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Short Communication

A dose-response curve for the negative bias pressure of an intrathoracic pressure regulator during CPR

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Abstract

An intrathoracic pressure regulator (ITPR) is a device that can be added to the external end of an endotracheal tube to create controlled negative airway pressure between positive pressure ventilations. The resulting downward bias of the airway pressure baseline promotes increased venous return and enhanced circulation during CPR and also during hypovolemic shock. In the present study we exercised a mathematical model of the human cardiopulmonary system, including airways, lungs, a 4 chambered heart, great vessels, peripheral vascular beds, and the biomechanics of chest compression and recoil, to determine the relationship between systemic perfusion pressure during CPR and the value of baseline negative airway pressure in an ITPR. Perfusion pressure increases approximately 50 percent as baseline airway pressure falls from zero to –10 cm H_2O. Thereafter perfusion pressure plateaus. Negative bias pressures exceeding –10 cm H_2O are not needed in ITPR-CPR.

Key words: Airway management, ACD-CPR, Cardiopulmonary resuscitation (CPR); Coronary perfusion pressure; Decompression, Device; Impedance, Mathematical model, Threshold, Valve

1. Introduction

A recurring theme of resuscitation research since the year 1980 has been the improved perfusion pressure and forward flow that can be obtained during cardiac arrest and CPR if methods are implemented to improve filling of the chest pump. One such method is interposed abdominal compression (IAC-CPR) in which manual compression of the abdomen between chest compressions encourages venous return to the right heart[1-3]. A complementary method is active compression-decompression (ACD-CPR) in which a plunger-like device is used to expand the anterior chest in between chest compressions to create negative pressure within the chest that encourages venous return to the right heart[4-6]. A third strategy is the use of an impedance threshold device (ITD) placed on the end of the endotracheal tube to prevent passive inflow of air between chest compressions, so that normal expansion of the anterior chest during recoil between compressions will generate a modest negative pressure to encourage venous return to the right heart[7, 8]. Clinical studies of cardiac arrest and CPR demonstrate improved survival when chest pump filling is augmented either by application of positive abdominal pressure[9-11] or negative intrathoracic pressure[12-14] between chest compressions during otherwise standard CPR. The most recent such strategy along these lines combines an ITD with an external vacuum line to create a negative bias pressure in the airway between positive pressure breaths. This system is known as the intrathoracic pressure regulator (ITPR) and can generate even greater negative intrathoracic pressure between chest compressions than does natural chest recoil, and in turn even greater venous return to the right heart[15]. This short paper addresses the question of the dose-response relationship for negative bias

pressure with an ITPR: that is, how much can be gained in terms of flow by adjusting the negative bias in airway pressure?

2. Methods

To study the benefit side of the risk-benefit equation, we employed a validated mathematical and computer model of the adult human circulation recently developed by one of us (CFB) to simulate the effects of an impedance threshold valve. Full details of the model have been recently published in ***Resuscitation*** and are not repeated here[16]. In this model it is a simple matter to vary the mouth pressure to create simulations of ITPR-CPR for various bias pressures. It is also a relatively simple matter to vary mouth pressure over time to simulate alternative patterns of positive pressure rescue breathing, including particular compression to ventilation ratios, such as 15:2 or 30:2, which are being discussed currently for the purpose of improving average blood flow and oxygen delivery during one-rescuer CPR[17-20]. Here we exercise the previously described model to create dose-response curves for baseline negative airway pressure in an ITPR and also to explore the effects of proposed alternative compression to ventilation ratios.

3. Results

Figure 1 illustrates average systemic perfusion pressure over one minute of cardiac arrest and CPR as a function of ITPR bias pressure in the computational model of Babbs[16]. The frequency of chest compression was always 100/min, and chest compressions were not stopped for ventilations, since with ITPR-CPR one may assume that an endotracheal tube is in place and the mechanical restrictions of single rescuer CPR do not apply. Compression to ventilation ratios of 15:2 and 30:2 were compared. Each pair of ventilations was administered over a period of 4.5 sec. The positive airway pressure during ventilations was 54 cmH_2O (40 mmHg) as in[15]. Inspiratory time for each ventilation was 1.1 sec. The curves in Figure 1 show that there is benefit to increasing negative bias pressure in the airway. However, as bias pressure is made more negative than –10 cmH_2O there are diminishing returns. Compared to current standard 15:2 compression to ventilation CPR at 100/min, the 30:2 compression to ventilation ratio diminished systemic perfusion pressure only slightly for negative bias pressures exceeding –10 cmH_2O. This level of bias pressure causes systemic perfusion pressure in the model to increase from 21 to 32 mmHg, in very good agreement with the steady state results of Yannopoulos et al. in a porcine model[15].

