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Effects of Chest Compression on Ventilation Quality during Cardiopulmonary Resuscitation

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***Abstract*—Ventilation is an important part of cardiopulmonary resuscitation (CPR). The advanced airway mode and 30:2 mode are used for intubated and non-intubated patients, respectively. It is debatable that passive produced by 30 compressions can provide adequate tidal volume for 30:2 mode. In addition, the fragmented ventilation caused by continuous compression may result in ineffective ventilation. In the study, one pig was anaesthetized and intubated for 2 CPRs. Continuous chest compressions with ventilation and continuous chest compressions without mechanical ventilation were performed in 2 CPRs, respectively. Three 10-minute data segments including a period of normal ventilation (V segment), a period of only compressions without ventilation (C segment), and a period of compressions with ventilation (C-V segment) were used to analyze peek flow (PF), peek pressure (PP) and tidal volume. All the data was presented as mean ± standard deviation. Chest compression resulted in 14.90% increase in mean PP (2401.40 ± 94.75 Pa vs 2822.06 ± 291.10 Pa, p<0.05), 81.46% increase in average PF (319.58 ± 56.93 ml/s vs 579.92 ± 80.27 ml/s, p<0.05). The mean tidal volumes for C segment, V segment and C-V segment were 189.13 ml, 514.72 ml, and 429.26ml, respectively. Continuous compressions reduced the accumulative tidal volume, but when five compressions were made in one inspiratory phase, there is almost no loss of tidal volume (510.86 ± 47.24 ml vs 514.72 ± 29.25 ml, p<0.05). The study suggested the ventilator without feedback regulation might reduce the peek pressure during CPR and 5 compressions in 2 s inspiratory phase provided higher tidal volume.**

***Clinical Relevance*— This study shows that 150 chest compressions per minute provided greater tidal volume than 100 and 120 compressions per minute; continuous chest compressions could also provide a certain amount of oxygen supply.**

I. Introduction

Cardiopulmonary resuscitation (CPR) consists of chest compressions and ventilations used to treat sudden cardiac arrest to deliver oxygen to vital organs [1, 2]. High-quality CPR depends on both adequate cardiac output and adequate oxygen content [3, 4]. Unfortunately, the role of ventilation during CPR is unsettled because few studies measured and reported ventilation during CPR [5, 6]. The guidelines of the European resuscitation council recommend the use of intermittent positive pressure ventilation with a ventilation rate of 10 breaths for intubated patients, which is called advanced airway mode, and a compression ventilation ratio of 30:2 for these patients not intubated [7].

The 30:2 mode need a pause in external chest compression to oxygenate alveolar gas [8]. There is increasing evidence that interruptions to external chest compression are disastrous to outcome from cardiac arrest. Even short interruptions in compressions lead to a fall in vital organ perfusion [9, 10]. In past studies, passive ventilation was mentioned [11, 12]. In animal models of CPR, chest compressions have been shown to generate limited ventilation [3, 13, 14]. The advanced airway mode can provide uninterrupted compression and suitable ventilation. But the high airway pressure is the questions that have always been pointed out [15, 16]. The peak pressure (PP) was 17–24 cm H_2O on average in ventilations without chest compressions, however PP was 43–61 cm H_2O when chest compressions were superimposed on the ventilations [16]. In a recently retrospective study, the advantage of advanced airway mode in tidal volume was queried [17]. Simultaneous chest compressions and ventilation could cause reversed flow (RF) and severe disruption of the airflow, which can lead to an overestimation of tidal volume.

The aim of the study is to use parameters of peep pressure (PP), peak flow (PF), and tidal volume quantify the effects of compression on ventilation through pig trials.

