

Short communication

# Use of an inspiratory impedance threshold valve during cardiopulmonary resuscitation: a progress report

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## Abstract

Building upon studies on the mechanism of active compression-decompression (ACD) cardiopulmonary resuscitation, a new inspiratory impedance threshold valve has been developed to enhance the return of blood to the thorax during the decompression phase of CPR. Use of this device results in a greater negative intrathoracic pressure during chest wall decompression. This leads to improved vital organ perfusion during both standard and ACD CPR. Animal and human studies suggest that this simple device increases cardiopulmonary circulation by harnessing more efficiently the kinetic energy of the outward movement of the chest wall during standard CPR or active chest wall decompression. When used in conjunction with ACD CPR during clinical evaluation, addition of the impedance valve resulted in sustained systolic pressures of greater than 100 mmHg and diastolic pressures of greater than 55 mmHg. The new valve may be beneficial in patients in asystole or shock refractory ventricular fibrillation, when enhanced return of blood flow to the chest is needed to 'prime the pump'. The potential long-term benefits of this new valve remain under investigation. © 2000 Elsevier Science Ireland Ltd. All rights reserved.

*Keywords:* Inspiratory impedance threshold valve; Cardiopulmonary resuscitation; Active compression-decompression (ACD)

## 1. Introduction

A number of new mechanical devices have been developed in recent years to improve the presently dismal outcomes for patients in cardiac arrest [1,2]. One new approach, active compression-decompression (ACD) CPR, utilizes a suction device to push down and pull up on the chest [1–6]. This is the first device that was designed to enhance blood return to the thorax by decreasing intrathoracic pressure during the decompression phase of

CPR. Use of this technique results in both an increase in positive intrathoracic pressure during the compression phase and an increase in negative intrathoracic pressure during the decompression phase. Clinical studies have consistently demonstrated an improvement in hemodynamic parameters with ACD CPR [1–5]. However, short- and long-term survival studies have been less consistent [5,7].

In the process of studying the mechanism of ACD CPR, we realized that intermittent yet complete occlusion of the airway during the chest wall decompression phase would result in a greater decrease in intrathoracic pressure and more blood return to the chest [8–11]. By occluding the airway

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during chest wall decompression when active ventilation is not being performed, negative intrathoracic pressure was enhanced and equilibration of the negative intrathoracic pressure-extrathoracic pressure gradient was achieved by an increase in blood flow into the thorax. Based upon this mechanism, a small inspiratory impedance threshold valve (Fig. 1a,b) was developed to occlude the



(a)



(b)

Fig. 1. (a) The inspiratory impedance threshold valve (Resusci-Valve ITV™ CPRxLLC, Minneapolis, MN) functions by occluding the airway during the decompression phase of CPR. When actively ventilating the patient during ACD CPR, there is no resistance to inspiration. However, when not actively ventilating the patient during CPR, the impedance valve prevents inspiration of respiratory gases during chest decompression, thereby lowering intrathoracic pressure. Expiration remains unimpeded at all times. (b) The valve is shown connected to an tracheal tube and a ventilation bag. Should the patient begin to breath spontaneously, the valve must immediately removed from the ventilation circuit.

airway intermittently during chest decompression without resistance to exhalation or active rescuer inhalation [1,2,8–10]. During chest wall decompression in the apneic patient, the impedance valve selectively impedes inspiratory gas exchange while allowing for unimpeded exhalation. This valve can be used with standard CPR, ACD CPR, and other methods of CPR including vest CPR, interposed abdominal counterpulsation CPR, and phased thoracic-abdominal compression-decompression CPR.

The inspiratory impedance valve has been tested in both animal models and patients in cardiac arrest [8–10]. It has been shown to increase vital organ blood flow significantly when used with either standard CPR or ACD CPR in a porcine model of ventricular fibrillation. In the animal model, it was found to reduce defibrillation energy thresholds when used together with ACD CPR [8]. More recently the new valve has been tested in patients undergoing ACD CPR. In those studies, patients randomized to the inspiratory valve group had significantly higher end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>), systolic, diastolic, and coronary perfusion pressures, and a shorter time to return of spontaneous circulation compared with patients treated with ACD CPR alone [10]. The purpose of this report is to review the animal and human experimentation of the inspiratory impedance valve during CPR to date, focussing on the mechanisms, potential improvements and limitations, and the future potential utility of this new device.

## 2. Mechanisms of action

### 2.1. General principles

An increase in negative intrathoracic pressure during the decompression phase of CPR can enhance both ventilation and return of venous and arterial blood into the thorax. Recently, this important mechanism has been rigorously studied [1–12]. The chest, during CPR, functions like a bellows. During standard CPR the natural recoil of the chest during the decompression phase of CPR is the principal driving force behind the return of blood to the thorax. While this may vary from patient to patient, the decrease in negative intrathoracic pressure from chest wall decompression during standard CPR provides for only mar-

