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Effects of Changes in Inspiratory Time on Inspiratory Flowrate and Airway Pressure during Cardiopulmonary Resuscitation: A Manikin-Based Study

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Objectives: Given that cardiopulmonary resuscitation (CPR) is an aerosol-generating procedure, it is necessary to use a mechanical ventilator and reduce the number of providers involved in resuscitation for in-hospital cardiac arrest in coronavirus disease (COVID-19) patients or suspected COVID-19 patients. However, no study assessed the effect of changes in inspiratory time on flowrate and airway pressure during CPR. We herein aimed to determine changes in these parameters during CPR and identify appropriate ventilator management for adults during CPR.

Methods: We measured changes in tidal volume (Vt), peak inspiratory flow rate (PIFR), peak airway pressure (Ppeak), mean airway pressure (Pmean) according to changes in inspiratory time (0.75 s, 1.0 s and 1.5 s) with or without CPR. Vt of 500 mL was supplied (flowrate: 10 times/min) using a mechanical ventilator. Chest compressions were maintained at constant compression depth (53 ± 2 mm) and speed (102 ± 2/min) using a mechanical chest compression device.

Results: Median levels of respiratory physiological parameters during CPR were significantly different according to the inspiratory time (0.75 s vs. 1.5 s): PIFR (80.8 [73.3 – 87.325] vs. 70.5 [67 – 72.4] L/min, $P < 0.001$), Ppeak (54 [48 – 59] vs. 47 [45 – 49] cmH_2O, $P < 0.001$), and Pmean (3.9 [3.6 – 4.1] vs. 5.7 [5.6 – 5.8] cmH_2O, $P < 0.001$).

Conclusions: Changes in PIFR, Ppeak, and Pmean were associated with inspiratory time. PIFR and Ppeak values tended to decrease with increase in inspiratory time, while Pmean showed a contrasting trend. Increased inspiratory time in low-compliance cardiac arrest patients will help in reducing lung injury during adult CPR.

Key Words: Airway pressure, Cardiopulmonary resuscitation, Inspiratory time, Mechanical ventilator

Globally, there has been a surge in coronavirus disease (COVID-19) cases, and COVID-19 patients have outnumbered healthcare systems in terms of the surge capacity during the COVID-19 pandemic. Healthcare systems have been facing burden by taking care of not only suspected and confirmed COVID-19 cases but also many critically ill patients with other diseases simultaneously.[1,2] In view of the COVID-19 pandemic, the safety of clinicians during cardiopulmonary

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resuscitation (CPR) has become concerning. CPR is known to be one of the aerosol-generating procedures, especially in high amounts, performed on patients.[3] This devastating pandemic has led to re-consideration of the risk–benefit balance for CPR. According to the American Heart Association interim guidelines, it is reasonable to reduce the number of providers involved in a resuscitation effort, for in-hospital cardiac arrest in COVID-19 patients or suspected COVID-19 patients.[4] Therefore, intubated patients should be placed on a mechanical ventilator (MV) equipped with a high-efficiency particulate air filter to maintain a closed circuit and reduce aerosolization.[4] Under such dreadful circumstances, the use of MVs during CPR has the advantage of reducing the workload of the medical staff and reducing the medical staff members' exposure of infectious diseases.

However, in real world, an increasing number of cardiac arrest patients are still using manual self-inflating bags rather than MVs owing to the fear about barotrauma generated with the use of MV. Additionally, there is a lack of evidence and experience about the use of MV during CPR and the difficulty in operating the MV machine. Moreover, no study has investigated the effect of changes in inspiratory time on the inspiratory flowrate and airway pressure during CPR.

This study aimed to measure changes in tidal volume (Vt), peak airway pressure (Ppeak), mean airway pressure (Pmean), and peak inspiratory flow rate (PIFR) with regard to inspiratory time of MV during CPR and to identify the appropriate ventilator method for adults during CPR.