II. Methods and Materials

A. Animal preparation

The experimental procedures were approved by the Institutional Review Board of Shandong University (KYLL-2020(KS)-340) and were in accordance with the Declaration of Helsinki. One Bama pig (weight, 31kg) fasted overnight was performed in the study. After the pig was placed supine in a U-shaped fixing frame, an intravenous catheter was inserted into a thigh vein and followed by propofol infusion to maintain anesthesia. A size 6.5 endotracheal tube was used to intubate. An animal ventilator (R419, Riward Life Science Co., Ltd, Shenzhen, China) used volume control ventilation mode to provide mechanical ventilation with a tidal volume of 500ml, a respiratory rate of 10 /min and a ratio of respiration of 1:2. Propofol was delivered to the pigs by ventilation to anesthetize the pig during the experiment. After induction of ventricular fibrillation and 4 minutes of stasis, CPR was started.

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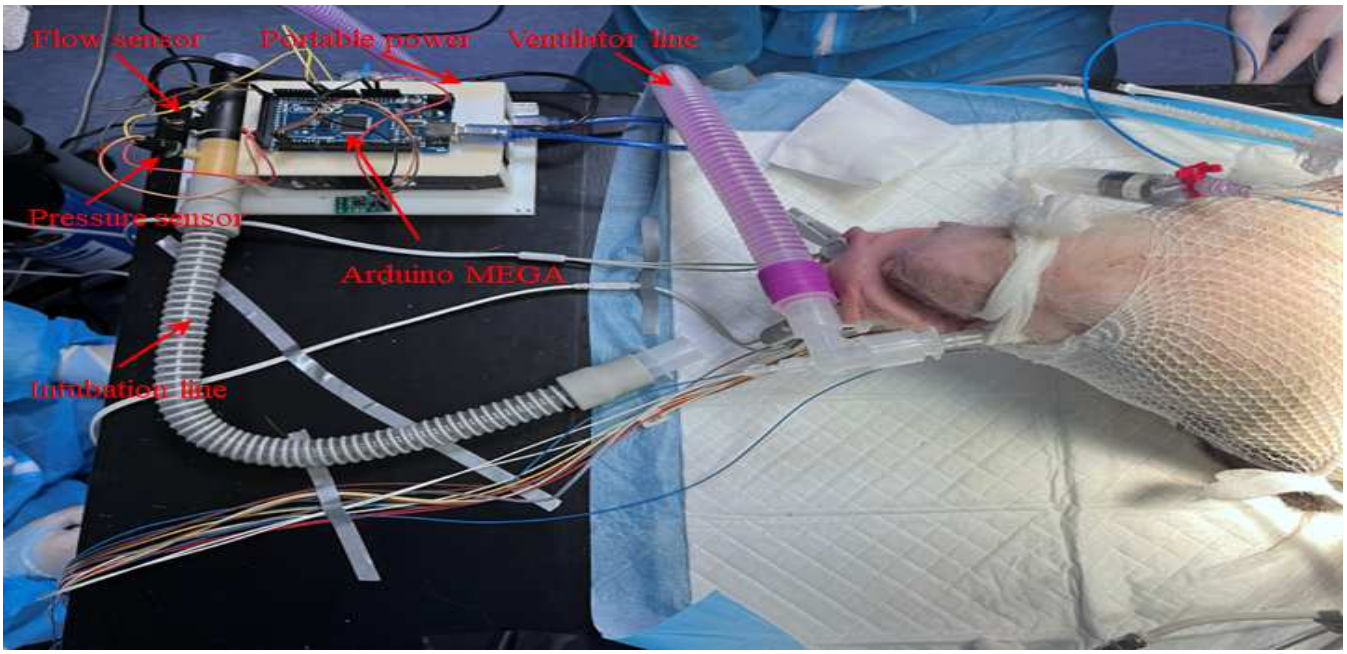

Fig.1. The self-designed recording device.

