Combination of active compression decompression cardiopulmonary resuscitation and the inspiratory impedance threshold device: state of the art

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Purpose of review

Over the past decade, the combination of active compression decompression (ACD) cardiopulmonary resuscitation (CPR) and an impedance threshold device (ITD) has been shown to significantly increase vital organ perfusion pressures and survival rates in animals and humans. The purpose of this review article is to summarize the recent advances with this new technology.

Recent findings

Building upon animal studies that demonstrated the benefit of the ITD used with either ACD CPR or standard CPR (S-CPR), four prospective, randomized clinical trials with ACD/ITD CPR have been recently completed. One blinded, out-of-hospital cardiac arrest trial (n = 21 patients) demonstrated that systemic blood pressures and coronary perfusion pressures were markedly higher when ACD/ITD CPR was used when compared directly with ACD CPR alone. The second blinded trial demonstrated that the combination of ACD/ITD CPR was effective with both a facemask and an endotracheal tube (n = 15 patients). A third randomized clinical trial (n = 210 patients) demonstrated that 24-hour survival rates for out-of-hospital cardiac arrest were more than 65% higher with ACD/ITD CPR than with S-CPR ($P < 0.01$). Neurologic function after cardiac arrest trended higher in patients with witnessed arrest who received ACD/ITD CPR than in those who received S-CPR ($P < 0.07$). In addition, when ACD/ITD CPR was applied later in the course of treatment, short-term survival rates were threefold higher in patients receiving ACD/ITD CPR (44%) than in those receiving S-CPR (14%)($P < 0.05$). In that study, patients with the greatest chance for survival–those with witnessed cardiac arrest and an initial rhythm of ventricular fibrillation–had a 23% 24-hour survival rate with S-CPR *versus* a 58% 24-hour survival rate with ACD/ITD CPR ($P < 0.01$). It should be noted that this trial was performed in a city where an earlier study found no difference in outcomes between ACD CPR alone and S-CPR. The fourth clinical trial was a randomized, double-blinded study of 400 patients with out-of-hospital cardiac arrest treated by advanced life support personnel. All patients received ACD CPR: half were treated with a sham ITD and the other half were treated with an active ITD. Twenty-four hour survival, the primary endpoint, was 32% in the active ITD group *versus* 22% in the sham group ($P < 0.05$).

Summary

On the basis of the cumulative findings of these studies, it is concluded that ACD/ITD CPR provides superior vital organ blood flow and results in significantly higher short-term survival rates than do ACD CPR alone or S-CPR. Use of the ACD/ITD CPR technology optimizes perfusion of the heart and brain during cardiac arrest and results in the highest reported survival rates of any CPR device technology. Use of this technology should be encouraged while additional studies are under way to examine the potential long-term impact of this new technology.

Keywords

active compression decompression, cardiopulmonary resuscitation, inspiratory impedance threshold device

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Abbreviations

ACD	active compression decompression
CPR	cardiopulmonary resuscitation
ITD	impedance threshold device
S-CPR	standard cardiopulmonary resuscitation

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Introduction

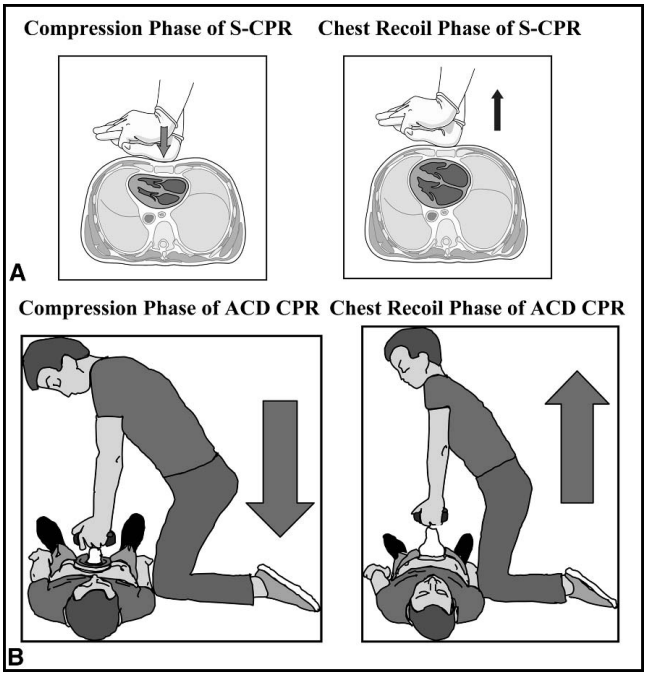
The concept of active compression decompression (ACD) cardiopulmonary resuscitation (CPR) was inspired by a patient who was successfully resuscitated on several occasions with the use of a common household plunger to facilitate CPR [1]. This led several investigators to study the potential benefit of transforming the chest into an active bellows during CPR [2–6]. To understand the significance of the chest acting as a bellows, one capable of forcing blood out of the chest during the chest compression phase and drawing blood back into

the chest during the chest recoil or decompression phase, it is first important to understand the mechanisms and shortcomings of manual closed chest cardiac massage, often referred to as standard CPR (S-CPR).

