Improving Standard Cardiopulmonary Resuscitation with an Inspiratory Impedance Threshold Valve in a Porcine Model of Cardiac Arrest

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To improve the efficiency of standard cardiopulmonary resuscitation (CPR), we evaluated the potential value of impeding respiratory gas exchange selectively during the decompression phase of standard CPR in a porcine model of ventricular fibrillation. After 6 min of untreated cardiac arrest, anesthetized farm pigs weighing 30 kg were randomized to be treated with either standard CPR with a sham valve ($n = 11$) or standard CPR plus a functional inspiratory impedance threshold valve (ITV™) ($n = 11$). Coronary perfusion pressure (CPP) (diastolic aortic minus right atrial pressure) was the primary endpoint. Vital organ blood flow was assessed with radiolabeled microspheres after 6 min of CPR, and defibrillation was attempted 11 min after starting CPR. After 2 min of CPR, mean ± SEM CPP was 14 ± 2 mm Hg with the sham valve versus 20 ± 2 mm Hg in the ITV group ($P < 0.006$). Significantly higher CPPs were maintained throughout the study when the ITV was used. After 6 min of CPR, mean ± SEM left ventricular and global cerebral blood flows were 0.10 ± 0.03 and 0.19 ± 0.03 mL · min^{-1} · g^{-1} in the Control group versus 0.19 ± 0.03 and 0.26 ± 0.03 mL · min^{-1} · g^{-1} in the ITV group, respectively ($P < 0.05$). Fifteen minutes after successful defibrillation, 2 of 11 animals were alive in the Control group versus 6 of 11 in the ITV group (not significant). In conclusion, use of an inspiratory impedance valve during standard CPR resulted in a marked increase in CPP and vital organ blood flow after 6 min of cardiac arrest.

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Standard closed chest manual cardiopulmonary resuscitation (CPR) provides suboptimal vital organ blood flow secondary to the inherent inefficiencies of this technique. Despite being the standard of care for >40 years, survival after cardiac arrest and standard CPR remains grim: on average <5% of patients survive to hospital discharge despite CPR. Because forward blood flow from the heart to the periphery during CPR is determined by adequate venous return to the chest after each compression cycle, new mechanical devices and improved pharmacologic therapies have been developed to enhance CPR efficiency (1). One new approach, active compression-decompression (ACD) CPR, is a technique that transforms the thorax into a more efficient bellows. During the decompression phase, the chest is actively pulled upward by using a handheld suction device. This creates greater negative pressure within the thorax, resulting in enhanced blood return to the heart during the decompression phase (2,3). In comparison, venous return to the heart during standard CPR is highly dependent on the degree of natural chest wall recoil.

Investigations of the mechanism of ACD CPR led to the realization that complete airway occlusion during the decompression phase of ACD CPR resulted in greater negative intrathoracic pressure, enhanced venous refill of the heart, and therefore, a significant increase in CPR efficiency (4). Consequently, a new inspiratory impedance valve was developed to prevent inspiratory gas exchange only during the decompression phase of CPR, when intrathoracic pressure is less than atmospheric pressure (4,5). Most recently,

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the new valve has been evaluated in laboratory (4,5) and clinical studies (6). When the impedance valve was used during ACD CPR, coronary perfusion pressure (CPP) and vital organ blood flow were significantly increased compared with ACD CPR alone (4,6).

Because the impedance valve can be easily inserted in the respiratory circuit and does not require specific training when used during standard CPR, this device may also enhance standard CPR efficacy (7). Furthermore, if the new valve improves CPP above a threshold that usually correlates with return of spontaneous circulation, the administration of vasopressor drugs during CPR, such as epinephrine, may be spared (8). Accordingly, the primary goal of the current investigation was to assess the effects of the new valve during standard CPR on CPP. Other endpoints, including vital organ blood flow and return of spontaneous circulation were also evaluated in an animal model of cardiac arrest.

Materials and Methods

The Committee on Animal Experimentation approved this project at the University of Minnesota. All animals were managed in accordance with the guidelines of the American Physiological Society, the University of Minnesota, and the position of the American Heart Association on Research Animal Use. Animal care and use were performed by qualified individuals, supervised by veterinarians, and all facilities and transportation complied with current legal requirements and guidelines. Anesthesia was used in all surgical interventions. All unnecessary suffering was avoided, and research was terminated if unnecessary pain or fear resulted. Our animal facilities meet the standards of the American Association for Accreditation of Laboratory Animal Care.