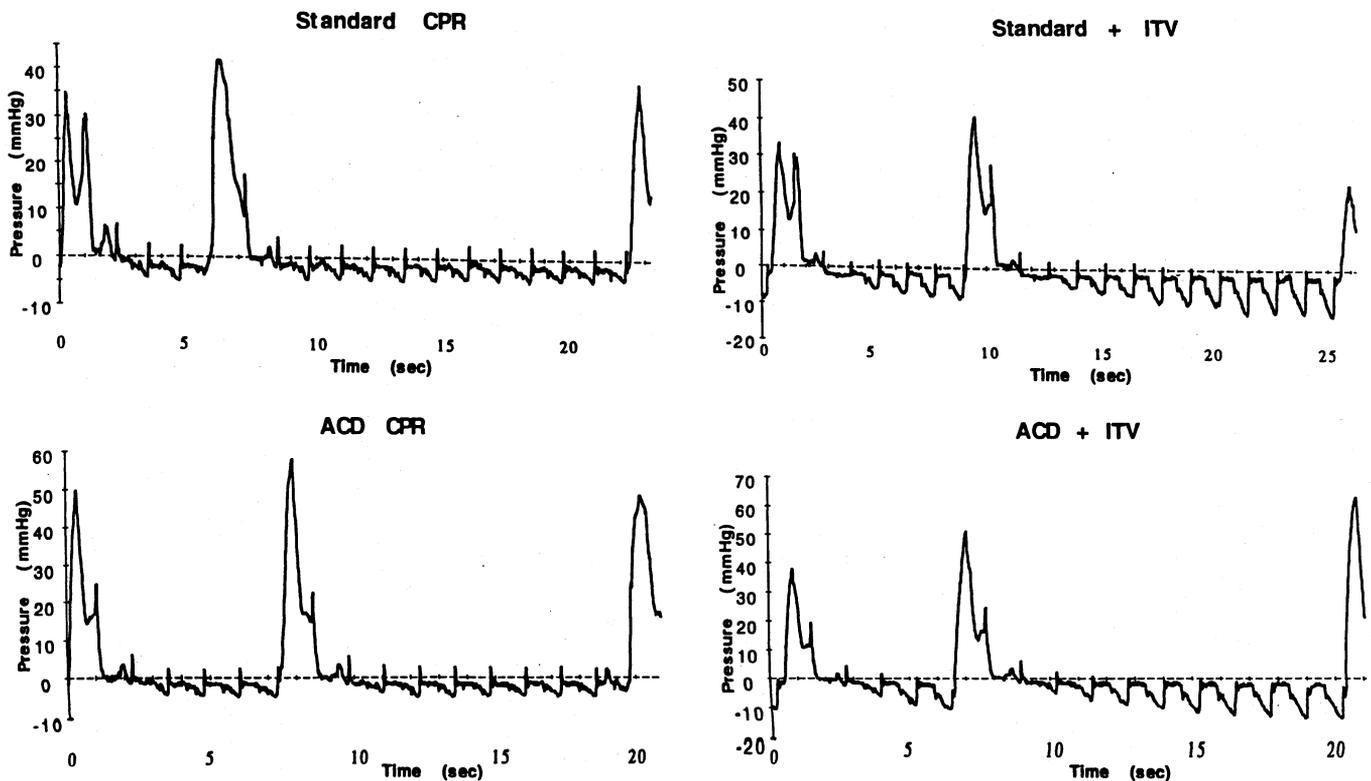


Fig. 2. Representative intrathoracic pressure changes observed when increasing the compression:ventilation ratio from 5:1 to 10:1 during standard (STD) CPR alone (Fig. 2a), standard CPR plus the impedance valve (ITV) (Fig. 2b), active compression-decompression (ACD) CPR alone (Fig. 2c), and ACD CPR plus ITV (Fig. 2d). In the presence of the valve, intrathoracic pressure becomes progressively more negative over time after each active ventilation.

ginal levels of blood return. Consequently, over time, forward blood flow out of the chest decreases secondary to decreasing venous return of blood to the chest.

Studies of the mechanism of standard and ACD CPR led to the realization that changes in intrathoracic pressures were relatively modest when the airway was maintained in the open position by a tracheal tube [11]. Measurement of esophageal pressure, as a reflection of intrathoracic pressures, and intratracheal pressures from a transducer placed at the distal end of a tracheal tube, demonstrated that the intrathoracic pressure rises from 5–25 mmHg during the compression phase of standard CPR [11]. During chest wall decompression, intrathoracic pressure fell to approximately  $-5$  mmHg with standard CPR in both a pig model of CPR and an acute model of ventricular fibrillation in patients [9,11]. While this slight decrease in intrathoracic pressure may seem trivial, it is sufficient to propel some movement of venous blood from the periphery back into the thorax.

Mueller has previously described the effects of inspiration while maintaining a closed glottis in awake patients [13]. The Mueller maneuver results in an enhancement of venous blood return to the chest and provides a clinical tool during the assessment of certain cardiac murmurs. During this maneuver, inspiratory effort results in a marked reduction in intrathoracic pressure when inspiratory gas exchange is prevented by closure of the trachea at the glottis. In the apneic patient, the Mueller maneuver is not possible. However, by using other approaches to 'inhale' and decrease intrathoracic pressure, such as active compression-decompression CPR, the Mueller maneuver can be mimicked despite the absence of active ventilatory effort by the patient. It is this principle that is further exploited by the impedance threshold valve. In this case, the impedance valve functions like the closed glottis but only during the decompression phase.

Moreover, during cardiac arrest and cardiopulmonary resuscitation, the respiratory gases are actively pushed out of the thorax with each chest

compression phase. In addition to the Mueller mechanism, we have observed that with inspiratory resistance the amount of respiratory gas within the thorax decreases over time when chest compressions are performed. Consequently, intrathoracic pressure decreases in a progressive manner over time following each active ventilation (Fig. 2). The decreases in intrathoracic pressure are not observed with either standard CPR alone or active compression-decompression CPR alone. With progressively less respiratory gas in the chest, the thorax may act as a more efficient bellows. As such more blood returns into the chest when the valve is used. Based upon these observations, it is likely that less frequent active ventilations, in the presence of an impedance threshold valve, will result in a more efficient flow of blood back into the chest during the decompression phase of CPR. Consequently, the preload to the heart is enhanced resulting in a more efficient delivery of blood to the vital organs during CPR. However, it is important to emphasize that regular rescuer ventilation must be performed, but the 'optimal' ratio of compressions to ventilation is not yet known.

### 2.2. 'Gasping' reflexes and the impedance valve

One intriguing and new potential application of the inspiratory impedance valve is to block inspiratory gas exchange during 'gasping' in the immediate post arrest period. 'Gasping' has been associated with improved outcomes in patients, and has been thought to be of benefit secondary to the resultant increase in ventilation [14–17]. We have recently observed that use of the impedance threshold valve in 'gasping' pigs after induction of ventricular fibrillation appears to also function like a Mueller maneuver. The decrease in intrathoracic pressure is markedly augmented by the impedance valve, resulting in a significant increase and resetting of arterial blood pressure after each 'gasp' (Figs. 3 and 4). Thus, the impedance valve may be of potential benefit to help patients with a gasping reflex to augment blood return to the chest, in the absence and presence of manual chest compressions.