The purpose of S-CPR is to pump blood from the chest to vital organs during the compression phase and to enhance the return of blood back into the chest during the chest wall recoil or decompression phase. Compression and decompression associated with S-CPR are illustrated in Figure 1A.

With S-CPR, chest compression results in an elevation of intrathoracic pressure and in direct compression of the heart. Both mechanisms result in forward blood flow out of the chest to perfuse the brain and other vital organs. However, the effectiveness of S-CPR is largely determined by the amount of blood returned to the heart. Blood flow back to the heart is highly dependent on the degree of chest wall recoil [3]. During the decompression (or passive relaxation) phase, the chest recoils, causing a small decrease in intrathoracic pressure (relative to atmospheric pressure) [5,6]. This small but important vacuum results in venous blood return to the right side of the heart [3,5]. S-CPR by itself is inherently inefficient, in large part because of the lack of adequate blood return to the thorax during the chest wall recoil phase. Blood flow to the vital organs is severely reduced during S-CPR, even under the best circumstances [2,3,4].

Figure 1. Phases of cardiopulmonary resuscitation (CPR)



(A) Standard CPR (S-CPR). Chest compression results in an elevation of intrathoracic pressure and in direct compression of the heart. Both these mechanisms result in forward blood flow out of the chest. During the decompression phase, the chest recoils, causing a small decrease in intrathoracic pressure to return venous blood to the right side of the heart. Incomplete recoil reduces the vacuum created during chest wall decompression. **(B)** Active compression decompression (ACD) CPR uses the same compression concept as S-CPR but increases the naturally occurring negative intrathoracic pressure by physically lifting the chest wall and helping it return to its resting position.

Additionally, in the vast majority of cases, the coronary perfusion pressures during S-CPR are only marginally adequate because the pressure gradient between the aorta, the right atrium, and left ventricle is far from optimal [3,7,8]. Myocardial perfusion predominantly occurs during the decompression phase. The difference between the diastolic aortic and the right atrial pressures (coronary perfusion pressure) is thought to be the critical determinant of CPR efficacy.

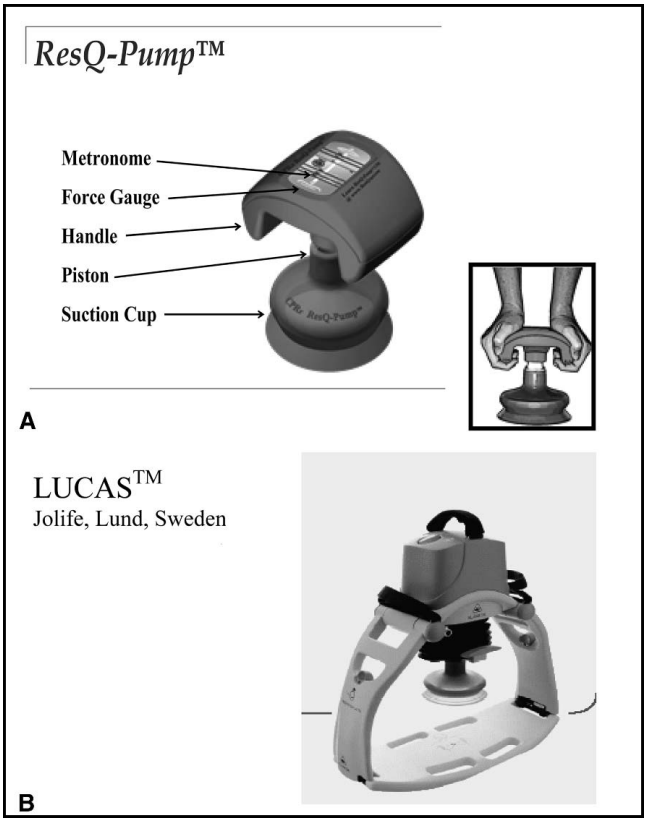
The American Heart Association recognized the inefficiencies of S-CPR in 2000 when they issued an updated guideline on the performance of CPR [9]. This guideline reinforces the importance of the chest decompression phase and emphasizes teaching correct decompression while performing CPR:

> Release the pressure on the chest to allow blood to flow into the chest and heart. You must release the pressure completely and allow the chest to return to its normal position after each compression [9].