The study was performed according to Utstein-style guidelines (9) on 22 healthy, 12–16-wk-old female domestic farm pigs weighing 28–34 kg. The pigs were anesthetized with a single bolus dose of ketamine (20 mg/kg IM), and pentobarbital (15 mg/kg IV bolus followed by 15 mg/kg per hour IV infusion) given via an ear vein. The pigs were intubated during spontaneous respiration with a 7.5-mm cuffed endotracheal tube (Mallinckrodt Critical Care, Glens Falls, NY), and mechanically ventilated (model 607; Harvard Apparatus, Dover, MA) at a volume-controlled setting of 20 mL/kg. During the preparation time, respiratory frequency was adjusted at 10–12 breaths/min according to end-tidal and arterial carbon dioxide partial pressure values to maintain the mean arterial carbon dioxide partial pressure at 35 torr; inspiratory oxygen concentration was 21%. Total fluid management consisted of 500–1000 mL of normal saline solution administered IV (Flo-Gard 6201; Baxter Healthcare, Hooksett, NH) throughout the 3-h preparation period.

Left ventricular and ascending aortic arch blood pressures were monitored by using a single high-fidelity micromanometer-tipped catheter (Millar Instruments, Houston, TX). This luminal aorto-left-ventricular micromanometer catheter was positioned under fluoroscopic guidance by femoral cutdown, and used for injection of radiolabeled microspheres. To monitor right atrial pressures, another micromanometer-tipped catheter was inserted through a right jugular vein sheath. Reference blood samples for measurement of organ blood flow were withdrawn from a 5F catheter placed in the descending aorta by femoral cutdown. Intrathoracic pressures were measured by using another micromanometer-tipped catheter positioned 2 cm below the tip of the endotracheal tube. It was inserted into the respiratory circuit via a sealed Y-connector, which was positioned between the endotracheal tube and the test valves, as described previously (4,5). Pressures were recorded continuously and the maximal negative intrathoracic pressure during each compression-decompression cycle was measured. For the measurement of body temperature, a thermometer was placed in the rectum. Body temperature was maintained with a heating blanket (Bair Hugger; Augustine Medical, Eden Prairie, MN) between 38.0° and 39.0°C.

Measurements

Pressure tracings obtained from the high-fidelity micromanometer catheters were continuously monitored with a data acquisition and recording system (Superscope II v1.295, GW Instruments, Somerville, MA). Digitized data were analyzed electronically to provide hemodynamic measurements. Heart rate was determined from a simultaneously recorded electrocardiogram signal. CPP calculated during diastole (relaxation) was defined as the arterio-venous pressure difference (time-coincident difference between aortic and right atrial pressure). Arterial blood gases were analyzed (IL 1301; Instrumentation Laboratory, Lexington, MA) every 30 min to ensure adequate acid base status and oxygenation. Organ blood flow was assessed with radiolabeled microspheres according to the method described by Heymann et al. (10).

Microspheres used in this study were radiolabeled with ^{141}Ce, ^{51}Cr, and ^{113}Sr (DuPont-New England Nuclear, Boston, MA). Each microsphere vial was placed in a water bath, and subjected to ultrasonic vibration for 1 min before injection. Approximately 5×10^6 microspheres were then immediately injected into the left ventricle through the lumen of the Millar catheter. The syringe and catheter were flushed with 10 mL of heparinized saline. With an automatic pump (Masterflex; Cole-Parmer, Vernon Hills, IL), arterial blood was continuously withdrawn from the descending aorta at

a rate of 6 mL/min just before the microspheres injection to 4 min thereafter. At the end of the experiment, the entire heart, cerebrum, and kidneys were removed. The left ventricular free wall was sectioned into three layers. Aliquots of each tissue were weighed, and placed into vials. Radioactivities from tissues and blood were measured with a γ scintillation spectrometer (COBRA II, Auto-Gamma; Packard Instrument Co., Downers Grove, IL), and blood flow values were subsequently calculated.

Experimental Protocol

This prospective randomized blinded evaluation was performed by using two different types of impedance valves (Figs. 1, 2). The functional impedance threshold valve (ITV™) (CPRx LLC, Minneapolis, MN) included the silicone diaphragm, which occludes the airway within the valve during the decompression phase. In the sham valve, the diaphragm was intentionally omitted during the manufacturing process. Both valves were pigmented so that it was not possible for the research team to know whether or not a functional or sham valve was used during the study. A computer-generated list was used to randomize the valves during the study.

