generated RF (Fig. 4B). Even though 45% of the chest compressions did not generate RF, these chest compressions reduced the magnitude of the inspiratory airflow by 71% (34; 0-100) on average at the moment of compression. The inspiratory airflow during the relaxation phase of the chest compression was on average 46.2 L/min compared to 13.3 L/min at the moment of the compression ($P<0.001$). The mean compression depth was 4.3 cm (0.7; 1.8-7.8). The mean compression rate was 104/min (8; 84-126). The mean ventilation rate and the mean minute volume were 18/min (6; 6-35) and 10.5 L/min (4.8; 4.4-22.1). The mean maximum inspiratory flow was 64 L/min (17; 23-124) and the mean peak inspiratory pressure was 40 cm H_20 (13; 16-83). The mean ventilation rate of 18/min was higher than the recommended 10 per minute in 22/25 patients. The mean tidal volume was higher than 600 ml in 19/25 patients.

There was no significant difference in compression depth between chest compressions associated with RF and chest compressions without RF. Ventilation pressure (Pvent) at the moment of a chest compression associated with RF was 22 cm H_2O on average compared to 18 cm H_2O in chest compressions without RF ($p<0.05$) (Fig 2B). Univariate analysis showed a low linearity between the volume of RF and compression depth ($r=0.18$; $R^2=0.03$; $RF= 0.013*CD+0.040$) and between the volume of RF and ventilation pressure at the moment of the chest compression ($r=0.16$; $R^2=0.03$; $RF=0.01*Pvent+0.082$).

No statistically significant difference was observed between patients who achieved ROSC and those who did not regarding volume and occurrence of RF, tidal volume, ventilation rate or minute volume (Table 1).

4. Discussion

To the best of our knowledge, this is the first report of a human study measuring RF and actually delivered minute volumes during ongoing chest compressions in OHCA. Our results show that in OHCA patients undergoing manual ventilation, chest compressions generated RF in most of the patients. Secondly, when RF did not occur, the magnitude of the inspiratory airflow was significantly reduced by chest compressions by 71% on average.

We observed wide variation in the number of episodes of RF between patients, ranging from 7% to 100%, as illustrated in Fig.4. There was also wide variation in volume of the RF between patients, ranging from 12 ml up to 364 ml. This variation might be the result of differences in chest compression depth and delivered ventilation volumes. Using linear regression, we were not able to show a clinical relationship between the volume of RF and both chest compression depth or ventilation pressure. Other factors such as chest wall mechanics, lung compliance or chest compression acceleration may influence the volume of RF. The current results regarding the occurrence and the volume of RF must however be interpreted within the constraints of potential shortcomings of this pilot study. Technical limitations allowed only a relatively low number of patients and short time period of available flow measurements, which might have an impact on the quality of the data.

We have shown that chest compressions may divide the ventilation flow into several smaller fragments (fig.3B). Although tidal volumes in this study may seem adequate, they were calculated as the sum of the individual inspiratory airflow fragments and do not accurately reflect true tidal volumes. This fragmentation of the ventilation flow may compromise gas exchange, when airflow fragments become smaller than the dead space, leading to carbon dioxide retention. For example, when a ventilation of 500 ml is split into five equal airflow fragments by chest compressions, then each fragment has a volume of 100 ml. If the RF episodes also have a volume of around 100 ml, then only dead space ventilation would occur. Dead space ventilation is most likely to occur when tidal volumes are small and when the RF phenomenon is excessive. Even when dead space ventilation occurs, gas transport mechanisms as described in high frequency ventilation (HFOV) may still generate some gas exchange. By means of end-tidal CO_2 measurements Deakin et al. demonstrated that chest compressions during compression-only CPR generated some gas exchange, even when these air volumes were smaller than the dead space.[19] It is probably unlikely that oxygenation would be compromised, as normal oxygenation can often be maintained with limited flow.[20,21]