Despite these recommendations, recent studies by Aufderheide *et al.* on patients in Milwaukee, Wisconsin, have established that even when CPR is performed by professional rescuers, there is often incomplete chest wall recoil [10]. Despite training, it is difficult to perform S-CPR correctly and allow for the chest to recoil fully after each compression. Incomplete chest wall recoil is a common error, especially for the overzealous rescuer. This results in significantly less blood flow back to the heart and reemphasizes that a device is needed to correct this widespread problem. Follow-up animal studies have revealed that the incomplete chest wall recoil observed in humans results in a decrease in coronary perfusion pressure, systemic blood pressure, and cerebral perfusion pressure [11]. One of the benefits of ACD CPR, as we shall describe, is that proper use of the ACD CPR device promotes full chest wall recoil and provides guidance to the user in the performance of CPR.

Active compression decompression CPR increases the naturally occurring negative intrathoracic pressure by physically expanding the chest wall and helping it return to its resting position (Fig. 1B). This can be accomplished by a hand-held device (Fig. 2A) or an automated device (Fig. 2B). During S-CPR, the chest wall's natural elasticity will partially recoil from compression. Many factors can contribute to less than optimal recoil. These included age, brittle or broken ribs, separated or broken sternum, barrel-shaped chest, chest concavity, and in-

Figure 2. Active compression decompression cardiopulmonary resuscitation (CPR) device variations



(A) Manual active compression decompression CPR device showing the handle, compression/decompression force gauge, and metronome. The insert shows how to compress/decompress the chest with elbows locked and wrists rigid. The device is attached to the sternum. By rocking back and forth, the chest can be compressed and decompressed. **(B)** Automated compression decompression device. The device is pneumatically driven. It is rapidly placed around the patient and provides continuous compressions with active recoil of the chest to the full resting position after each compression [52].

complete release of pressure by the performer of CPR. ACD CPR actively re-expands the chest wall and generates the negative intrathoracic pressure needed to allow passive filling of the heart. By actively re-expanding the chest wall, ACD CPR resolves many of the problems encountered with S-CPR. There have been many clinical trials with the ACD CPR device used by itself, as well as several descriptions of how to use this device [12–26].

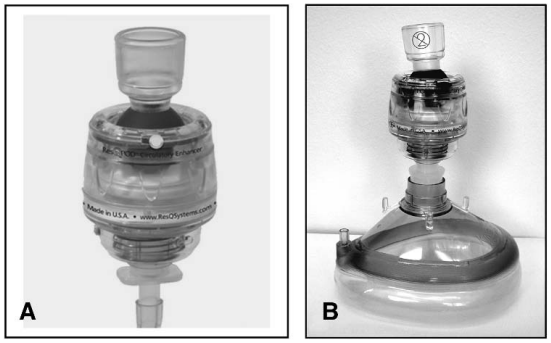
Over the past 13 years, this method has been extensively evaluated in animals and humans [12–26]. Although ACD CPR does increase vital organ perfusion and minute ventilation when compared with S-CPR, it has been difficult to show a consistent benefit from one city to the next in the evaluation of the clinical benefit of this technique in patients in cardiac arrest. In St. Paul, Minnesota, and Paris, France, the performance of ACD CPR significantly increased the chances for resuscitation [21,23]. In Paris, the use of ACD CPR more than doubled 1-year survival compared with S-CPR [21]. However, the results with ACD CPR in other cities were no different from those observed with S-CPR [24–26]. It has been speculated that these disparities may be the result of variability in training, a lack of on-scene advanced life support, and inexperience in the use of the device by the emergency medical system personnel.

By themselves, both S-CPR and ACD CPR create a vacuum in the thorax with each chest wall decompression. However, much of the potential hemodynamic benefit of this vacuum is lost by the influx of inspiratory gas. The inspiratory impedance threshold device (ITD) was developed to prevent the influx of respiratory gases during the decompression phase of CPR [3,5,13,27–30]. The ITD harnesses the kinetic energy of chest wall recoil, thereby augmenting the bellows-like action of the chest with each compression-decompression cycle.

When the ITD is used during the active decompression phase of CPR, intrathoracic pressures became markedly more negative with each decompression. This small, lightweight device containing pressure-sensitive valves is designed to selectively impede an influx of inspiratory gas during chest wall decompression, thereby augmenting the amplitude and duration of the vacuum within the thorax. When used with S-CPR or ACD CPR, the ITD prevents respiratory gases from entering the lungs during the decompression phase [3,5,13,27]. The increased intrathoracic vacuum generated from using the ITD draws more venous blood back into the heart, resulting in increased cardiac preload, followed by improved cardiac output and vital organ perfusion.