4. Discussion

In conventional standard CPR the generation of negative intrathoracic pressure between chest compressions depends upon the intrinsic elastic recoil of the chest wall, which is diminished both with age and with the presence of broken ribs. Moreover, many rescuers may fail to allow the chest wall to recoil fully[21, 22], especially at faster compression rates

such as 100/min. The ITPR offers a relatively easy solution to maximize venous return in CPR for patients who are endotracheally intubated.

The present model studies suggest that near maximal benefit of ITPR-CPR occurs with a negative bias pressure of –10 cmH_2O in the external airway. There is little effect of the compression to ventilation ratio upon this result when chest compression is not interrupted for ventilation. In this case positive pressure ventilations increase both right atrial pressure and thoracic aortic, so systemic perfusion pressure remains about the same. When there is interruption of chest compression for ventilations, a 30:2 ratio allows for significant improvement of vital organ perfusion pressures and blood flow compared to a 15:2 ratio in animal models of CPR[23, 25]. Although the present methodology does not directly address risk and safety, since mathematical models are indestructible, there is indirect evidence for maximal benefit/risk at –10 cmH_2O. This is because animal studies have shown no evidence of harm with –10 cmH_2O, while benefit rises steadily to just this level in our model and then plateaus. Hence benefit/risk should peak near –10 cmH_2O and then decline. The ability of the ITPR to improve systemic perfusion pressure with a trivial modification of CPR protocols (placing a short extension, approximately 5 x 3 x 3 cm in size on an endotracheal tube) appears to offer potential benefit and negligible risk.

5. Conflict of interest statement

One of us (DY) has published jointly with several employees of Advanced Circulatory Systems Inc., a company currently developing the intrathoracic pressure regulator, and has been involved in the evaluation and testing of early prototypes. There are no financial connections between DY and Advanced Circulatory Systems.

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Figure and legend

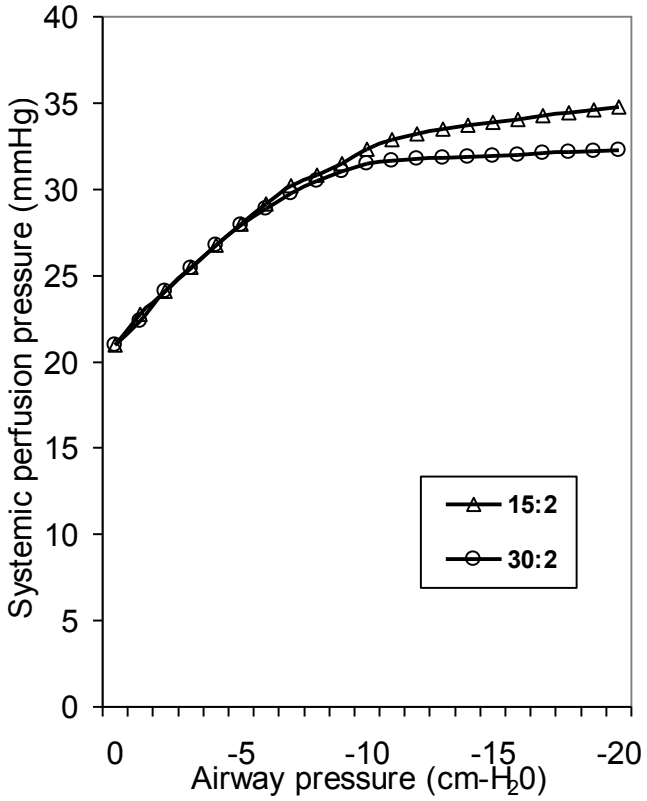

Figure 1. Systemic perfusion pressure during ITPR-CPR for various negative airway pressures with 100 chest compressions per minute and two regimes of positive pressure ventilations (15:2 and 30:2). Systemic perfusion pressures are average values for first 60 sec of CPR after cardiac arrest. Thoracic pump factor = 0.75. The abscissa indicates negative airway pressure.