MATERIALS AND METHODS

Simulation Design and Data Collection

To observe the physiological changes according to the changes in inspiratory time in MV use during CPR, we considered different inspiratory times (0.75 s, inspiration-to-expiration (I:E) ratio 1:7; 1.0 s, I:E ratio 1:5; and 1.5 s, I:E ratio 1:3). The 2015 International Consensus on Cardiopulmonary Resuscitation suggested the use of an inspiratory time of 1 s along with enough volume to produce a normal chest rise.[5] Therefore, we planned to compare the inspiratory time between 1 s and more and less. We used a CPR manikin (Resusci Anne Skill Reporter™, Laerdal, Stavanger, Norway). The lung compliance of the CPR manikin was set within the range of 0.022 – 0.03 L/cmH_2O, which was similar to the lung compliance measured in patients who had out-of-hospital cardiac arrest (OHCA) (0.022 L/cmH_2O).[6] An endotracheal tube (ETT) (Covidien, Dublin, Ireland), with an internal diameter of 7.0 mm, was connected to the artificial airway of the CPR manikin performed by the same emergency medicine doctor. The ETT was fixed at 21 cm. A flow analyzer (Flowanalyser™ PF-300, Imtmedical, Switzerland) was connected to the ETT to measure the changes in Ppeak, Pmean, PIFR, and Vt among the various inspiratory times in MV use. For constant ventilation support, an MV (H-C3, HAMILTON, Switzerland) was used under the following conditions:

volume-controlled mandatory ventilation, Vt of 500-mL, constant flow pattern, ventilation rate of 10 breaths/min, positive-end expiratory pressure of 0 cmH_2O, maximum trigger limit, and maximum pressure limit.[7]

To compare the physiological changes that occur during CPR, we divided the study into two groups (no CPR group and CPR group). In the CPR group, physiological changes were continuously measured during chest compression. A mechanical chest compression device (LUCAS2, Stryker Medical, 3800 E. Centre Ave. Portage, Michigan, USA) was used to maintain constant chest compression depth and rate (LUCAS2, mode: active continuous, chest compression rate 102 ± 2/minute, chest compression depth 53 ± 2 mm). Each group was continuously measured using the flow analyzer for 10 min.

Statistical Analyses

Between-group comparisons were conducted using the Mann–Whitney *U* test and Kruskal–Wallis test for continuous variables. Continuous variables are presented as medians with interquartile ranges. Bonferroni correction was used for multiple comparisons ($P < 0.05/3$ in the three groups). Data were analyzed using PASW/SPSS, version 22.0 (IBM Inc., Chicago, IL, USA). Significance level was set at $P < 0.05$.

RESULTS

In the no CPR group, compliance and resistance were 27 mL/cmH_2O and 18 cmH_2O/L/s, respectively. Constant ventilation volume was measured despite changes in inspiratory time (Table 1). The manikin used in this study is shown in Figure 1.

No CPR Group

PIFR (0.75 s: 40.8 L/min [40.5 – 41.2], 1.0 s: 31.15 L/min [30.1 – 31.8], and 1.5 s: 22.9 L/min [22.2 – 23.2]) and Ppeak (0.75 s mm: 34 cmH_2O [32 – 36], 1.0 s: 31 cmH_2O [24 – 33], and 1.5 s: 28 cmH_2O [24 – 29]) tended to decrease with increasing inspiratory time. However, Pmean (0.75 s: 3.15 cmH_2O [3 – 3.4], 1.0 s: 3.8 cmH_2O [2.9 – 3.9], and 1.5 s: 4.9 cmH_2O [4.2 – 5.1]) tended to increase with increasing inspiratory time (Fig. 2).

There was a significant difference when comparing inspiratory time (1.0 s vs. 1.5 s): PIFR (31.15 L/min [30.1 – 31.8] vs. 22.9 L/min [22.2 – 23.2], $P < 0.001$), Ppeak (31 cmH_2O

Table 1. Tidal volume of the simulation manikin according to inspiration time

	Tidal volume (ml)	
Inspiration time	no CPR	CPR
0.75s	500.5 (497 - 503)	480 (465.75 - 543)
1.0s	493 (493 - 505)	502 (461.5 - 544)
1.5s	499 (487 - 504)	496.5 (466 - 532)

CPR : cardiopulmonary resuscitation

Fig. 1. Manikin model : CPR manikin with airway circuit connected to the flow analyzer. A mechanical chest compression device is installed in the manikin.