The ITD can be used with both a facemask (Fig. 3A) and an endotracheal tube (Fig. 3B) [13] The ITD is inserted into the respiratory circuit, between the endotracheal tube or facemask and the ventilation source (*eg* mouth-to-mouth, bag-valve resuscitator, transport ventilator). It is designed to prevent inspiratory gas exchange, except when the rescuer is actively ventilating or the patient begins to breathe spontaneously. The device allows the rescuer to ventilate the patient without any resistance and allows for unobstructed patient exhalation. Timing lights on the device prompt the rescuer when to ventilate. This visual aid works to avoid hyperventilation, given that excessive ventilation rates have been found to be harmful if not deadly [29]. When CPR is not being performed or if the patient has successfully been resuscitated, the ITD should be removed. Spontaneous inspiration through the ITD is possible but may be difficult for a recently resuscitated patient. The cracking pressure, the inspiratory pressure needed to allow for spontaneous inspiration within the device, can be varied at the time of manufacturing. Clinical trials to date have been performed with ITD cracking pressures between –15 cm and –24 cm H_2O. Over the past several years, with the measure of actual intrathoracic pressures in pa-

Figure 3. Impedance threshold device (ITD)



(A) ITD on endotracheal tube. **(B)** ITD on facemask. Lower intrathoracic pressure in the chest during the decompression phase of cardiopulmonary resuscitation enhances venous return to the thorax. Each time the chest wall recoils after a compression, the ITD transiently blocks oxygen from entering the lungs, creating a small vacuum in the chest, resulting in improved preload.

tients undergoing CPR, it has become clear that a cracking pressure of –7 cm H_2O is adequate for standard CPR and –15 cm H_2O is adequate for ACD CPR [31]. With lower cracking pressures, normal subjects can breathe through the ITD [32].

Preclinical studies

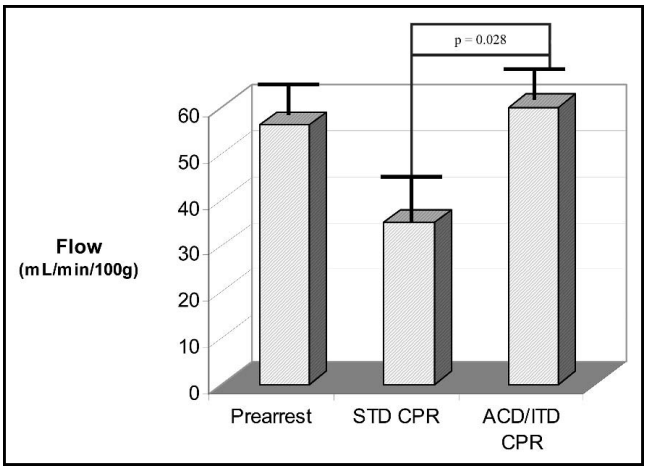
Since 1995, there have been multiple animal and clinical investigations related to ACD/ITD CPR [3,5,28,29,33–44]. The first study established the importance of the concept [27]. Addition of the ITD in that animal model demonstrated a doubling of vital organ blood flow compared with ACD CPR alone. The most remarkable finding from this study was the discovery that blood flow to the brain was greater in the arrested pig treated with ACD/ITD CPR than in the prearrested pig (Fig. 4). Compared with S-CPR alone, ACD/ITD CPR resulted in a 300% increase in blood flow to the brain and a 400% increase in blood flow to the heart [27]. With S-CPR, cerebral and cardiac perfusion was doubled by the addition of the ITD [3,5,38•]. These positive effects quickly dissipated when the valve was taken out of the respiratory circuit. Subsequent animal studies have also demonstrated a significant survival benefit with an associated markedly improved neurologic function with use of the ITD during S-CPR [38•].

The first independent, confirmed benefit of the ITD, by Langhelle *et al.*, demonstrated that the ITD doubled blood flow to the heart when compared with S-CPR [33]. There was also a trend toward increased blood flow to the heart with ACD/ITD CPR. However, no cerebral blood flow benefit was observed with the ITD in this study. The study design and/or the way the animal was prepared may have accounted for the differences. Unlike earlier and subsequent studies, in the Langhelle *et al.* study [33], cerebral blood flow in the control state was already 50% of normal. In addition, Langhelle *et al.* [33] attached an automated piston-like device to the chest wall with small screws and used it to achieve active compression and decompression. This attachment process may alter the elasticity of the chest and may affect outcomes. Finally, each pig received multiple interventions, including S-CPR and ACD CPR, with and without the ITD. This could have increased the potential for cross-contamination. Nevertheless, those studies still confirmed that the use of the ITD doubles blood flow to the heart during CPR.

A recent study by Voelckel *et al.* [37•] demonstrated the remarkable benefit of ACD/ITD CPR when compared with S-CPR. Ventricular fibrillation was induced in 10-kg piglets. To simulate patient "downtime," no treatment was administered for the first 10 minutes. After 10 minutes, the animals received S-CPR delivered with an automated device, followed by ACD/ITD CPR, also delivered with an automated device. Mean arterial pressure, coronary perfusion pressure, and end-tidal carbon dioxide levels increased by approximately 30% when ACD/ITD CPR was used *versus* S-CPR. By contrast, cerebral and cardiac perfusion doubled with the addition of ACD/ITD CPR, despite the prolonged time between arrest and initiation of ACD/ITD CPR. This study also demonstrates how much the measured coronary perfusion pressures underestimate the actual delivery of blood flow to vital organs.