The experimental protocol is shown in the timeline of Figure 3.

Fifteen minutes before cardiac arrest, 5000 U IV heparin was administered and prearrest hemodynamic variables as well as blood gases were measured. Immediately before induction of ventricular fibrillation, the anesthetic was discontinued. Ventricular fibrillation was induced by applying a 50 Hz, 7.5 V AC electrical current through a right ventricular endocardial electrode. Cardiac arrest was defined as the point at which the aortic pulse pressure decreased to zero, and the electrocardiogram showed ventricular fibrillation; ventilation was stopped at that point. After 6 min of untreated cardiac arrest, pigs were randomly assigned to receive either the functional or sham valve inserted between endotracheal tube and the bag valve. The functional or sham valve was placed in the respiratory circuit at that time. Closed-chest standard CPR was performed at 80 bpm with a pneumatically driven automatic piston device for CPR (ACD Controller; AMBU, Glostrup, Denmark) for 11 min. With this device, chest compression was consistently performed at a depth of 25% of the anterior-posterior diameter of the chest. This was monitored continuously by using a displacement gauge as described previously (4,5). With this device, the compression piston is lifted upward so that the chest can return to its baseline position after each compression. The compression and decompression phase duty cycle was set to 50%. Ventilation was performed by manual bag valve ventilation with 10 L/min oxygen, an approximately 450-mL tidal volume, and a 5:1 compression/ventilation ratio. After 6 min of untreated ventricular fibrillation, and then 6 min of standard CPR with a functional or sham valve, radiolabeled microspheres were injected into the left ventricle to assess vital organ blood flow. Perfusion pressures were monitored throughout the entire study. After 17 min of cardiac arrest, including 11 min of CPR, up to 3 counter shocks (200 J) were administered with a defibrillator (Lifepak 7; Medtronic Physio-Control, Redmond, WA).

We defined return of spontaneous circulation as an unassisted pulse with a systolic arterial pressure of >80 mm Hg, and pulse pressure of >40 mm Hg, a slightly more stringent criteria than the >60 mm Hg arterial pressure as recommended by the Utstein guidelines in laboratory CPR research. In animals that were successfully resuscitated, an IV saline infusion (300 mL per hour) was started. No other therapy was delivered. Fifteen minutes after return of spontaneous circulation, a third radiolabeled microsphere was injected into the left ventricle to assess vital organ perfusion after return of spontaneous circulation. After finishing the experimental protocol, the animals were euthanized with an overdose of pentobarbital and potassium chloride. All pigs underwent necropsy to check correct positioning of the catheters, damage to the rib cage, and to harvest organs for blood flow measurement.

Values were expressed as mean ± SEM. The primary endpoint was CPP. The comparability of weight and baseline data was tested with the *t*-test for continuous variables. A two-factor analysis of variance with repeated measures on one factor was used to determine statistical significance between groups. Because blood flow data were unevenly distributed, the Mann-Whitney *U*-test (two-tailed) was applied to determine a significant difference between each group. Statistical significance was considered to be at $P < 0.05$ after Bonferroni correction for multiple comparisons.

Results

Before induction of cardiac arrest, there were no statistically significant differences in weight, temperature, hemodynamic variables, organ blood flow, and blood gases between groups (Tables 1 and 2).

The CPPs remained significantly higher during the entire experiment (Fig. 4). Mean ± SEM negative intrathoracic pressures were lower in the Functional Valve group (−5.2 ± 0.5 mm Hg) when compared with standard CPR alone (−1.1 ± 0.2 mm Hg) ($P <$ 0.0001 between groups). Mean ± SEM arterial carbon dioxide partial pressure after 10 and 16 min of CPR was significantly higher in the Functional Valve group. Arterial pH values were significantly lower when the functional valve was used (Table 1).