Although interruptions of chest compressions cause rapid decline in coronary and cerebral perfusion, a large randomised trial could not demonstrate significant differences in survival rates or favourable neurologic function between OHCA patients treated by EMS providers with either continuous chest compressions with positive-pressure ventilation or chest compressions interrupted for ventilations at a ratio of 30:2. Oxygenation and ventilation were not measured in this trial. [22] It is possible that fragmentation of airflow as shown in our study counterweights the beneficial effects of continuous chest compressions on haemodynamics, partly explaining

the absence of a significant difference between the continuous compression group and the 30:2 ratio group. Pauses in chest compressions for ventilation may be less critical, and less detrimental for survival, than is currently believed.[23] So one might consider maintaining a 30:2 strategy after insertion of an advanced airway, to minimise fragmentation of airflow by chest compressions and to prevent excessive ventilation rates.

Alternative ventilation strategies have been suggested to optimise the interaction between continuous chest compressions and ventilations to enhance not only oxygenation and ventilation, but also haemodynamics, without compromising venous return. Examples are chest compression synchronised ventilation, decompression-triggered positive-pressure ventilation and passive continuous positive airway pressure ventilation.[11,24,25,26]

Chest compressions with simultaneous ventilations produce elevated peak inspiratory pressures. The average peak inspiratory pressures in our study were 40 cm H_20 and not as high as those found by O'Neill et al, who reported mean peak inspiratory pressures of 61 cm H_20.[7] The compressions depth in our study was 4.3cm on average, which is below the ERC standard and may have reduced peak pressures compared to chest compressions of more than 5 cm. Also we measured the pressures intratracheally, whereas O'Neill measured the pressures at the proximal end of the tube. O'Neill also measured the pressures in-hospital, after on average 43 minutes of CPR, when lung compliance may have been reduced and he used a LUCAS thumper to deliver chest compressions. It is believed that elevated peak inspiratory pressures may cause barotrauma, but there is a paucity of scientific evidence reporting pulmonary barotrauma in humans after ROSC.[27]

This is the first human study reporting maximum inspiratory flow during resuscitation. We measured a mean maximum inspiratory flow of 64 L/min, which is similar to the value of 61 L/min reported by Wagner-Berger et al during chest compressions in a manikin model.[28]

Several studies have demonstrated varying degrees of hyperventilation during resuscitation.[7,12,13] We confirm these findings, as we found a mean tidal volume of 690 ml, a mean ventilation rate of 18/min and a mean minute volume of 10.5 L/min. This is in agreement with the observations of O'Neill et al. who stated that hyperventilation appears to be caused by excessive ventilation rates rather than by high ventilation volumes and we demonstrate that this conclusion also applies to the prehospital setting.[7] Hyperventilation may result in increased intrathoracic pressures, decreased venous return, and cerebral vasoconstriction, which may be

hazardous.[12] These results underscore the importance of appropriate resuscitation training and real-time audio-visual feedback to improve the quality of resuscitation and patient outcomes.[29]

Our study has several limitations. It is a single centre study and the results may not be applicable to a larger population. The number of patients was small and only one minute of CPR per patient was analysed. We did not study the potential change of RF over time. Only manual ventilated patients were included in the study, as in all mechanical ventilated patients in PEEP was administered. The combination of PEEP and chest compressions creates a HFOV-like pattern, which will be the subject of further study. In this study airflow was calculated by means of tracheal pressure measurements, which needs validation, for instance, by comparison with synchronous spirometry.

5. Conclusions

Chest compressions have a significant effect on the ventilation flow during CPR, as chest compressions during ventilation generated RF in most patients. There was wide variation in the number of episodes and in the volume of RF between patients. Delivering simultaneous chest compressions and ventilations induces significant fragmentation of the airflow which may result in an overestimation of the delivered tidal volumes. The effect of RF on the efficacy of ventilation during resuscitation and on outcome remains unknown.

6. Acknowledgements

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CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest to declare.

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Legends to figures

1. Figure 1: External recording device consisting of two pressure sensors, two amplifiers and a logger (top) and the catheter system (bottom) for tracheal pressure measurements.

2. Figure 2: (A) Pressure waveform of a manual ventilation without chest compressions. The pressure difference between proximal and distal (=tracheal) measurement point allows the calculation of airflow. Pproximal= proximal pressure; Pdistal=distal pressure. (B) Pressure waveform of a manual ventilation with superimposed chest compressions. Pvent=ventilation pressure at the moment of compression.