In addition to an increase in mean arterial pressures and cerebral blood flow, the beneficial effects of ACD/ITD CPR can also be seen on cerebral metabolism. Bahlmann *et al.* [43] reported on the use of ACD/ITD CPR on pigs in hypothermic cardiac arrest. The group used microdi-

Figure 4. Porcine model of cardiopulmonary resuscitation (CPR)



Addition of the impedance threshold device (ITD) demonstrated a doubling of vital organ blood flow compared with automated compression device (ACD) CPR alone. Blood flow to the brain was greater in the arrested pig treated with ACD/ITD CPR than in the prearrested pig [37•].

alysis techniques to measure metabolic intermediates, lactate/pyruvate ratios, and glucose levels. When compared with S-CPR, the use of ACD/ITD CPR resulted in higher cerebral perfusion pressures and lower pyruvate/lactate ratios, indicating better oxygen delivery and less anaerobic cerebral metabolism. Additionally, measured cerebral glucose levels were lower in animals treated with ACD/ITD CPR, a finding consistent with a more favorable metabolic status. These findings are consistent with those in other animal and clinical trials, where neurologic outcomes are better with the ITD secondary to improved cerebral perfusion and metabolism.

Studies by Bahlmann *et al.* [43] and Raedler *et al.* [44] also demonstrated another important benefit of ACD/ITD CPR. They found that the use of this device combination enhances drug efficacy during CPR. In hypothermic pigs, the effects of exogenous vasopressin were significantly enhanced with ACD/ITD CPR, as reflected by higher coronary and cerebral perfusion pressures and improved cerebral metabolic profiles.

Clinical studies

Four randomized clinical trials have been performed to date to evaluate the effectiveness and safety of ACD/ITD CPR in humans [30,39••,40••,42••]. The first study focused on hemodynamics in patients with out-of-hospital cardiac arrest [30]. All patients received ACD CPR, the standard of care in France. A total of 10 patients were randomized, in a blinded, prospective trial, to receive ACD CPR with a sham (nonfunctional) ITD. Eleven patients were treated with an active (functional) ITD. In that study, end-tidal carbon dioxide levels rose more rapidly and reached higher levels with the active ITD. Systolic and diastolic blood pressures were nearly normal (109/57 mm Hg) in the active ITD group *versus* 89/35 in the sham ITD group. In addition, return of spontaneous circulation occurred more rapidly in the active ITD group than in the sham ITD group. On the basis of these data, the use of ACD/ITD CPR was recommended as an alternative to S-CPR in the 2000 American Heart Association guidelines (Class IIb recommendation) [9].