After 6 min of CPR, vital organ blood flow was significantly higher in the Functional Impedance

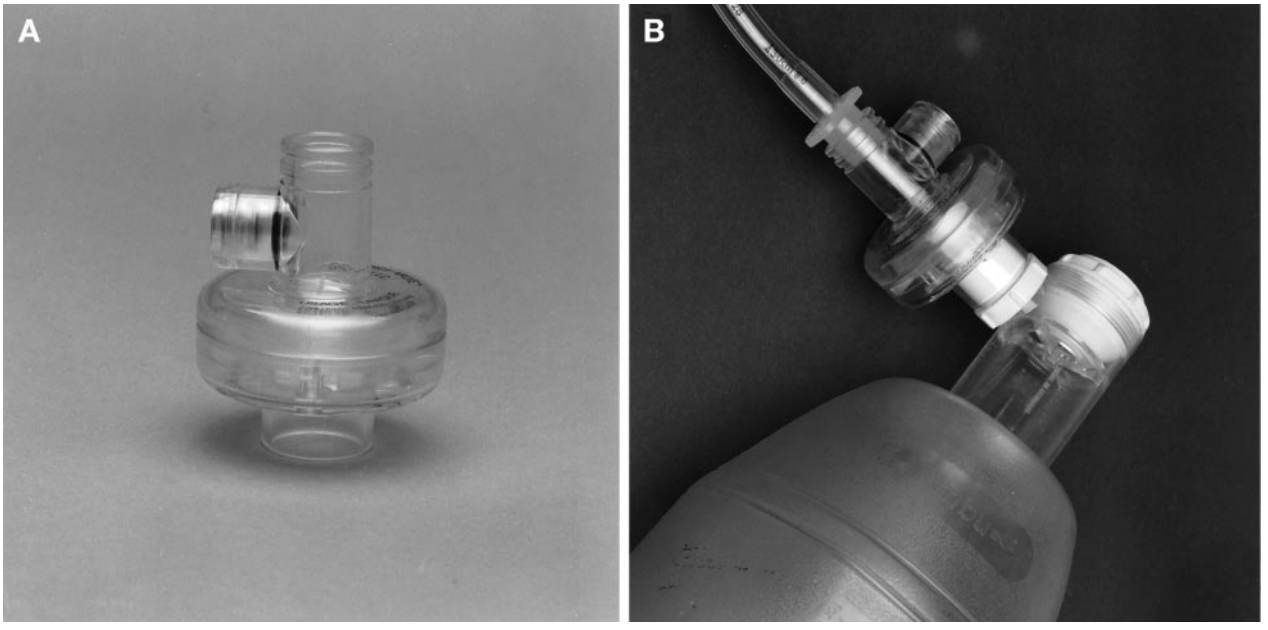

Figure 1. Photograph of the impedance valve alone (A) and attached to a resuscitation bag (B).

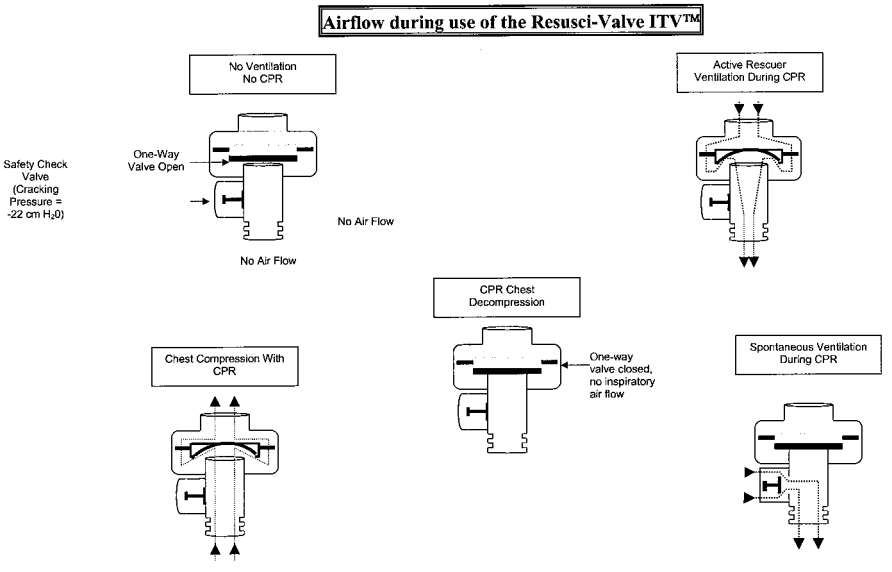

Figure 2. Schematic representation of respiratory gas flow through the impedance valve in the absence and presence of cardiopulmonary resuscitation (CPR). During a spontaneous breath, a safety check valve opens at a cracking pressure of −22 cm H_2O, enabling inspiration. ITV = impedance threshold valve.