3. Figure 3: (A) Airflow during a ventilation without chest compressions. (B) Airflow during a ventilation with four superimposed chest compressions. Every single compression during the inspiratory phase of the ventilation produces reversed airflow.

4. Figure 4: Bar chart showing (A) the percentage of ventilations with and without reversed airflow (RF) during CPR and (B) the percentage of chest compressions superimposed on a ventilation generating RF in each patient, illustrating the wide variation in occurrence between patients.

Figure 1

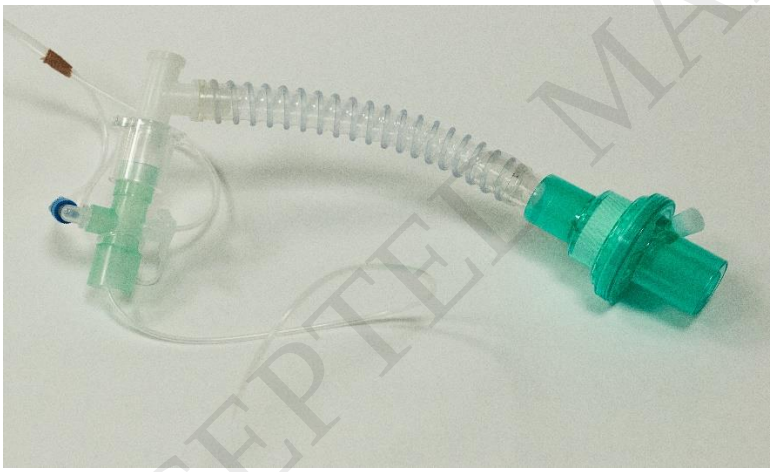


Figure 2

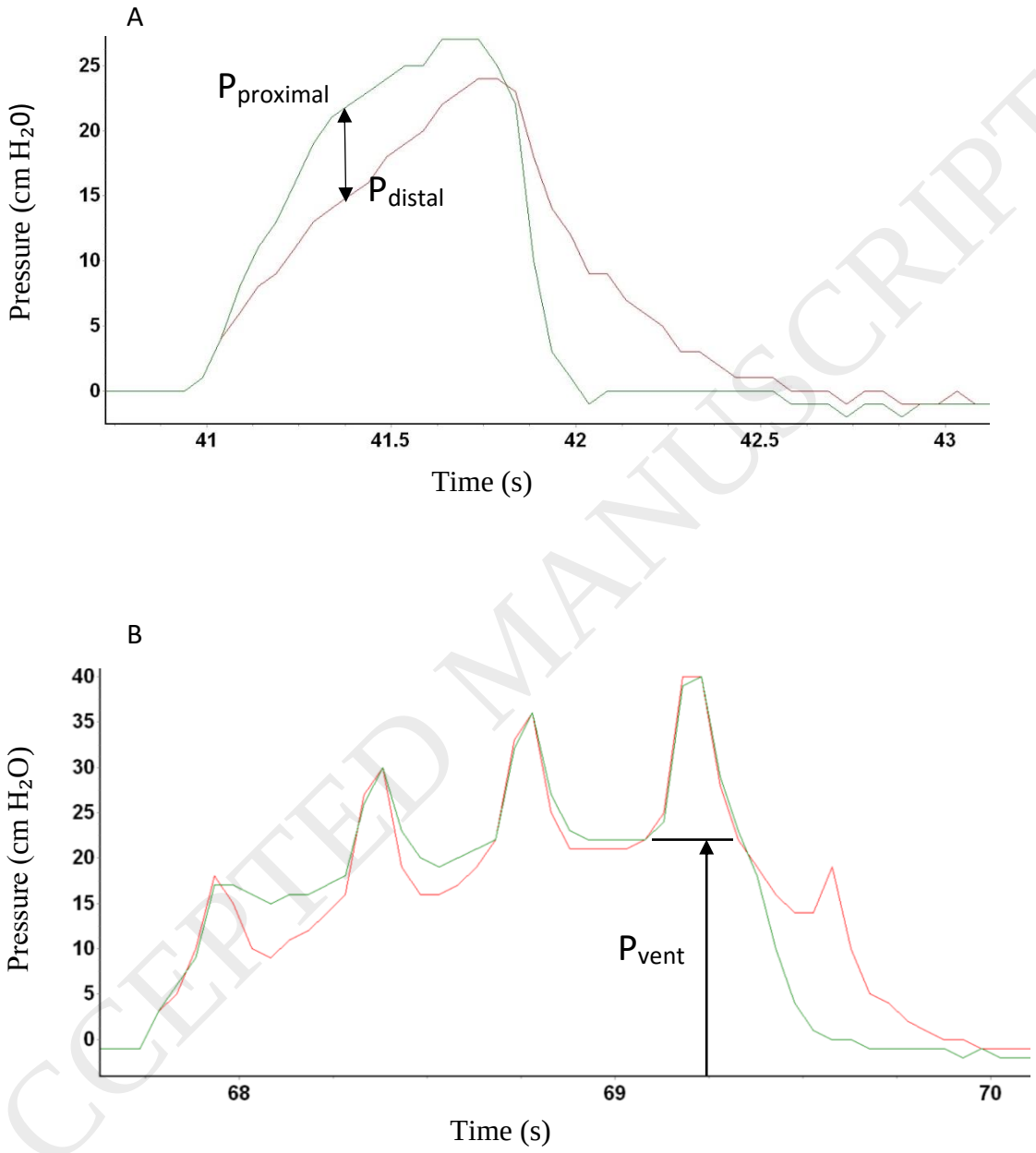
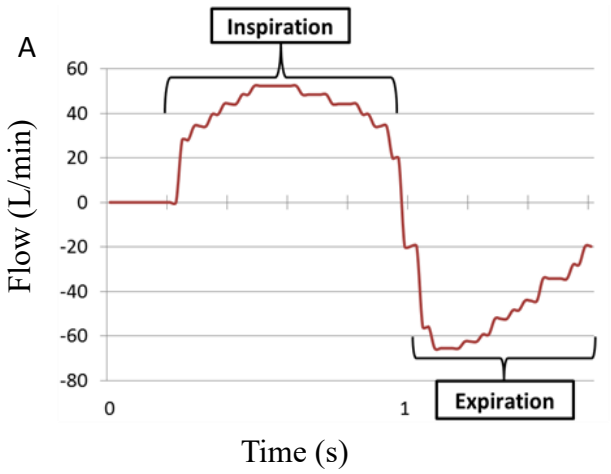