### 2.3. Use with positive expiratory pressure

More recently, small levels of positive expiratory pressure have been used to further enhance overall CPR efficacy when performing CPR with the impedance valve [18]. While positive expiratory

pressure during performance of CPR has been shown to enhance overall CPR efficacy in hypervolemic animal models, it has been traditionally thought to have a potential negative effect on overall CPR efficacy [19]. Positive expiratory pressures can result in a slight increase in intrathoracic pressure during the compression phase but also may inhibit venous return. However, recent data suggests that when venous return is enhanced with either ACD CPR alone or in the presence of the inspiratory impedance valve and either standard or ACD CPR, then low levels of positive expiratory pressure result in both improved alveolar gas exchange (higher arterial  $p_{O_2}$  content) as well as an increase in coronary perfusion pressures. When a CPR technique that enhances venous blood return to the chest is used in combination with low levels of positive expiratory pressure, this results in a reduction in pulmonary atelectasis. We speculate that with increased blood flow through the pulmonary vasculature the lungs become less compliant. As a consequence of the venous return enhancing techniques of CPR, low levels of positive expiratory pressure enhance the cardiac compression mechanisms and decrease capillary leakage into the interstitium (i.e. reduce pulmonary edema). Thus, with each compression phase the increased blood volume in the pulmonary vasculature results in greater cardiac compression due to the decreased compliance of the lung and also prevents the build-up of interstitial fluid secondary to capillary leak. As such, the potential beneficial effects of this 'pneumatic pump' mechanism of CPR, when continuous positive expiratory pressure is applied during performance of CPR with 'venous-enhancing techniques', may be analogous to those observations made in a hypervolemic model of cardiac arrest [19].

## 3. Description of the impedance threshold valve

The one-way valve was designed to close when intrathoracic pressure is less than atmospheric pressure. Thus, as soon as the chest wall recoils after the compression phase of CPR, the silicone diaphragm occludes the lumen within the valve (Resusci-Valve ITV™, CPRxLLC, Minneapolis, MN), thereby preventing all inspiratory gas exchange. Flow of gases through the valve is shown in Fig. 5. In an awake patient, the valve impedes inspiration. Given the possibility that during the-

rescue effort a patient will regain spontaneous respiratory efforts, a small pop-off valve is attached to the side of the inspiratory impedance valve. If the patient actively inspires the check valve will open at  $-22$  cm  $H_2O$ . The opening pressure of the valve can be reduced if the valve is anticipated to be used with standard CPR. Inspiration by the patient would therefore be possible albeit with some difficulty. The opening pressure of  $-22$  cm  $H_2O$  was chosen based upon observations that negative intrathoracic pressures approach  $-20$  cm  $H_2O$  in patients undergoing ACD CPR [11]. Moreover, the pressure of  $-22$  cm of  $H_2O$  is sufficiently low to reduce the chances of developing negative pressure pulmonary edema should a patient regain consciousness and begin breathing spontaneously. Sim-

ilar findings were observed when a model test lung was used to evaluate the impedance valve [20].

The impedance valve is designed to fit standard ventilation bags and to attach to a tracheal tube, face mask, laryngeal mask airway, Combitube™, or other airway adjuncts. It also contains a bacteriostatic filter. While most studies in animals and in patients are performed after tracheal intubation, we have observed in normal volunteers that the impedance valve functions well and the seal is adequate when it is used in combination with a face mask (Fig. 6). It is anticipated that the impedance valve may be used with a mouthpiece and face mask to improve the efficiency of mouth-to-face mask ventilation when chest compressions are performed concurrently.

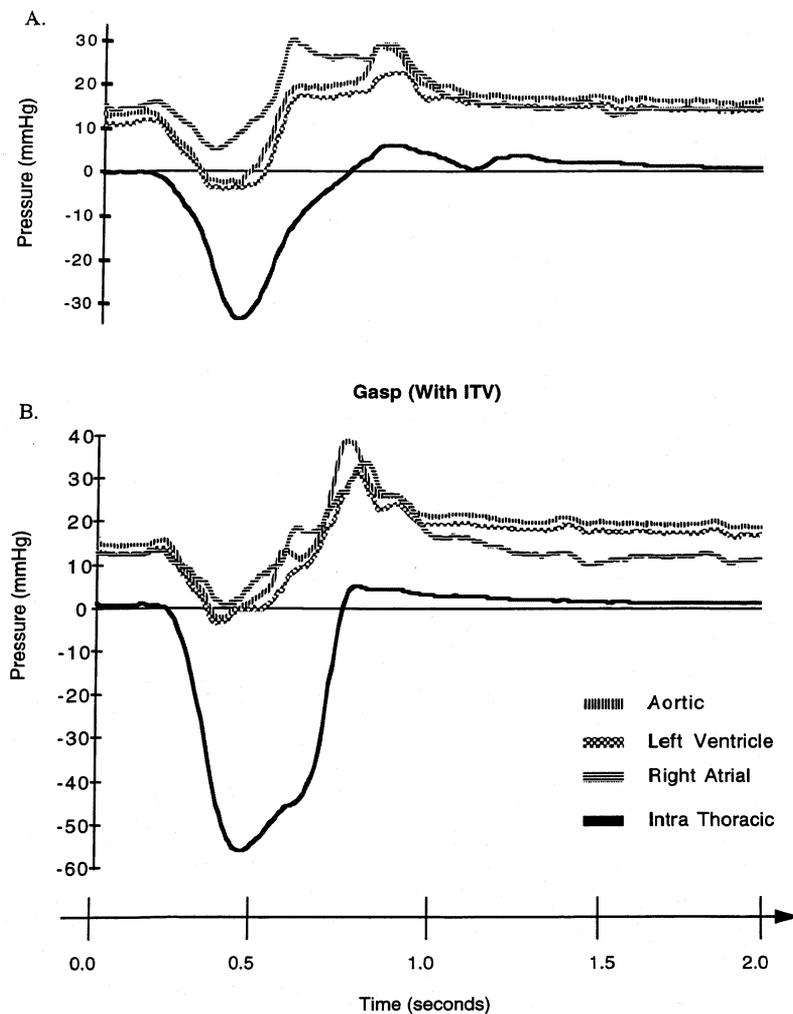


Fig. 3. A representative spontaneous gasp is shown from a pig in ventricular fibrillation in the absence (A) and presence (B) of the inspiratory impedance valve (ITV) in the absence of active chest compressions. Intrathoracic pressures measured in the trachea at the level of the carina, and ascending aortic, left ventricular, and right atrial pressures, were obtained with electronic micromanometer-tipped (Miller) catheters. In this case, the gasp with the valve in place was obtained 60 s after the gasp without the valve and 3 min after induction of ventricular fibrillation.