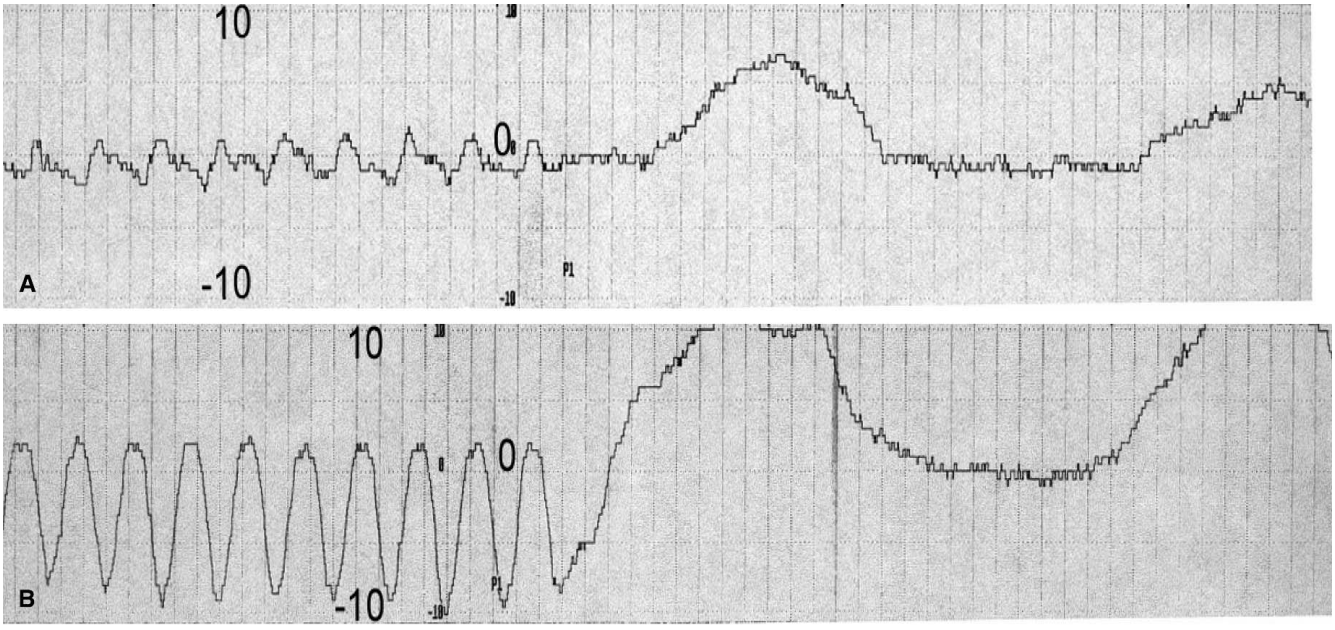
Another interesting recent study showed that the ITD can work well when applied to a facemask [5,30]. This is important because it indicates that inspiratory impedance can be added during basic life support airway management (by first responders and, perhaps, even lay rescuers). In other words, the patient does not have to be endotracheally intubated to achieve benefit from the ITD. Therefore, inspiratory impedance would not be limited to the intubated patient receiving advanced life support. Additionally, this was the first study to measure intrathoracic pressure in cardiac arrest patients undergoing ACD CPR. To perform this study, Plaisance *et al.* [5,30] randomized patients before endotracheal intubation for either a sham or an active ITD. A facemask and device (either sham ITD or active ITD) was first used to ventilate the patient. The same ITD was then used after endotracheal intubation. Intrathoracic pressure was measured by use of an air-filled pressure transducer connected to either the facemask or the endotracheal tube.

A pilot study demonstrated no difference between pressures measured at the level of the cricothyroid membrane and those at the facemask. As shown in the representative tracing (Fig. 5), addition of the active ITD to the facemask resulted in an immediate decrease in intrathoracic pressure during ACD CPR. In many but not all patients, there was no discernible difference between the pressure tracings obtained with the facemask and those obtained with the endotracheal tube. It is important to note that when the facemask was used to ventilate patients, a two-person technique was used to maintain a tight seal between the face and the mask. Additionally, in some patients, a greater intrathoracic vacuum was observed when the endotracheal tube was used. Nonetheless, each time the active ITD was used, a significant reduction in the decompression phase intrathoracic pressure resulted. The active ITD helped both the facemask and the endotracheal tube to generate an intrathoracic vacuum during ACD CPR.

These studies demonstrated, for the first time, the degree of negative intrathoracic pressure achieved with ACD/ITD CPR in humans. The average maximum negative intrathoracic pressure was –11 mm Hg with the active ITD *versus* only –3 mm Hg with the sham ITD. These results demonstrate how useful the ITD is in achieving a vacuum effect during CPR. A second important finding is that it takes as many as 5 compression/decompression cycles to achieve the maximum negative intrathoracic pressure. Moreover, each time an active positive-pressure ventilation was delivered, the decompression phase intrathoracic vacuum was destroyed and required regeneration. Thus, the less frequent the ventilation rate, the greater the blood flow back to the heart. A recent study using S-CPR with the ITD in pigs confirmed this important observation [45]. This has become an important theme for all types of CPR: ventilations interrupt coronary perfusion pressure and should be kept at the minimum required to maintain oxygenation [31].

ACD/ITD CPR improves survival rates after cardiac arrest. A recent prospective, controlled trial was performed in Mainz, Germany, in which the two-tiered emergency response included early defibrillation [39••]. Patients with out-of-hospital arrest of presumed cardiac cause were sequentially randomized to ACD/ITD CPR or S-CPR (the control individuals) by the advanced life support team after intubation. Patients with an initial heart rhythm of ventricular fibrillation (42% of the total) who

Figure 5. Example of intratracheal pressures, a surrogate for intrathoracic pressures, in a patient undergoing automated compression device cardiopulmonary resuscitation (CPR) with the impedance threshold device (ITD) attached to a facemask vs without