Figure 3



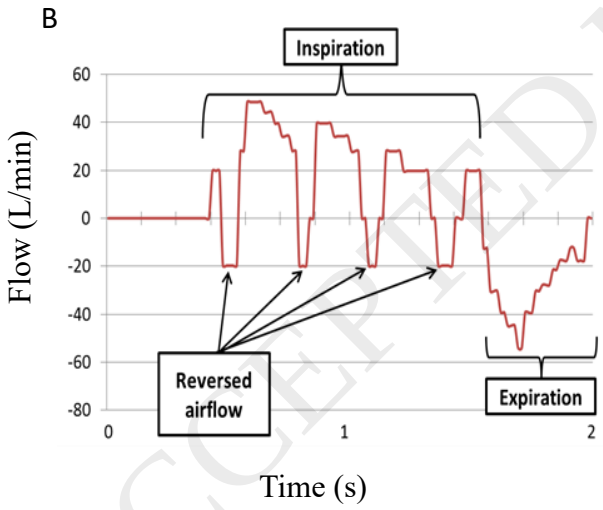
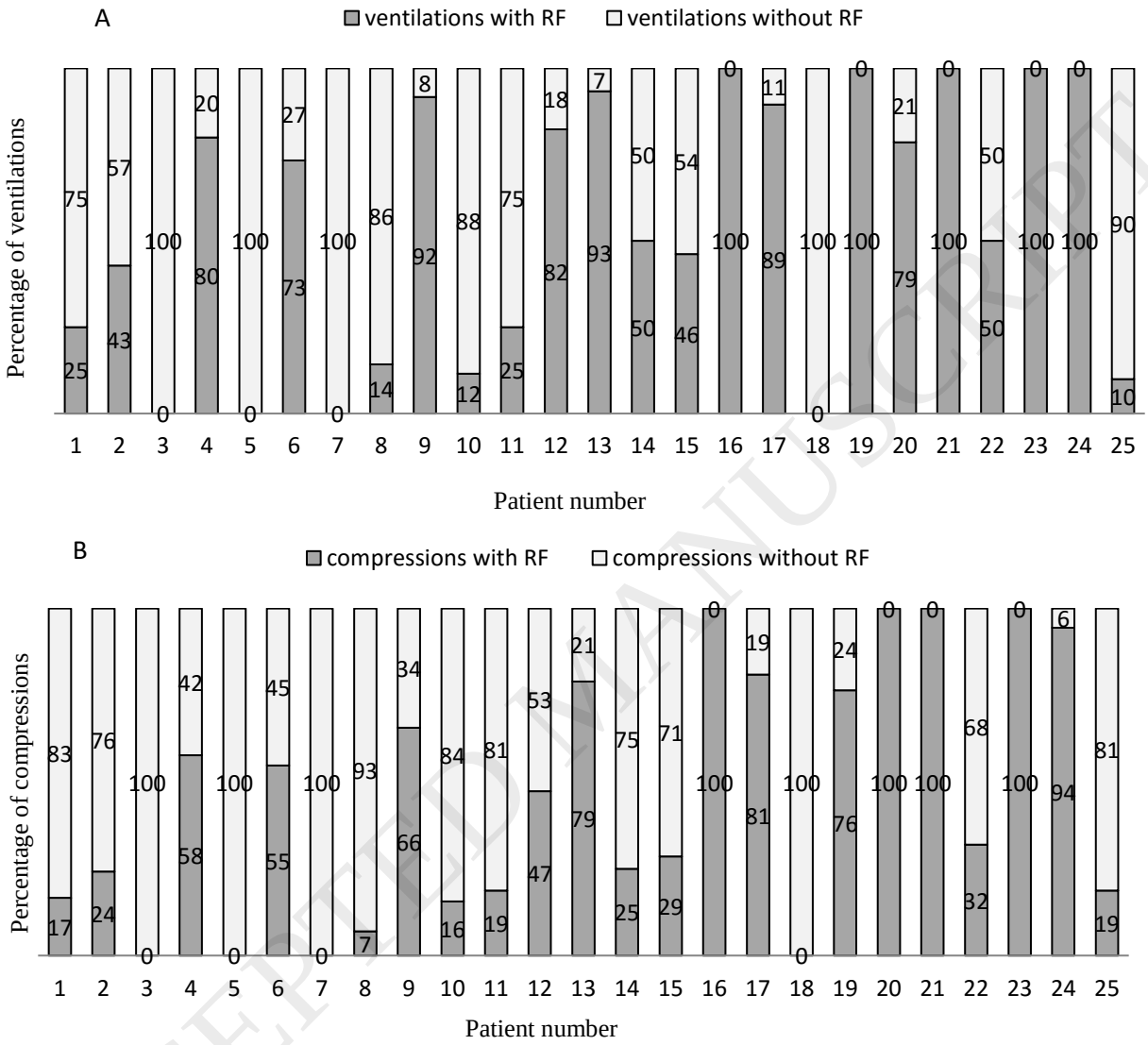


Figure 5



Table

Table 1: Relation between reversed airflow and return of spontaneous circulation (ROSC)

	ROSC	No ROSC	P-value
Number of patients (n, percentage)	8 (32%)	17 (68%)	
Volume reversed airflow (mL) (mean, SD)	104 (50)	92 (52)	0.11
Tidal volume (mL) (mean, SD)	643 (208)	713 (137)	0.06
Ventilation rate (per min) (mean, SD)	16 (4)	19 (9)	0.28
Minute ventilation (L) (mean, SD)	9.8 (3.8)	10.7 (5.3)	0.91