B. Data Acquisition

During the CPR of the pig, the pressure and flow in the airway was measured using a self-designed recording device (16.5 cm×8.0 cm×4.0 cm) (Fig. 1). The device consisted of one flow sensor (CAFS4000, Consensic electronics CO., LID, USA), one press sensor (XGZP6847A, Amiga electronic technology CO., LTD, Shenzhen, China), an Arduino MEGA (Version 3, LANDZO, China) and a portable power. The trachea of the pig was intubated with an endotracheal tube. After securing the airway, the one end of recording device was attached to the endotracheal tube and the other one end was attached to the ventilator. Once the CPR process is over, the recording was stopped. The use the recording device recorded multiple respiratory variables during CPR such as ventilation rate, peak inspiratory pressure, maximum inspiratory flow, inspiratory time, tidal volume.

C. Statistical analysis

We used the MATLAB to analyze the pressure and flow data. 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. Tidal volumes were derived from the flow signal for every single respiratory cycle. Cumulative inspiratory volume was calculated as the volume of the total airflow over the whole inspiratory phase; thus, tidal volumes due to chest compressions and ventilation. Independent sample T test was conducted between the data. Statistical significance was fixed at a P value of less than 0.05. We selected three 10-minute data segments, including a period of normal ventilation (V segment), a period of only compressions without ventilation (C segment), and a period of compressions with ventilation (C-V segment). For the C segment, we analyzed its tidal volume indicators every thirty compressions. All the data was presented as mean (standard deviation; range).

III. Result

A. Pressure Analysis

The pressure analysis results are presented in Fig. 2. (A). We compared the PP in V segment with the pressure in C-V segment (p<0.05). The mean PP was 2401.40 Pa (94.75; 2651.52-2236.07) for 81 ventilation events in V segment; The mean PP was 2822.06 Pa (291.10; 3506.84 -1942.82) for 94 ventilation events in C-V segment. External chest compression resulted in 14.90% increase in PP.

B. Flow Analysis

We compared the PF in C segment with the PF in C-V segment (p<0.05). There were 947 chest compressions in the C segment. The all PFs of RFs observed in every compression are shown in Fig. 2. (B), with an average PF of 319.58 ml/s (56.93; 207.15–459.68). For the C-V segment, we only calculated PFs in the inspiratory phase. The 351 PFs in all the inspiratory phase are shown in Fig. 2. A, with an average PF of 579.92 ml/s (80.27, 410.80-866.98). Mechanical ventilation resulted in 81.46% increase in average PF.

C. Tidal Volume Analysis

Tidal volume data are presented in Fig. 3. A total of 82 normal ventilation events with average tidal volume of 514.72 ml (29.25; 488.20-573.70) was included in the V segment. For the C segment, 30 compressions were used as one period to calculate tidal volumes. 31 periods of compression with average tidal volume of 189.13 ml (55.92; 74.77-299.64) was included in the C segment. For the C-V segment, there were 83 ventilation events with average tidal volume of 429.26ml (80.32; 660.22-254.36). The comparison between groups was significant (p<0.05). The effect of the number of compressions on tidal volume is shown in Fig. 4. In the C-V segment, average tidal volume was 343.74 ml (90.52; 505.07 - 259.29) for 13 ventilation events in which 3 compressions