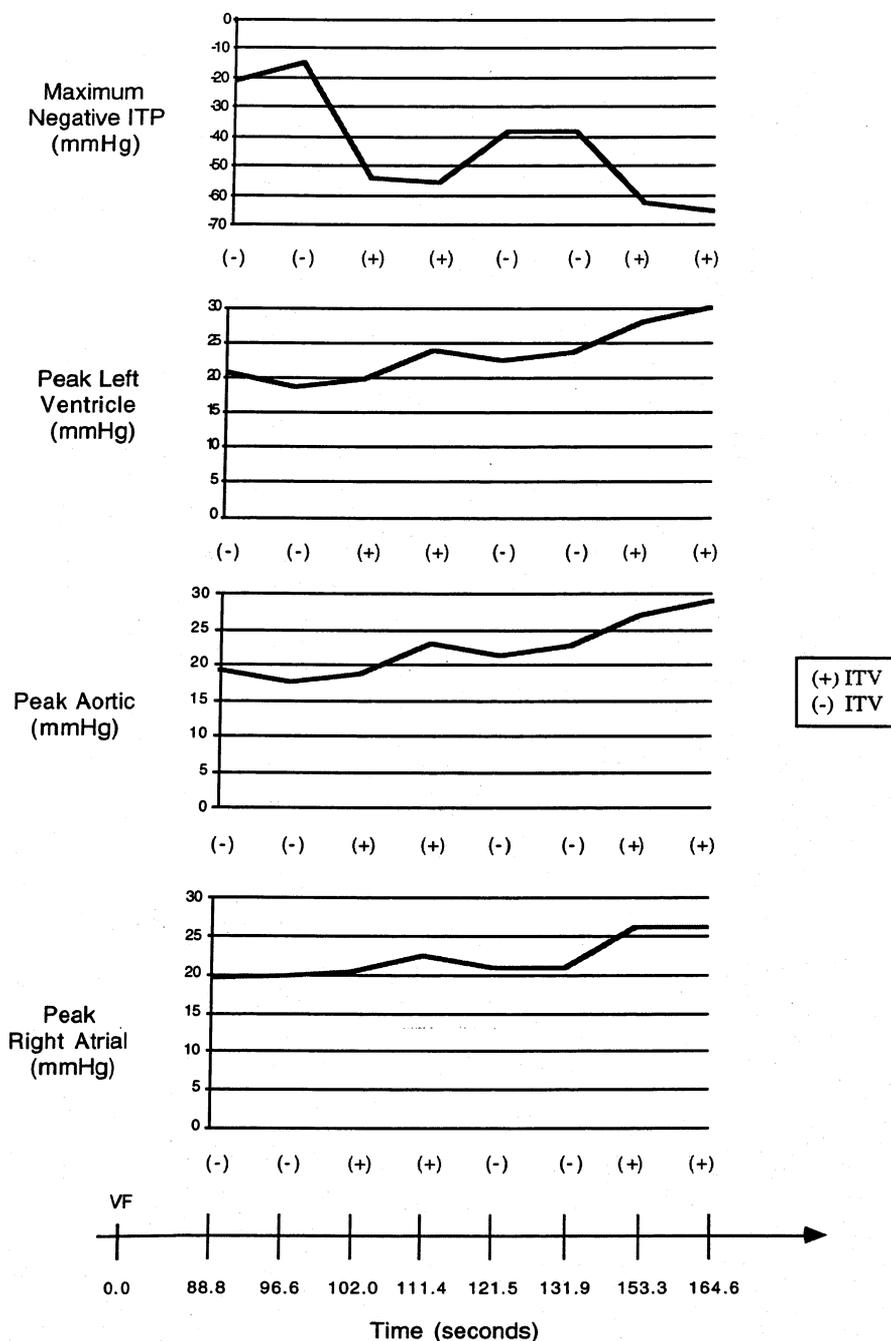


Fig. 4. The relationship between peak aortic, left ventricular, right atrial, and intrathoracic pressures over time from a representative pig that was spontaneously gasping after induction of ventricular fibrillation. Use of the impedance threshold valve (ITV™) was associated with lower intrathoracic pressure and higher peak aortic pressure after each spontaneous gasp.

#### 4. Animal studies

The first investigation of the concept of inspiratory impedance during CPR was performed with two 20-cm PEEP valves placed in reverse in a standard respiratory circuit [8]. In these studies, ACD CPR was performed in a well established

porcine model of ventricular fibrillation. The importance of enhancement of blood return to the thorax by enhancing negative intrathoracic pressure was clearly demonstrated in these studies. The addition of the inspiratory impedance valve concept resulted in a marked enhancement of vital organ perfusion and a decrease in the total energy

required for effective defibrillation [8]. In this study, cerebral perfusion during ACD CPR with the impedance valve was similar to that observed in the baseline state when the animals were not in ventricular fibrillation. Similarly, myocardial perfusion increased from 35% to greater than 65% of baseline flows. All animals which received the ITV™ during ACD CPR could be resuscitated after 19 min of ventricular fibrillation. Moreover, significantly less energy was needed for successful defibrillation in the valve groups.

