Utilization of a model lung system to assess the effects of an inspiratory impedance threshold valve on the relationship between active decompression and intra-thoracic pressure

Atsushi Sugiyama a,*, Keith G. Lurie b, Yoshikane Maeda c, Yoshioki Satoh a, Mutsuaki Imura d, Keitaro Hashimoto a

a Department of Pharmacology, Yamanashi Medical University, Tamaho-cho, Nakakoma-gun, Yamanashi 409-3898, Japan
b Cardiac Arrhythmia Center, University of Minnesota, Minneapolis, MN 55455, USA
c Department of Critical Care Medicine, Yamanashi Medical University, Tamaho-cho, Nakakoma-gun, Yamanashi 409-3898, Japan
d IMI Co. Ltd., Koshigaya, Saitama 343-0824, Japan

Received 16 March 1999; received in revised form 9 July 1999; accepted 9 July 1999

Abstract

Use of an inspiratory impedance valve has recently been shown to increase the efficacy of standard and active compression-decompression cardiopulmonary resuscitation. We evaluated the effects of a prototypic impedance valve on the relationship between the active decompression and intra-thoracic pressure using a mechanical model lung system. Intermittent impedance to inspiratory flow of respiratory gases during the decompression phase of active compression-decompression cardiopulmonary resuscitation significantly decreased the intra-thoracic pressure, while the pressure was almost zero cm H_2O during the cardiopulmonary resuscitation cycle when the impedance threshold valve was not functioning. Thus, this mechanical model will help in evaluating the valve as well as in optimizing its function by simulating various forms of lung disease. © 1999 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Active compression-decompression; Impedance; Lung

1. Introduction

Active compression-decompression (ACD) cardiopulmonary resuscitation (CPR) increases venous blood return by enhancing the decrease of intra-thoracic pressure during the decompression phase [1,2]. Recently, a new inspiratory impedance threshold valve (ITV) was developed to induce intermittent impedance of inspiratory gases during CPR, which will further enhance the overall efficacy of standard and ACD-CPR [3,4]. A porcine model of ventricular fibrillation has been used to evaluate the valve [3,4]; however, this model can be influenced by multiple conditions during the experimental period and may be less flexible to simulate the clinical chest pathology.

In this study, we adopted a mechanical model lung system to test the effects of a prototypic ITV on the relationship between active decompression and intra-thoracic pressure. This model mimics the function of human respiratory system under normal physiological as well as pathological states, including the altered chest wall compliance [5,6]. In this paper, we describe the evaluation of the ITV by the model lung system to begin to develop this new approach.

2. Materials and methods

Experiments were carried out using an inspiratory ITV (Resusci-valve ITV™, CPRX LLC, Min-
The principle of the valve is to completely occlude the endotracheal tube when the pressure in the intra-tracheal pressure is below atmospheric pressure [3,4]. In practice, when the patient was actively ventilated with the ventilator bag, oxygen is passed through the fenestrated mount of the valve during insufflation directly into the endotracheal tube without any significant impedance. When active ventilation is not performed, the silicone diaphragm occludes the airway whenever the intrathoracic pressure is $< -0 \text{ cm H}_2\text{O}$, that is, during the active decompression phase. During exhalation and during the compression phase of CPR, respiratory gases pass in the reverse direction pushing the silicone diaphragm out of the way without any resistance by the valve. The prototype is a one-way valve designed to occlude the airway when intrathoracic pressures are less than atmospheric pressures. When the intrathoracic pressure exceeds $-40 \text{ cm H}_2\text{O}$, a threshold check valve opens to permit inflow of inspiratory gases. A yes/no stopcock valve can be used to bypass the occluding channels completely.

The compliance of the model lung was set in a normal physiological range of 0.2 l/cm H$_2$O. The left chamber was used to obtain the ACD-CPR power on the right chamber. The left endotracheal tube of the model lung was connected to the ventilator and both sides of the chamber of the model lung were connected each other using a metallic plate. After the start of the ventilation of the left chamber, the right chamber was compressed and decompressed by the metallic plate connecting both sides of the chamber. The intra-thoracic pressure was estimated using the pressure gauge of the model lung. Meanwhile, the flow-volume and pressure at the right endotracheal tube were continuously and simultaneously monitored using a D-lite flow sensor (Datex Ohmeda, Finland) positioned between the ITV and the right endotracheal tube.

ACD-CPR was performed at the rate of 30 and 80/min. The effect of the active decompression in volumes of 50, 60, 70, 80, 90, 100, 150, 200, 250, 300, 400, 500 and 600 ml on the intra-thoracic as well as intra-tracheal pressures was examined. The experiment was repeated 6 times at each active decompression volume in the presence and absence of inspiratory ITV function.

The data are presented as the mean ± SE. The statistical comparisons of mean values were evaluated by one-way repeated-measures ANOVA. A $P$-value $< 0.05$ was considered significant.
3. Results

The effects of ITV at the simulated chest compression rate of 80/min were essentially the same as those obtained at 30/min, indicating that the mechanical model can be used at various rate of CPR. The pressure in the right endotracheal tube was exactly the same as that of the right intra-thoracic pressure during the whole ACD-CPR cycle. Total CPR duration was less than 15 min in the present experiment. Typical experiment showing the relationship between the active decompression volume of right chamber and the pressure in the right endotracheal tube, when the decompression volume and CPR rate were set at 150 ml and 30/min, respectively, is depicted in Fig. 2A. The relationship between maximal active decompression volume and intra-thoracic peak pressure is summarized in Fig. 2B. In the absence of the ITV function (no), intra-thoracic pressure was almost zero cm H₂O during the ACD-CPR cycle and no significant change was detected in the intra-thoracic pressure at any active decompression volume. Meanwhile, in the presence of the valve function (yes), the intra-thoracic pressure was always below 0 cm H₂O. The peak pressure became more negative with increasing the volume of the active decompression. While the valve was closed, actually moved volume of air through the valve was zero. Since the threshold valve was opened at –40 cm H₂O, the intra-thoracic pressure never decreased to below –40 cm H₂O even at larger active decompression volume of >500 ml and it was kept at –40 cm H₂O during the active decompression phase.

4. Discussion

The present study clearly demonstrated that the intermittent impedance to inspiratory flow of respiratory gases during the decompression phase of ACD-CPR can significantly decrease the intra-thoracic pressure, while the pressure was almost zero during ACD-CPR cycle when the ITV was not functioning. Thus, the ITV will further lower the intra-thoracic pressure in cardiac arrest patients, which may lead to the improvement of over all ACD-CPR efficacy [3,4].

The current model lung system may offer some advantages to better understand the relationship between changes in intrathoracic pressure with ACD-CPR. The compliance of the bellow of the model lung can be determined by the distance from the pivot, where the compliance spring are set [5]. The flow resistance in each lung depends on the diameter of the tube chosen for the bronchus [5]. Since the chest wall becomes less compliant in certain status and with aging and potential value of ACD-CPR is highly dependent on the chest wall compliance and diaphragmatic tone [4], the mechanical model lung system may provide a very useful tool to evaluate the ITV and to determine ‘optimal’ threshold valve cracking pressures based upon patient status [5]. Experiments are now on-going to evaluate the pressure required by the ventilator on the left side bellow which will produce the desired changes in intrathoracic pressure on the right side bellow of the model with various pathological states.
In conclusion, this mechanical model will help in evaluating the valve as well as in optimizing its function by simulating various forms of lung disease, moreover the current approach may allow one to understand under what circumstances the valve is unlikely to add any significant benefit.

References