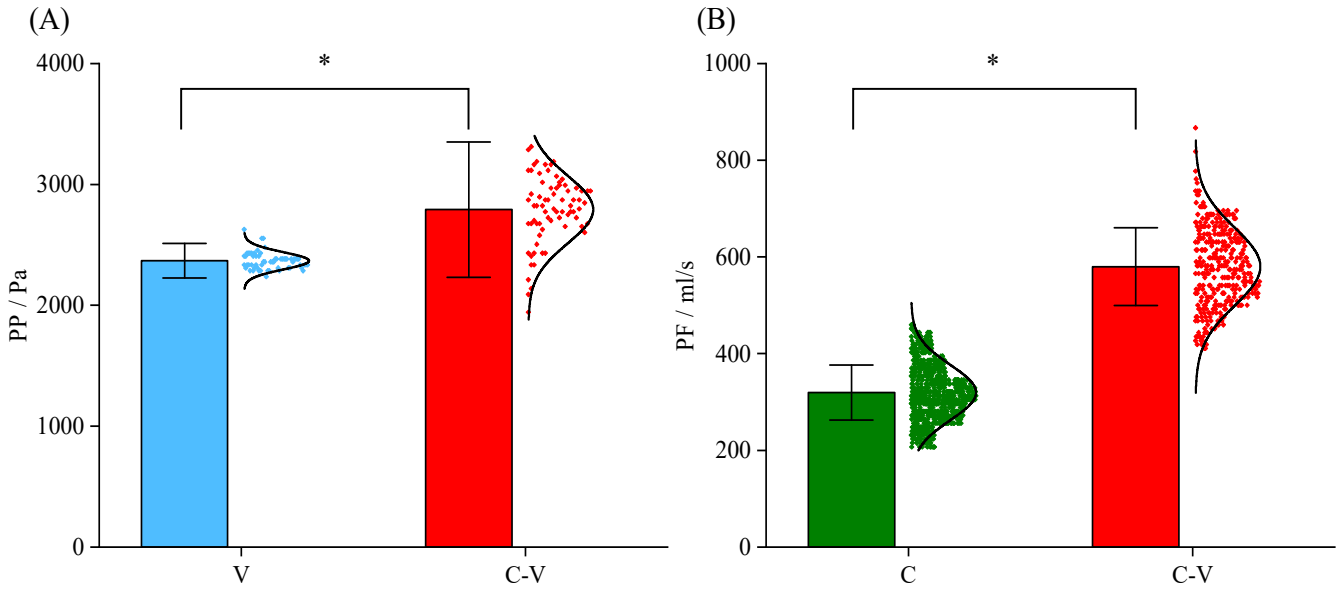

Fig.2. Effects of manual chest compressions on PP (peek pressure) and PF (peek flow). (A) The PP in V (normal ventilation) and C-V (compressions with ventilation) segment; (B) The PF in C (compressions without ventilation) and C-V (compressions with ventilation) segment.

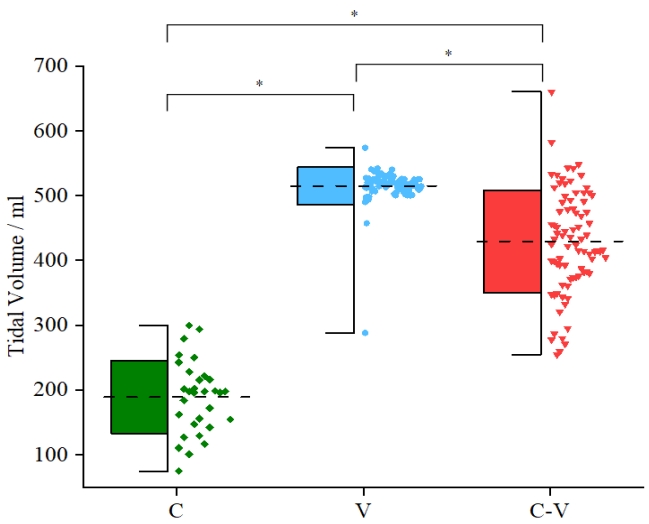

Fig.3. Effects of manual chest compressions on tidal volume, C expresses a period of only compressions, V expresses a period of normal ventilation, and C-V expresses a period of compressions with ventilation.

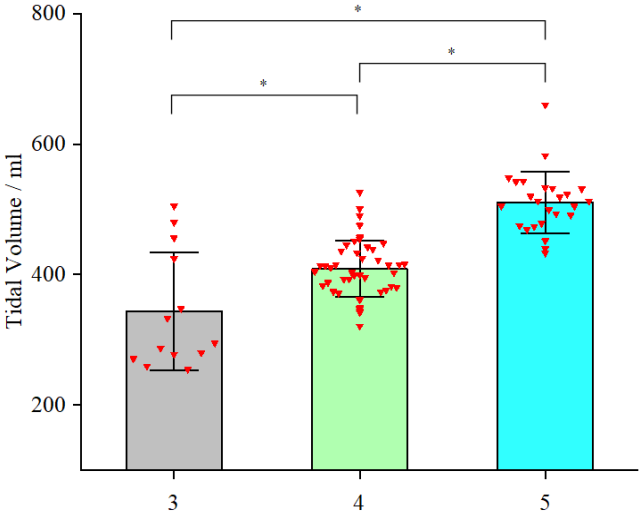

Fig.4. The effect of the number of compressions on tidal volume, 3, 4, and 5 represent three, four, and five compressions in one inspiratory phase, respectively