Following the studies with ACD CPR with inspiratory impedance, another series of studies was performed with standard CPR with the impedance

valve [9]. The differences between the control and valve groups during standard CPR were similar to those observed with ACD CPR with and without the valve [8,9]. However, due to the overall lower baseline blood flow observed with standard CPR it has been more challenging to demonstrate the value of the impedance valve concept with standard CPR. The protocol that demonstrated the advantages of using the impedance valve during standard CPR most effectively was one in which the valve was added or removed in a sequential manner in the same animal during the performance of CPR [9]. While vital organ perfusion and coronary perfusion pressures showed a trend to-

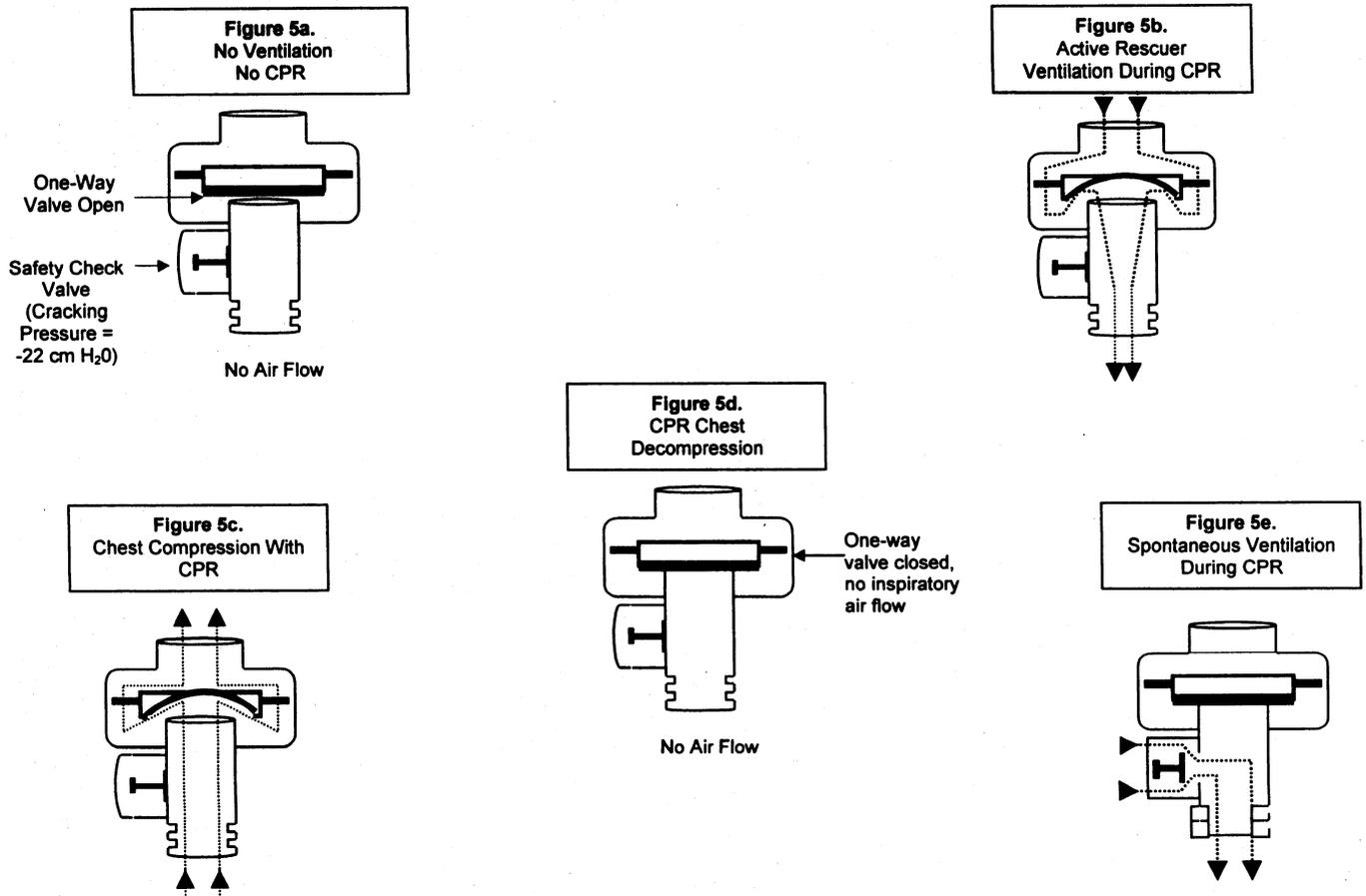


Fig. 5. Respiratory gas flow during CPR performed with the inspiratory impedance valve is illustrated. The impedance valve includes a silicone diaphragm that provides impedance in inspiration during the decompression phase of CPR. A safety check valve enables respiratory gas exchange upon return of spontaneous ventilation. When CPR is not being performed the one-way valve remains open, the safety check valve remains closed and no airflow passes through the valve (Fig. 5a). During active rescuer ventilation, respiratory gases bypass the occlusion (the silicone diaphragm) and flow into the lungs unimpeded (Fig. 5b). Similarly, when chest compressions are performed air bypasses the silicone diaphragm and flows out of the lungs unimpeded (Fig. 5c). During chest decompression, however, the one-way valve closes impeding inspiratory airflow thus lowering intrathoracic pressure within the chest (Fig. 5d). Upon the return of spontaneous ventilation the safety check valve opens when intrathoracic pressure reaches a threshold of  $-22$  cm H<sub>2</sub>O allowing air to bypass the occlusion so that the patient may breath freely spontaneously through the valve (Fig. 5e).