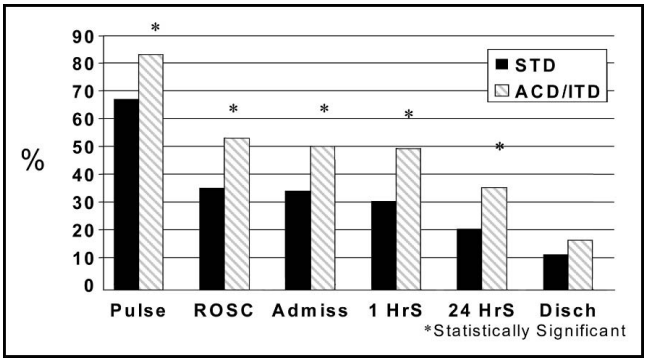
CPR was delivered at 100 compression/decompression cycles/min with a synchronized compression:ventilation ratio of 15:2. **(A)** Use of sham ITD. **(B)** Use of active ITD. Scale: −10 mm Hg to +10 mm Hg. Note the absence of significant decreases in intratracheal pressures with a sham device. With the active ITD, wide fluctuations in intratracheal pressure are seen with each compression and decompression. After 15 compressions and decompressions, two breaths are delivered, resulting in a slow rise in intratracheal pressures [5,30].

could not be resuscitated by basic life support early defibrillation were enrolled in this clinical trial, as well as patients with an initial rhythm of asystole or pulseless electrical activity. Rescuers learned which method of CPR to use at the start of each work shift. The primary endpoint was 1-hour survival after a witnessed arrest. With ACD/ITD CPR (n = 103), return of spontaneous circulation and 1-hour and 24-hour survival rates were 55%, 51%, and 37% *versus* 37%, 32%, and 22% for S-CPR (n = 107) (P = 0.016, 0.006, and 0.033), respectively (Fig. 6).

One-hour and 24-hour survival rates in witnessed arrests were 55% and 41% with ACD/ITD CPR *versus* 33% and 23% in control subjects (P = 0.011 and 0.019), respectively [39••]. One-hour and 24-hour survival rates in patients with a witnessed arrest in ventricular fibrillation were 68% and 58% after ACD/ITD CPR *versus* 27% and 23% after S-CPR (P = 0.002 and 0.009), respectively, as shown in Figure 7. Hospital discharge rates were 18% after ACD/ITD CPR *versus* 13% in control participants (P = 0.41). Overall neurologic function trended higher in patients receiving ACD/ITD CPR than in control participants (P = 0.07), as shown in Figure 8. Importantly, patients randomized more than 10 minutes after the call for help to the ACD/ITD CPR group had a greater than three times higher 1-hour survival rate (44%) than did control participants (14%) (P = 0.002). These time-related benefits were observed regardless of presenting rhythm. Neurologic outcomes in the survivors with delays to treatment with ACD/ITD CPR were similar to those who were treated with ACD/ITD CPR more rapidly. These important clinical data emphasize the importance of an active compression decompression device used in combination with an ITD.

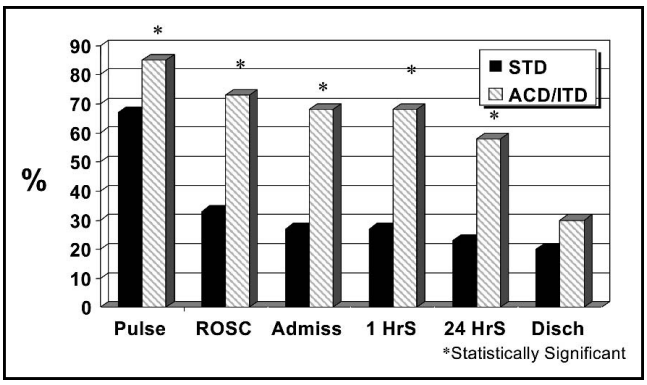
Additional support of these findings was provided by another study recently performed in France [40••]. All 400 patients in that study received ACD CPR. ACD

Figure 6. Outcomes in a comparison of standard (STD) with active compression decompression/impedance threshold device (ACD/ITD) cardiopulmonary resuscitation (n = 210) in Mainz, Germany



Pulse, palpable pulse with CPR; ROSC, return of spontaneous circulation; Admiss, hospital admission; 1HrS, 1 hour survival; 24HrS, 24 hour survival; Disch, discharge [39••].

Figure 7. Outcomes in patients randomized to either standard (STD) or active compression decompression/impedance threshold device (ACD/ITD) cardiopulmonary resuscitation (CPR) with witnessed ventricular fibrillation in Mainz, Germany (n = 70)



Pulse, palpable pulse with CPR; ROSC, return of spontaneous circulation; Admiss, hospital admission; 1HrS, 1 hour survival; 24HrS, 24 hour survival; Disch, discharge [39••].