Baseline	0	6	10	12	16	17	+5	+15
time (min)								
Inject Microsphere 1 ABG	VF	Start CPR ABG	ABG	Inject Micro. 2	ABG	Shock 3x200 J	ABG	Inject Micro. 3 ABG

Standard CPR ± Functional Impedance Valve

Figure 3. Experimental protocol. ABG = arterial blood gas; Micro. = microspheres; VF = ventricular fibrillation induced.

Valve group (Table 2). Left ventricular blood flow nearly doubled with the functional impedance valve but remained very low compared with prearrest values. In contrast, cerebral blood flow increased from 58% of baseline flow to 75% of baseline flow with the functional valve ($P < 0.05$).

Return of spontaneous circulation could be achieved in 6 of 11 animals in the ITV group versus 3 of 11 in the standard CPR group (not significant). No vasopressors were used to achieve successful resuscitation. In surviving animals, there were no statistically significant differences in hemodynamic variables, organ blood flow, and blood gases between groups during the postresuscitation phase. However, only 2 of 11 pigs in the Control group survived for 15 min, whereas 6 of 11 animals treated with the functional valve were still alive at this point (Tables 1 and 2). Necropsy confirmed appropriate catheter positions, and revealed no injuries of the rib cage or intrathoracic organs in any animals.

Table 1. Blood Gas Variables at Baseline Before Cardiac Arrest, During Cardiac Arrest, During Cardiopulmonary Resuscitation, and During the Postresuscitation Phase in Pigs

	Prearrest	Cardiac arrest	CPR (min)		Postresuscitation phase	
			10	16	5	15
Pao_2 torr						
STD CPR	95 ± 4	115 ± 35	344 ± 45	465 ± 55	105 ± 40	125
STD CPR + ITV	85 ± 3	130 ± 30	306 ± 45	320 ± 55	115 ± 10	130 ± 10
$Paco_2$ torr						
STD CPR	32 ± 1	26 ± 2	27 ± 3*	24 ± 2*	37 ± 4	44
STD CPR + ITV	33 ± 1	30 ± 3	37 ± 3	31 ± 2	40 ± 3	25 ± 3
pHa units						
STD CPR	7.46 ± 0.01	7.51 ± 0.02	7.44 ± 0.03*	7.42 ± 0.05*	7.29 ± 0.06	7.25
STD CPR + ITV	7.44 ± 0.01	7.45 ± 0.03	7.30 ± 0.02	7.29 ± 0.02	7.24 ± 0.04	7.30 ± 0.03

All variables are given as mean ± SEM.

Prearrest = measurements before induction of cardiac arrest, CPR = cardiopulmonary resuscitation, STD CPR = standard cardiopulmonary resuscitation with the sham valve, STD CPR + ITV = standard cardiopulmonary resuscitation with the functional valve, Po = oxygen partial pressure, $Paco_2$ = carbon dioxide partial pressure. The CPR values of 10 and 16 min refer to the period of time after induction of ventricular fibrillation. CPR was started after 6 min of untreated ventricular fibrillation. Postresuscitation times refer to the total amount of time after successful resuscitation. Eleven pigs/group were studied. In the postresuscitation phase, 6/11 animals in the STD CPR + ITV group survived for 15 min whereas 3/11 in the STD CPR group alone survived for >5 min and only 2/11 lived for 15 min.

* $P < 0.05$ STD CPR versus STD CPR + ITV.

Table 2. Hemodynamic and Blood Flow Variables at Baseline Before Cardiac Arrest, During Cardiopulmonary Resuscitation, and During the Postresuscitation Phase in Pigs