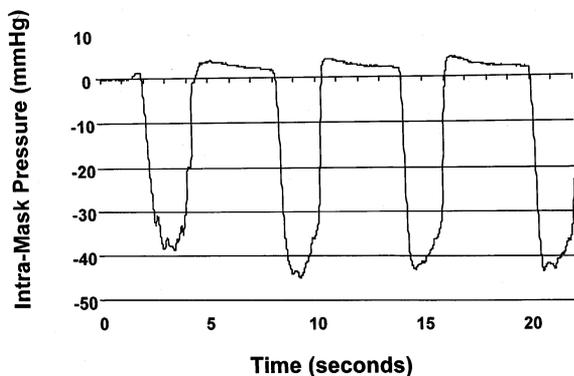


Fig. 6. Representative pressure waveforms obtained from a micromanometer-tipped catheter inside of a face mask during spontaneous ventilation in a normal volunteer. When the impedance valve is attached to a facemask covering the nose and mouth, inspiration reduced the pressure in the airway until a cracking pressure was achieved, allowing for inspiratory gas exchange. In this case the valve cracking pressure was set at  $-22$  cm  $H_2O$ .

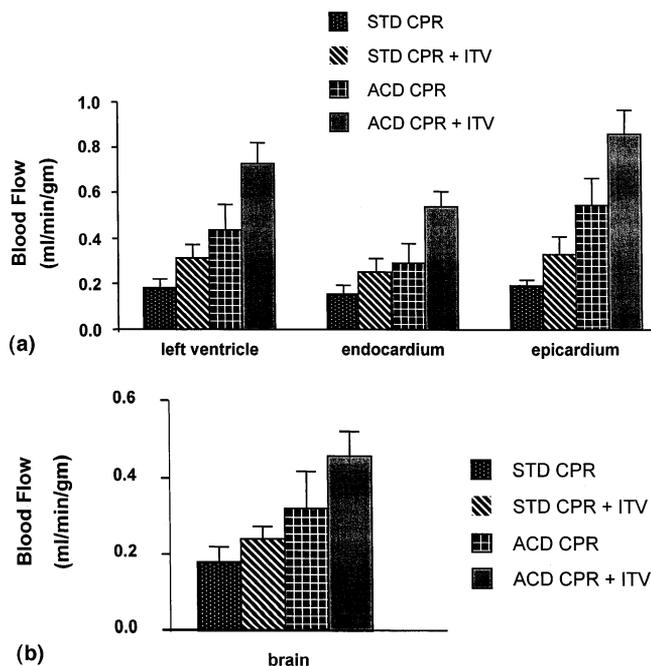


Fig. 7. This graph illustrates blood flow during CPR in a porcine model of ventricular fibrillation. Both left ventricular, epicardial, endocardial blood flow (a) and brain blood flow (b), measured using radiolabeled microspheres, increased with the addition of the impedance valve (ITV) with standard (STD) CPR and when the ITV was used during active compression-decompression (ACD) CPR [2,7,12].

wards higher initial values in the group that received standard CPR with the valve, removal of the valve resulted in a reduction in perfusion pressures and vital organ perfusion by nearly 50%.

In contrast, in those animals that received standard CPR initially, vital organ perfusion and coronary perfusion pressures stayed the same or increased with the serial addition of the impedance valve. Use of the impedance valve during standard CPR augmented left ventricular blood flow by more than 75% and cerebral perfusion by more than 35% when compared with standard CPR alone [9] (Fig. 7). When results of these studies are combined, there appears to be a linear relationship between the degree of left ventricular blood flow and the reduction negativity of intrathoracic pressure during the decompression phase [1,9] (Fig. 8).

## 5. Clinical studies

The importance of inspiratory impedance during ACD CPR was discovered when measuring intrathoracic pressures during ACD CPR in patients [11]. By occluding the tracheal tube with a thumb during the performance of intraoperative ACD CPR, we observed that intrathoracic pressures were markedly more negative when the airway was occluded. This led to the development of the concept of impedance threshold valve [4].

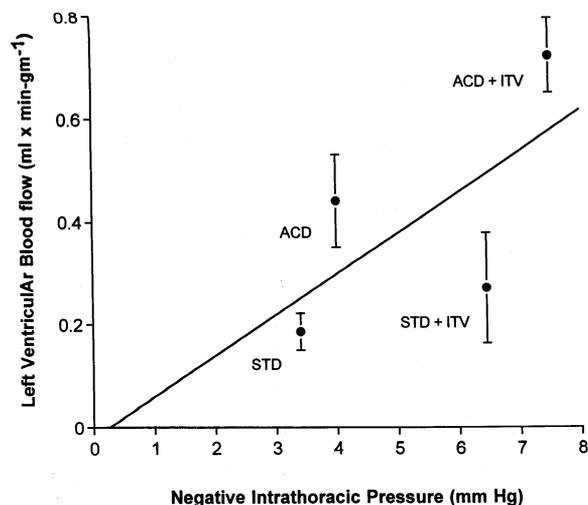


Fig. 8. The relationship between negative intrathoracic pressure measurements with left ventricular blood flow was derived from measurements during several different studies [2,7,12]. There appears to be a linear relationship between intrathoracic pressure during decompression phase and left ventricular blood flow.

