Combined active compression-decompression cardiopulmonary resuscitation and inspiratory impedance threshold devices may improve survival after out-of-hospital cardiac arrest

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Introduction

In recent decades, a number of devices and alternatives to conventional manual cardio-pulmonary resuscitation (CPR) have been developed. The driving force behind these developments has been the desire to enhance perfusion during attempted resuscitation from cardiac arrest. The ultimate goal of device-assisted resuscitation during cardiac arrest is long-term survival with preservation of brain function.

The Lancet has published two studies [1, 2] comparing standard CPR to CPR using ResQPod combined with another assist device called the ResQPump (Fig. 1), both from Advanced Circulatory Systems, Inc (Roseville, MN, USA).

ResQPump is an active compression-decompression (ACD) hand-held device and ResQPod is an inspiratory impedance threshold valve device (ITD). The application of these two devices during CPR (ITD + ACD-CPR) as shown in Fig. 1 results in an increase in blood flow to the heart and brain compared to standard (S-) CPR and improves clinical outcomes (Fig. 2).

The ResQPump (Fig. 3) is a hand-held, ACD CPR device placed in the same position on the sternum as the hands, enabling rescuers to perform similar chest compressions as in conventional CPR. Instead of allowing the chest wall to recoil passively however, rescuers pull up on the ResQPump’s handle with its suction cup. This provides active decompression of the chest, promotes optimal chest wall recoil, and creates a negative intrathoracic pressure (vacuum) that helps the return of blood to the heart. The handle contains a force gauge and metronome that guide compression depth, recoil and rate. The ResQPump is identical to the CardioPump, an ACD-CPR device available outside the United States.

The ITD (Fig. 4) is a small, 35-mL device that fits on a face mask or an endotracheal tube. The pressure-sensitive valves within the ITD impede the influx of inspiratory gas during chest wall decompression, thereby augmenting the amplitude and duration of the vacuum within the thorax. This vacuum draws more venous blood back into the heart, resulting in increased cardiac preload, followed by improved cardiac output and vital organ perfusion.

It is attached within the ventilation circuit between the airway device and the ventilation source. By selectively restricting airflow during CPR, the device creates a small but important negative pressure (vacuum) in the chest. This has been shown in numerous human and animal clinical trials to increase blood flow back to the patient’s heart during CPR.

ResQTrial summary

The trial was conducted at seven co-ordinating EMS sites in the US: Minneapolis, MN; St. Paul, MN; Whatcom County, WA; Oshkosh, WI; Oakland and Macomb Counties, MI; Washtenaw and Livingston Counties, MI; Indianapolis, Indianapolis.

It included 46 EMS agencies in urban, suburban and rural areas, covering a total population of 2.3 million. The study protocol was reviewed and approved by 25 participating Hospital Review Boards.

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Figure 1. Cardio-pulmonary resuscitation using ResQPod combined with an active compression-decompression assist device called a ResQPump.

Figure 2. Improved perfusion using an active compression-decompression ResQPump device.

Figure 3. An active compression-decompression hand-held device, ResQPump.

Figure 4. Inspiratory impedance threshold valve device, ResQPod.
**Study population**

A total of 2,470 patients were randomized, with 1,653 of them meeting the final criteria: 813 in the control group and 840 in the intervention group.

Eligible patients experiencing presumed cardiac arrest were randomized over about three and a half years to undergo CPR either with, or without, the ACD technique.

The hospital-survival rate was statistically similar in the intervention group and the control group, but the primary end point of hospital discharge with good neurological function (defined as a modified Rankin score ≤ 3) was significantly higher in the intervention group (odds ratio [OR] 1.58, 95% confidence interval [CI] 1.07–2.36; p = 0.019). Those patients also showed better one-year survival, but a significantly increased rate of pulmonary edema (Table 1).

**Key trial results**

- Patients in the intervention group had a 53% relative increase in survival to hospital discharge with a mRS score of ≤ 3 (primary endpoint): 75/840 (8.9%) vs 47/813 (5.8%), p = 0.019, OR 1.58, 95% CI 1.07–2.36.
- There were no survivors in either group if CPR was initiated > 10 min after the call to the emergency services.
- For patients with an initial recorded rhythm of VF/VT: survival to hospital discharge with MRS ≤ 3 was greater in the intervention group: 23% vs 17%, p = 0.0645 (non-significant).
- One year after cardiac arrest, there was a 49% increase in survival in the intervention group: 74/840 (8.8%) vs 48/813 (5.9%) in the control group, p = 0.030.
- The overall rate of major adverse events was not significantly different between groups. There were more reports of pulmonary edema in the intervention group, but there was also increased survival in this group.
- Neurological function was similar between groups at 90 days and one year after cardiac arrest. There was no increase in the number of patients with severe neurological impairment in the intervention group.
- Results were consistent across study sites, patient age groups, gender.

**Conclusions**

- ACD-CPR with augmentation of negative intrathoracic pressure using an ITD improves survival to hospital discharge with favorable neurological function. The survival benefit persisted to one year following cardiac arrest.
- The combination of ITD + ACD-CPR has an acceptable safety profile for use in patients with cardiac arrest.
- The combination of ITD + ACD-CPR is feasible to teach and implement in a variety of EMS environments.
- These results support the routine use of ITD + ACD-CPR during cardiac arrest to increase survival to hospital discharge with favorable neurological function.

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**References**