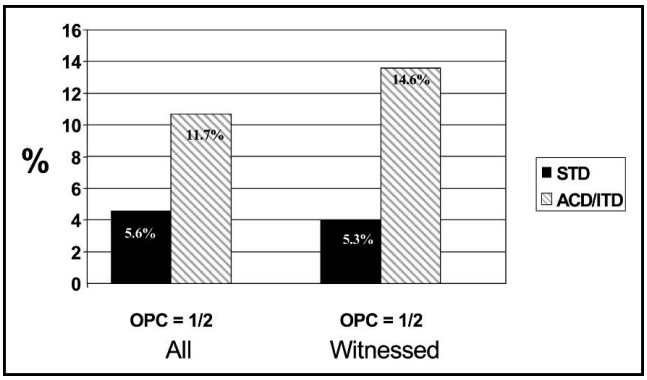
CPR has been the method of CPR used by the emergency services in France for nearly a decade. In one arm, 200 patients were treated by advanced life support personnel with ACD CPR and an active ITD, and the other 200 patients were randomized to the control group and received treatment with ACD CPR and a sham ITD. The study was sized and powered from a statistical standpoint to determine a potential difference in 24-hour survival rates. As in other studies from France [21,22,40••], most of the patients had an initial rhythm of asystole. The group treated with ACD CPR and an active ITD had a 24-hour survival rate of *32%*, compared with a 24-hour survival rate of *22%* in the control population ($P < 0.05$). Survival rates in both groups were very low, but differences in neurologic function in the survivors trended in favor of the ACD/ITD CPR group. Only 1/8 survivors treated with the sham device had normal cerebral function at the time of hospital discharge *versus* 6/10 in the function ITD group ($P < 0.07$). The vast majority of these patients, including the survivors, had an initial heart rhythm of asystole. Despite having a rhythm with a very poor prognosis, *32%* of the patients lived for longer than 24 hours after treatment with ACD/ITD CPR.

Conclusion

It has been nearly 50 years since the concept of mouth-to-mouth resuscitation and closed chest cardiac massage was first described. Only recently have mechanical means been developed that are capable of achieving nearly normal blood pressures in patients undergoing cardiac arrest. While these patients still received low-dose epinephrine, four recent clinical studies have demonstrated the benefit of ACD/ITD CPR over S-CPR for patients in cardiac arrest. In patients with an initial heart rhythm of ventricular fibrillation, the vital importance of perfusion before and after defibrillation has been demonstrated by both Cobb *et al.* [46] and Wik *et al.* [47]. Priming the pump increases the chances for successful conversion of ventricular fibrillation and for resuscitation of nonventricular fibrillation rhythms. In addition, animal and human studies demonstrate that in patients with prolonged cardiac arrest, regardless of the initial heart rhythm, the use of ACD/ITD CPR has a marked impact on the chances for successful resuscitation. [37•, 39••,42••] From this perspective, the improved perfusion achieved with ACD/ITD CPR fits nicely into the three phases of CPR paradigm recently described by Weisfeldt and Becker [48]; after the first 5 minutes, increased circulation is essential for successful resuscitation.

Despite the increased perfusion achieved with ACD/ITD CPR, most patients still die before hospital discharge. From this perspective, the use of ACD/ITD CPR is not a panacea. Much has been learned in recent years about the importance of postresuscitation care. Without additional measures to help preserve brain and cardiac function in the postresuscitation phase, many patients will still be lost. However, recent studies suggest that improved perfusion with ACD/ITD CPR, coupled with hypothermia and other metabolic hibernation strategies, improves measures to preserve vital organ function after resuscitation [49,50]. This combination promises to markedly increase the chances for survival in years to come. A recent study by Nadkarni *et al.* [51] suggests that the combination of ACD/ITD CPR and cooling can result in increased survival rates and jump-start the cooling process. This may be important, given the rapidity of cellular damage and apoptosis after cardiac arrest and successful resuscitation in the absence of postresuscita-

Figure 8. In patients randomized to treatment with standard (STD) CPR, neurologic outcomes tended to be worse in comparison with active compression decompression/impedance threshold device (ACD/ITD) treatment in a study from Mainz, Germany



Overall performance category (OPC), overall performance category score. OPC of 1 = normal neurologic function. OPC of 2 = mild neurologic deficit. OPC 1/2 = patient scored either a 1 or a 2 [39••].

tive care that includes some form of metabolic preservation.

The ITD was recently approved as a circulatory enhancer device by the United States Food and Drug Administration. The ACD CPR device has not yet been approved. The ITD can be used with any method of CPR that involves external chest compression. On the basis of the study from Mainz, Germany, widespread use of ACD/ITD CPR in patients with a witnessed, out-of-hospital, ventricular fibrillation cardiac arrest could increase 24-hour survival rates by an additional 100,000 patients out of the 300,000 patients known to die each year in Europe and North America [39••]. When coupled with improved postresuscitation care, the potential impact of widespread use of this technology on the number one cause of death in adults could be striking [50,51].

Modern, rigorous, scientific methods have been used to study ACD/ITD CPR—a scrutiny S-CPR has not undergone. This device combination has given researchers new hope for finally realizing a CPR technique that is superior to S-CPR. The present data support the widespread use of this new technology. Ongoing clinical trials will help determine the effect of ACD/ITD CPR use on hospital discharge rates and neurologic outcomes.

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