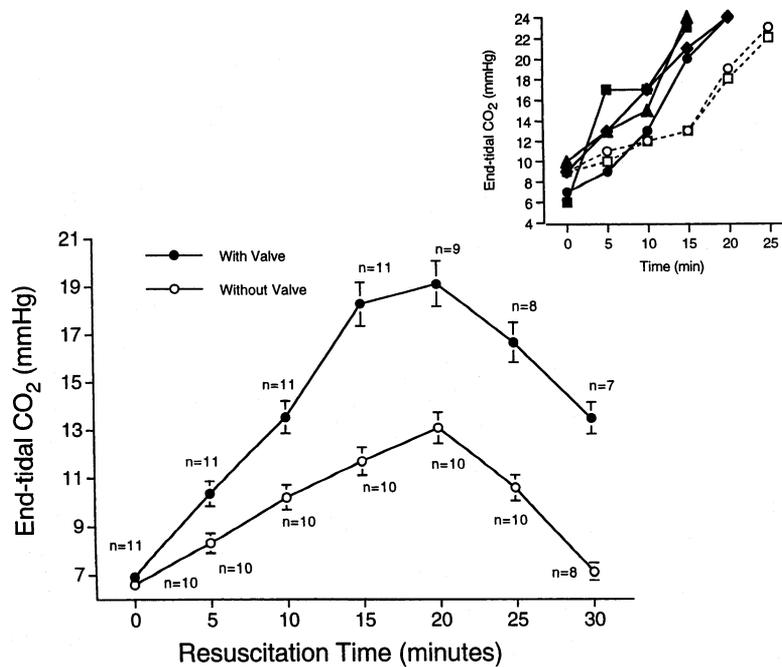


Fig. 9. End tidal CO<sub>2</sub> (ETCO<sub>2</sub>) levels in patients receiving active compression-decompression (ACD) CPR plus the impedance valve (ITV™) versus ACD CPR alone (open circles) are compared. Within minutes following intubation ( $T=0$ ) there was a significantly greater increase and more rapid rise in peak ETCO<sub>2</sub> levels in the ACD CPR plus valve group when compared to ACD CPR alone ( $P < 0.001$  for peak values). Numbers in parentheses reflect the number of patients still in cardiac arrest at each time point. Insert graph shows data from each individual patient who survived in each group (solid symbols represent ACD + valve; open symbols represent ACD alone) [8].

The first clinical evaluation of the impedance valve was performed in Paris, France [10]. A total of 21 patients in out-of-hospital cardiac arrest were studied. Of these patients, ten received ACD CPR during advanced life support while 11 patients received ACD CPR plus the valve. All patients had asystole when the mobile intensive care unit arrived at the scene. Investigators were blind to the technique used and a randomization schedule was used to determine which patients received ACD CPR with a functional impedance valve versus ACD CPR with a disabled valve. In the disabled valves the occluding diaphragm was removed but this could not be easily observed visually. The time between the call for help and the arrival of the basic life support (BLS) team was  $\sim 8$  min. These patients received BLS for  $\sim 8$  min prior to the arrival of the mobile intensive care unit. Patients were intubated by the medical intensive care unit staff and then randomized to receive ACD CPR with or without a functional valve. Patients received 1 mg of epinephrine every 5 min until the return of spontaneous ventilation. In the protocol resuscitation efforts were maintained for

a minimum of 30 min prior to discontinuation of CPR. As shown in Fig. 9, the end tidal CO<sub>2</sub> values were similar at the time of intubation ( $T=0$ ). In the group that received ACD CPR plus the valve, end tidal CO<sub>2</sub> rose more rapidly and to a higher value than in those patients who received ACD CPR alone. Moreover, four of the 11 patients in the valve group had a spontaneous return of circulation after a mean time of  $19.8 \pm 2.8$  min. In comparison, two of ten patients in the ACD group alone had a return of spontaneous circulation after a mean time of  $26.5 \pm 0.7$  min of CPR ( $P < 0.05$  for time from initiation of advanced life support CPR to return of spontaneous circulation). Thus, it appears as though the combination of ACD CPR plus the valve led to a more rapid 'priming of the pump' resulting in a more rapid return of spontaneous circulation. It is important to note that the vast majority of patients in Paris are found to be in asystole at the time that the first rhythm is recorded when the mobile intensive care unit arrives. A total of six out of 21 patients had a spontaneous conversion from asystole to a blood perfusing rhythm. It is also important to empha-

size how long it took to ‘prime the pump’ in those patients who received ACD CPR with a disabled valve. If ACD CPR had been discontinued in these patients with asystole after only 15 min, as is often the case in many cities, then no patients would have been resuscitated in the ACD CPR alone group.

In addition to end tidal  $\text{CO}_2$ , arterial and transvenous pressures were also measured (Figs. 10 and 11). Central lines were placed within the first 10 min of the cardiac arrest. Invasive monitoring was performed from minute 10 to minute

30 during CPR. The mean peak systolic blood pressure in patients who received ACD CPR plus an active valve was  $108 \pm 3.1$  versus  $90 \pm 6.4$  mmHg in the ACD CPR alone group ( $P < 0.05$ ). More striking, however, were the differences in diastolic blood pressure, mean arterial pressure and coronary perfusion pressure. As shown in Fig. 10, diastolic blood pressures in patients who received ACD CPR plus the ITV™ were greater than 55 mmHg, and coronary perfusion pressures in that same group were greater than 35 mmHg