were completed for each inspiratory phase; average tidal volume was 408.52 ml (43.54; 526.26 - 320.57) for 45 ventilation events in which 4 compressions were completed in each inspiratory phase; average tidal volume was 510.86 ml (47.24; 660.22 - 432.97) for 25 ventilation events in which 5 compressions were completed in each inspiratory phase. The comparison between groups was significant ($p<0.05$).

IV. Conclusion

External chest compression resulted in 14.90% increase in PP. The mean PP is 2822.06 Pa (291.10; 3506.84-1942.82) in C-V segment. This compressions during ventilation only slightly increased PP values. The max PP (3506.84 Pa) in C-V segment is below 4000 Pa, which indicates there was no risk of barotrauma during compression [18]. The result is different from previous study in which the mean PP was 4000–6000 Pa [17]. Due to air tightness was checked before the start of the test, we believe that the difference in airway pressure comes from the specificity of the ventilator. The ventilator used in the experiment is a piston-type ventilator. The volume control ventilation mode is not modulated by feedback. Ventilators without feedback regulation may reduce the PP when chest compressions are superimposed on the ventilation.

The PF has significant change compared with the PP. Mechanical ventilation results in an 81.46% increase in PF during compressions. The PF parameter better indicates the occurrence of compression than the PP that is small and submerged in noise. The more pressure-sensitive feature of PF allows the PF to be used to record the number of compressions, which can complete automatic 30:2 ventilation.

The tidal volume of C segment was the result of passive ventilation. We recorded the accumulated tidal volume of 30 compressions to simulate the 30:2 ventilation, with average

tidal volume of 189.13 ml (55.92; 74.77-299.64). The result was similar to the previous study [14, 19]. But the accumulated tidal volume of 189.13ml for 30 passive ventilation designated that dead space ventilation would occur [11]. In some previous studies, high frequency ventilation might still generate some gas exchange when dead space ventilation occurred [20, 21]. The average tidal volume for the C-V segment was 429.26ml (80.32; 660.22-254.36). The tidal volume for normal ventilation was 514.72 ml (29.25; 288.20-573.70). In a ventilation, compression caused the tidal volume decrease 16.60%, but the tidal volume of 429.26 ml could still support sufficient oxygenation. In this study, compression divided a complete ventilation into 3 to 5 segments, but the segmented ventilation did not produce sufficient reverse tidal volume, which was different from recent study [17]. Conversely, from the Fig. 2. (B) chest rebound during compression resulted in the inhalation of more gas.

Uneven manual chest compression caused inspiratory phase to be divided into 2 to 5 numbers of segments. Segments containing only 2 compressions were considered failed compressions and were removed. Fig. 4 shows the effect of the number of compressions on tidal volume. When 5 compressions were completed in an inspiratory phase, the tidal volume (510.86) was approximate to the tidal volume of normal ventilation (514.72 ml); When 3 compressions were completed in an inspiratory phase, the tidal volume (343.74) was 66.78% of the tidal volume of normal ventilation (514.72 ml). In one inspiratory phase, a quicker compression rate will lead to more tidal volume.

V. Conclusion

By quantifying the pressure, flow, and tidal volume parameters, we suggested the ventilator without feedback regulation might reduce the occurrence of barotrauma during CPR, the PF was more sensitive than PP to compression and had potential to determine the occurrence of a compression event, and 5 compressions in 2 s inspiratory phase provided tidal volume approximating normal ventilation. The results of the study are limital due to the analysis of a single animal.

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