	Baseline	12 min CPR	5 min ROSC	15 min ROSC
HR (bpm)				
STD CPR	155 ± 10	NA	160 ± 30 (n= 3)	183 (n = 2)
STD CPR + ITV	155 ± 8	NA	170 ± 10 (n= 6)	176 ± 21 (n = 6)
AO compression pressure (mm Hg)				
STD CPR	92 ± 7	50 ± 3	77 ± 11 (n= 3)	74 (n = 2)
STD CPR + ITV	100 ± 3	57 ± 4	87 ± 10 (n= 6)	86 ± 1 (n = 6)
AO decompression pressure (mm Hg)				
STD CPR	77 ± 7	15 ± 4	44 ± 13 (n= 3)	58 (n = 2)
STD CPR + ITV	87 ± 3	20 ± 1	60 ± 8 (n =6)	62 ± 9 (n = 6)
Mean right atrial pressure (mm Hg)				
STD CPR	3 ± 0	4 ± 1	6 ± 2 (n = 3)	3 (n = 2)
STD CPR + ITV	5 ± 0	4 ± 1	6 ± 2 (n = 6)	4 ± 1 (n = 6)
Left ventricular blood flow ($mL \cdot min^{-1} \cdot g^{-1}$)				
STD CPR	1.28 ± 0.11	0.11 ± 0.03*	NA	1.2 (n = 2)
STD CPR + ITV	1.69 ± 0.28	0.19 ± 0.03	NA	1.7 ± 0.40 (n = 6)
Global cerebral blood flow ($mL \cdot min^{-1} \cdot g^{-1}$)				
STD CPR	0.32 ± 0.04	0.19 ± 0.04*	NA	0.23 (n = 2)
STD CPR + ITV	0.33 ± 0.05	0.26 ± 0.03	NA	0.22 ± 0.03 (n = 6)
Kidney blood flow ($mL \cdot min^{-1} \cdot g^{-1}$)				
STD CPR	4.05 ± 0.27	0.20 ± 0.05	NA	2.96 (n = 2)
STD CPR + ITV	5.58 ± 0.73	0.37 ± 0.07	NA	2.77 ± 0.25 (n = 6)

All variables are given as mean ± SEM.

Baseline = measurements before induction of cardiac arrest, CPR = cardiopulmonary resuscitation, AO = aortic, HR = heart rate, STD CPR = standard cardiopulmonary resuscitation with the sham valve, STD CPR + ITV = standard cardiopulmonary resuscitation with the functional valve, NA = not applicable, ROSC = return of spontaneous circulation. The CPR values 12 min refers to the period of time after induction of ventricular fibrillation. CPR was started after 6 min of untreated ventricular fibrillation. Postresuscitation times refer to the total amount of time after successful resuscitation. Eleven pigs/group were studied. Only 6/11 in the STD CPR + ITV group survived for 5 min after ROSC versus 3/11 in the Control group.

* $P < 0.05$ STD CPR versus STD CPR + ITV.

Assessment of all valves after the experiment revealed proper function in all cases.

Discussion

Passive chest wall recoil and subsequent intrathoracic pressure changes determine the degree of blood return to the lungs and heart during standard CPR. Closed chest standard CPR alone, without vasopressors, often fails to maintain minimal levels of vital organ blood flow needed to sustain and restore life (12–15). The results from this study demonstrate that intermittent airway occlusion with the impedance valve during the decompression phase of standard CPR improved cardiopulmonary blood return, thereby significantly improving CPP and vital organ

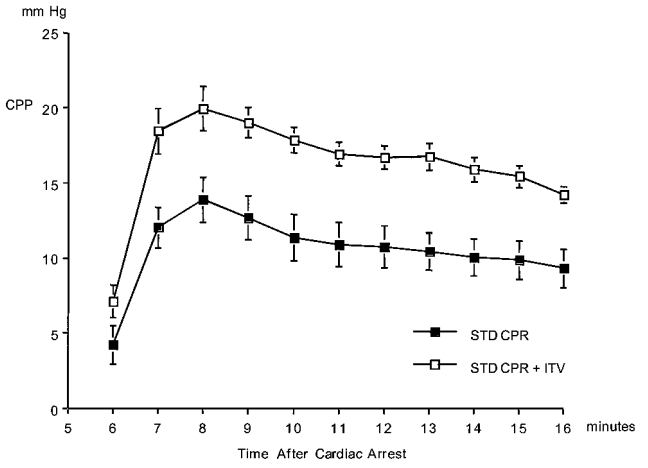

Figure 4. Mean ± SEM coronary perfusion pressure (CPP) during impedance threshold valve (ITV) cardiopulmonary resuscitation (CPR) (□), or standard CPR (■). STD CPR = standard CPR with a sham valve, STD CPR + ITV = standard CPR with a functional valve. *$P < 0.0001$ for comparisons between groups.

blood flow. The addition of the ITV improved the efficiency of standard CPR and resulted in a nearly 40% improvement in cerebral blood flow and a 50% increase in myocardial perfusion.