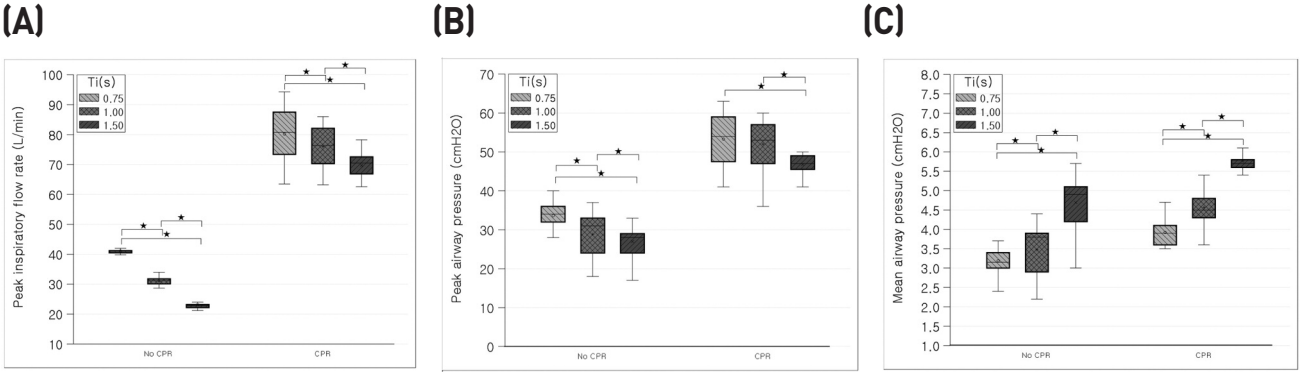

Fig. 2. Changes of the flow rate (A), peak airway pressure (B), and mean airway pressure (C) according to each inspiratory times (0.75 s, 1.0 s and 1.5 s). Bonferroni correction for multiplicity, $P < 0.05/3$.

[24 – 33] vs. 28 cmH_2O [24 – 29] $P = 0.009$), and Pmean (3.8 cmH_2O [2.9 – 3.9] vs. 4.9 cmH_2O [4.2 – 5.1] $P < 0.001$). Furthermore, a significant difference was observed when comparing other inspiratory time (0.75 s vs. 1.0 s): PIFR (40.8 L/min [40.5 – 41.2] vs. 31.15 L/min [30.1 – 31.8], $P < 0.001$), Ppeak (34 cmH_2O [32 – 36] vs. 31 cmH_2O [24 – 33], $P < 0.001$), and Pmean (3.15 cmH_2O [3 – 3.4] vs. 3.8 cmH_2O [2.9 – 3.9], $P = 0.002$) (Fig. 2).

CPR Group

The CPR group showed the same result as the no CPR group. PIFR and Ppeak tended to decrease with increasing inspiratory time in the CPR group: PIFR (0.75 s: 80.8 L/min [73.3 – 87.325], 1.0 s: 76.5 L/min [70.8 – 82.1], and 1.5 s: 70.5 L/min [67 – 72.4]), Ppeak (0.75 s: 54 cmH_2O [48 – 59], 1.0 s: 53 cmH_2O [48 – 57], and 1.5 s: 47 cmH_2O [45 – 49]). However, Pmean tended to increase with increasing inspiratory time in the CPR group, with Pmean (0.75 s: 3.9 cmH_2O [3.6 – 4.1], 1.0 s: 4.5 cmH_2O [4.3 – 4.8], and 1.5 s: 5.7 cmH_2O [5.6 – 5.8]).

There was a significant difference when comparing the inspiratory time (1.0 s vs. 1.5 s): PIFR (76.5 L/min [70.8 – 82.1] vs. 70.5 L/minute [67 – 72.4], $P < 0.001$), Pmean (4.5 cmH_2O [4.3 – 4.8] vs. 5.7 cmH_2O [5.6 – 5.8], $P < 0.001$). Furthermore, a significant difference was observed when comparing the inspiratory time (0.75 s vs. 1.0 s): PIFR (80.8 L/min [73.3 – 87.325] vs. 76.5 L/min [70.8 – 82.1], $P < 0.001$) and Pmean (3.9 cmH_2O [3.6 – 4.1] vs. 4.5 cmH_2O [4.3 – 4.8], $P < 0.001$) (Fig. 2).