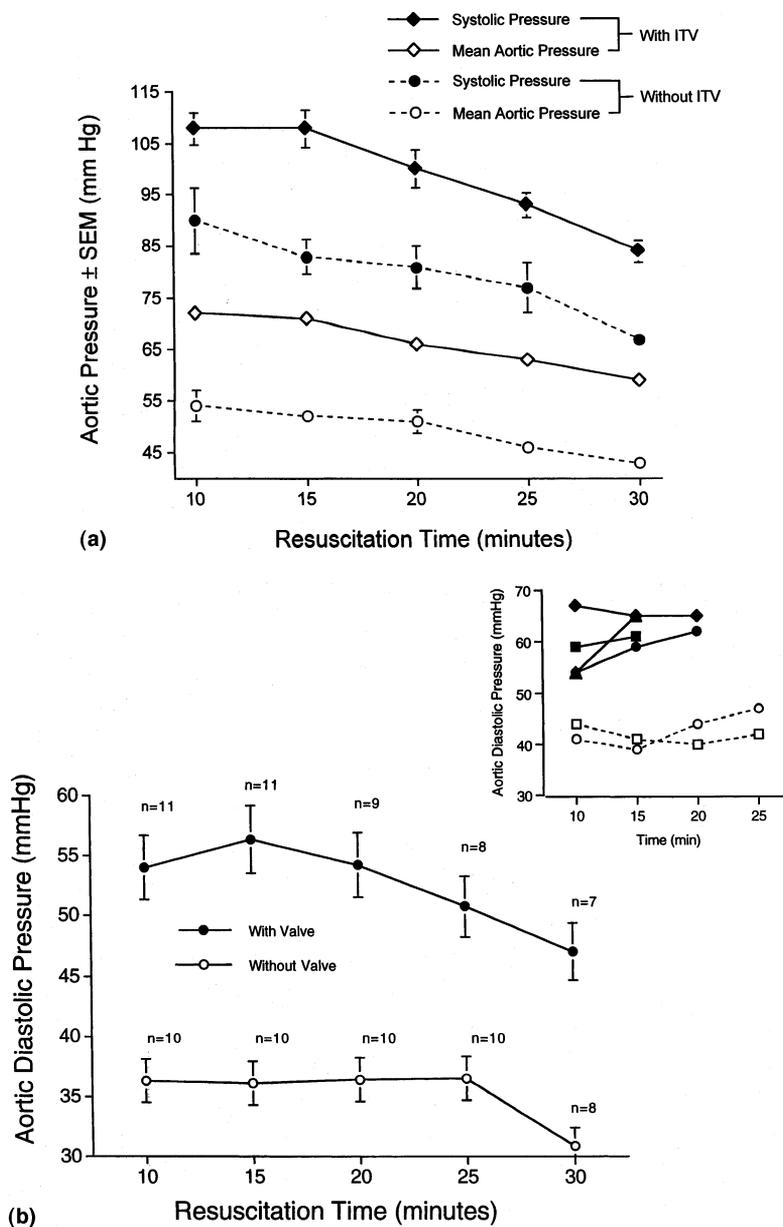


Fig. 10. Systolic, mean arterial and diastolic blood pressures in patients with out-of-hospital cardiac arrest receiving active compression-decompression (ACD) CPR alone versus ACD CPR plus the valve (ITV™) are illustrated. Patients underwent tracheal intubation at  $T=0$  min. The differences between systolic (a), mean arterial (a) and diastolic (b) pressures were highly statistically significant ( $P < 0.01$ ) between groups [8].

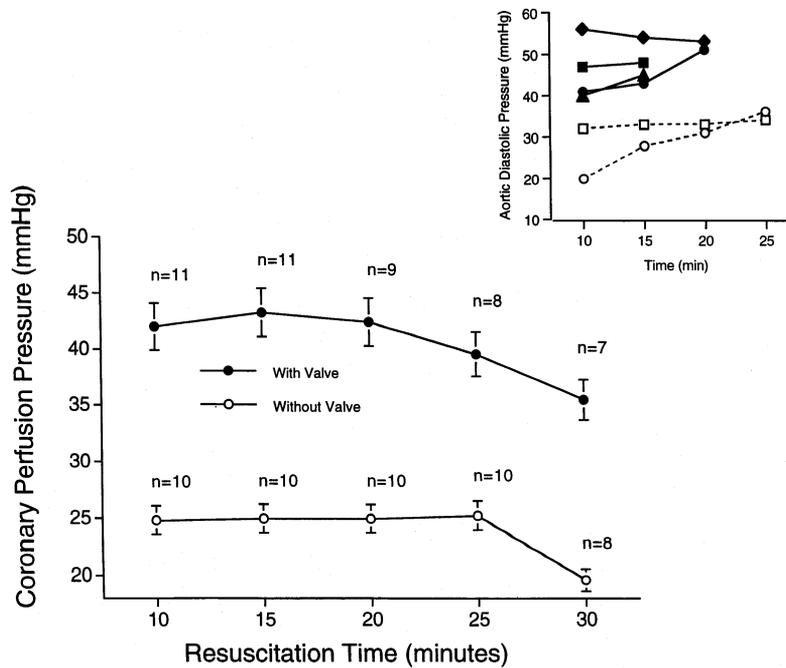


Fig. 11. Coronary perfusion pressures (CPP) from patients with out-of-hospital cardiac arrest during ACD CPR alone or during ACD CPR with an inspiratory threshold valve are shown. Numbers in parentheses reflect the number of patients still in cardiac arrest at each time point. Patients underwent tracheal intubation at  $T=0$  min. Coronary perfusion pressures were significantly higher in the ACD plus valve group versus those who received ACD CPR alone ( $P < 0.001$  for peak values). Insert graph shows data from each patient who survived in each group (solid symbols represent ACD + valve; open symbols represent ACD alone) [8].

( $P < 0.05$ ). Diastolic blood pressures and coronary perfusion pressures of this magnitude have not previously been reported in patients in cardiac arrest.

In standard CPR, we know from previous studies that end tidal  $\text{CO}_2$  levels in similar patients rise to  $\sim 15$  mmHg and maximal diastolic blood pressures are less than 20 mmHg [3]. These new data support the hypothesis that the combination of ACD CPR plus the ITV<sup>TM</sup> optimizes the bellows-like action of the chest by enhancing venous return to the chest with each decompression cycle. Moreover, the data are strikingly similar in those observed in the animal model of CPR [8]. They suggest that the combination of ACD CPR plus the impedance valve may provide an important alternative to standard CPR.

While these results are encouraging, at the present time we still lack data in patients regarding the potential benefits of the ITV<sup>TM</sup> with standard CPR alone. In addition, long-term data related to the potential of the ITV<sup>TM</sup> with any method of CPR in patients is not available. Such studies are on-going and we hope to be able to report those results at a future time.

## 6. Conclusions

Building upon studies with ACD CPR which demonstrate a potential benefit of reducing intrathoracic pressure during the decompression phase of CPR, the use of a one-way valve to occlude the airway during the decompression phase has led to a marked increase in overall CPR efficiency. While we await more data on the potential long-term benefits of the inspiratory impedance valve, it is clear that the use of a one-way valve to prevent inspiratory gas exchange and to enhance venous return during CPR can result in a significant increase in vital organ perfusion in both animal models and patients in cardiac arrest. Although the inspiratory threshold valve is relatively simple to use, the mechanisms underlying vital organ perfusion during CPR are complex. It seems clear, from both animal and human data, that enhancement of pre-load by a decrease the intrathoracic pressures in the decompression phase, improves overall CPR efficacy. Future studies are needed to define further the potential of low levels of positive expiratory pressure during the compression phase when the new valve is used.

Taken together, these data suggest that it may be possible in the not-too-distant future to enhance the overall efficacy of CPR significantly by relatively simple mechanical devices, thereby offering hope for those patients who suffer a cardiac arrest.

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