A distinctive difference between key hemodynamic variables indicates that use of the new valve is beneficial for "priming the pump" and increasing CPR efficiency. The results from these studies suggest that preventing inspiratory gas exchange during standard CPR, when not actively ventilating the pigs, caused an increased negative intrathoracic pressure during the decompression phase of CPR. The consequences, in the absence of powerful vasopressors typically used to increase systemic vascular resistance, were striking. Both hemodynamic data and blood flow measurements support the fundamental importance of enhancing blood return into the thorax during CPR. Accordingly, we found a significant improvement in myocardial and cerebral perfusion. Although increasing cardiac arrest times will increase the resistance to sufficient reflow in vital organ systems (11), brain blood flow was nearly 75% of normal values in the Functional Valve group. Myocardial perfusion was also increased by nearly twofold when compared with standard CPR with the addition of the valve. However, even with the addition of the valve, total myocardial perfusion fell far short of normal values. This is in contrast to our findings when we compared vital organ blood flow during ACD CPR with and without an impedance valve (4,6). With active decompression and the valve, left ventricular and brain blood flow were approximately 55% and 125% of baseline, respectively (4). In patients undergoing CPR, the mean blood pressure was 110/56 when ACD/CPR was performed with the valve versus 90/35 with ACD/CPR alone (6).

The hemodynamic effects of the impedance valve during standard CPR may be closely related to the arrest time, and other further factors such as fluid status, levels of endogenous hormones, diaphragmatic tone, the compliance of the ventricular wall, and duration of CPR. Both chest wall and respiratory system compliance during CPR decrease significantly over time (12–16). The effectiveness of the valve depends on many of these factors during standard CPR. This may explain why we observed a greater effect of the valve when the cardiac arrest time, in the absence of CPR, was increased from four to six minutes. In an earlier study, with a shorter arrest time, the effect of the impedance valve was evident but not as striking until the arrest time was lengthened (5).

As reported previously, insertion of the impedance valve in the respiratory circuit decreased minute ventilation by preventing airflow during chest compressions (4). This resulted in higher-than-normal arterial carbon dioxide values in the Functional Valve group (4,5). Moreover, progressive arterial acidosis occurred to a greater extent when the valve was used. The latter finding is likely to reflect the beneficial effects of airway occlusion on systemic circulation, whereas normal arterial pH values during low flow states such as CPR may not reflect the degree of cardio-circulatory failure (16). Because we did not measure mixed venous blood samples, it is not possible to state definitively whether the lower pH in the Active Valve group is secondary to increased metabolic acidosis or worsening respiratory acidosis. Although the decrease in gas exchange in the Functional Valve group was obvious, all variables remained within adequate levels. Furthermore, the trend of 6 of 11 ITV versus 3 of 11 standard pigs with return of spontaneous circulation suggests that decreased minute ventilation in the Functional Valve group had no negative impact on survival rates. Fifteen minutes after return of spontaneous circulation, 6 of 11 pigs treated with the functional valve were alive versus 2 of 11 in controls.

Some limitations of the present study should be noted. We did not assess mixed venous blood gases; accordingly, we are unable to interpret gradients between mixed venous and arterial blood gas values. Ventilation was synchronized with chest compressions and delivered primarily during the decompression phase. Therefore, we were unable to report on effects of the valve when chest compressions and ventilation were not synchronized. In addition, we determined the sample size based on CPP as the primary endpoint. Consequently, a potential survival benefit from the valve will require another study (continuing) with a larger number of animals.

Additional limitations include that we did not measure chest wall compliance in this study, which varies

in pigs and humans from one chest to the next. Interindividual differences in chest wall may strongly influence the effect of intermittent airway occlusion on hemodynamics during standard CPR. As such, it is important to reemphasize that, when performing standard CPR on a pig or on a patient, the chest must be allowed to recoil to its natural resting position to provide the chest recoil needed to create a slight vacuum within the thorax relative to the rest of the body and atmospheric pressure. We also observed some differences in the baseline CPPs between the groups. Although these were not statistically significant, they could have altered the overall results. Finally, it is important to note that we observed no significant adverse effects of the impedance valve in this model.

In conclusion, the use of an impedance valve during standard CPR resulted in a marked increase in CPP and vital organ blood flow when compared with standard CPR alone. On the basis of these favorable results, examination of the potential benefit of intermittent impedance of inspiratory gas exchange during standard CPR in patients seems warranted.

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