A significant difference was observed in Ppeak between 1.5 s and the other inspiratory times: Ppeak (1.0 s vs. 1.5 s: 53 cmH_2O [48 – 57] vs. 47 cmH_2O [45 – 49], $P < 0.001$; and 0.75 s vs. 1.5 s: 54 cmH_2O [48 – 59] vs. 47 cmH_2O [45 – 49], $P < 0.001$). However, between the inspiratory times 0.75 s and 1.0 s, there was no significant difference in Ppeak (0.75s vs. 1.0s: 54 cmH_2O [48 – 59] vs. 53 cmH_2O [48 – 57], $P = 0.153$) (Fig. 2).

DISCUSSION

During chest compression, ventilation is an important part of high-quality CPR.[8-10] Several investigations have demonstrated that adequate ventilation during CPR has a strong association with coronary perfusion pressure (CPP) and return of spontaneous circulation.[4,11,12] For this reason, various monitoring methods, such as metronome, thoracic impedance, capnography, and tracheal pressure monitoring, have been proposed in previous studies to assess adequate ventilation and prevent excessive ventilation.[13-17] However, it is difficult to maintain adequate ventilation during CPR. Inadvertent hyperventilation occurs commonly during CPR, excessive positive-end pressure is created, leading to increased intrathoracic pressure and peak airway pressure,[18,19] which could decrease survival rates by reducing venous return, cardiac output, and CPP.[20-23] High airway pressure and high tidal volume are known to be associated with lung injury. When the peak airway pressure is 50 cmH_2O or higher, it is suggested that the peak airway pressure causes progressive impairment in lung function and leads to acute respiratory failure in the animal model.[24,25] Therefore, it is important to maintain a constant ventilation volume and appropriate peak airway pressure during resuscitation. Use of MV can be a good alternative to manual self-inflating bag ventilation.

Previous studies about the use of the automatic transport ventilator showed better outcomes than using self-inflating bag resuscitator,[26,27] and one study using a manikin model showed that an MV

was superior to a self-inflating bag resuscitator for maintaining adequate ventilation and adequate peak pressure during chest compression.[28] According to Lee et al., who included patients undergoing one-lung ventilation for thoracic surgery,[29] prolonged inspiratory time on gas exchange decreased Ppeak but increased Pmean, similar to that observed in our study; this also resulted in a modest improvement in oxygenation and decreased shunt fraction during one-lung ventilation, although this study was not conducted in cardiac arrest patients.

In our study, the longer the inspiratory time, the lower the peak pressure. A peak pressure of 1.5 s was seen as a significant difference when comparing the inspiratory times 0.75 s and 1.0 s, almost kept below 50 cmH_2O. Therefore, in cardiac arrest patients with poor lung compliance, the option of increasing the inspiratory time will prevent lung injury.

Pmean was increased with increasing inspiratory time. Although the increase in Pmean may be associated with reducing venous return to the right heart and thereby decreasing the hemodynamic effectiveness of CPR, the level of Pmean is not high (0.75 s: 3.9 cmH_2O [3.6 – 4.1], 1.5 s: 5.7 cmH_2O [5.6 – 5.8] and the impact is likely to be minimal.

The limitation of this study is the use of a manikin model has clear differences compared to normal human being. Wright et al[30] showed that the difference in resistance, which was higher in a manikin than in a human, derived from having a tissue rigidity different from that of the human beings. This is because First, the branching structure of the airways and the irregular surface of the large airways cause more turbulence flow. Secondly, the ET tube may be deformed at body temperature or partial occlusion caused by secretion. Thirdly, the tube tip may also be pushed against tracheal wall or tending to bend tube with changes in head and neck position Therefore, animal study also may be an appropriate alternative.

In conclusion, changes in PIFR, Ppeak, and Pmean varied according to the inspiratory time. As the inspiratory time increased, PIFR and Ppeak tended to decrease, and Pmean tended to increase. Therefore, the use of increased inspiratory time in low compliance cardiac arrest patients will help in reducing lung injury during adult CPR. An adequate MV mode for human CPR needs to be identified in further studies.

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