Introduction

Standard cardiopulmonary resuscitation (S-CPR) refers to the entire body of techniques of external chest compression and securing positive pressure ventilation for the purpose of achieving adequate blood and oxygen flow into vital organs such as the heart and brain following cardiac arrest (1). The current application of S-CPR is based on the technique of “external chest compression” that was defined by Kouwenhoven in 1960 and comprises the phases of active compression and passive decompression. Despite the evolution of resuscitation medicine, the limited improvement in survival rates following cardiac arrest has led researchers to explore the possibility of different CPR techniques and also to develop devices that support ventilation and circulation (2-6). This manuscript was prepared to review the experimental and clinical studies conducted on the historical progress and effectiveness of mechanical chest compression devices (MCCD).

Limitations of S-CPR

The fundamental goal of effective CPR applications is to achieve return of spontaneous circulation (ROSC) and a good neurological outcome and the return of the patient to the patient’s previous quality of life and functional level of health. Guidelines emphasize the importance of effective chest compression for successful CPR. The effectiveness of chest compressions depends on a couple of parameters (such as application of compressions to the right place, at an adequate depth and rate, on a regular and uninterrupted basis; letting the chest to fully recoil after each compression; avoiding over-ventilation; and maintaining a balance between compression and ventilation) (1, 5, 7, 8).

One of the basic problems related to S-CPR techniques is that even in the most effective chest compressions, a physiologically adequate amount of cardiac output may not be reached because the quality of compression may change over time, this may cause cerebral and coronary blood flow to reduce even further as a result of the interruptions (9-11). If the chest compressions could reach the needed depth, as it does in infants and children, a higher intrathoracic pressure and cardiac output would be possible (12, 13). Another problem is that the quality of CPR is limited to the degree of knowledge, experience, and endurance of the rescuer (9, 14-17). Transferring the patient into an ambulance, discontinuing CPR prior to defibrillation, the difficulty of effectively applying the technique in a moving ambulance, failure to maintain the relationship between compression and ventilation, and reduced elastic recoil of the chest...
Mechanical Chest Compression Devices: Definition, History, and Classification

Definition
MCCDs are noninvasive circulation support devices that function manually, pneumatically, or electrically and in accordance with CPR guidelines, provide uninterrupted and effective external chest compression to achieve an adequate blood flow to the heart and other vital organs during non-traumatic adult cardiac arrest. MCCDs can be used as an alternative to S-CPR in cases that may hinder effective compressions such as prolonged CPR during the transport of the patients or in the shortage of personnel (2, 6, 10, 18, 19).

These devices are included in the guidelines of the American Heart Association (AHA) under the heading “circulatory support devices” (6). They are described by the U.S. Food and Drug Administration (FDA) as “External cardiac compressors” (19). In the literature, the nomenclature varies, and “external cardiac compression devices,” “automatic chest-compression devices,” and “mechanical CPR devices” are some of the terms of reference (2, 10, 13, 18).

History
The advent of MCCDs is not new. These devices began to be developed in the beginning of the 1960s, when resuscitation medicine was merely in its infancy. Chronologically speaking, the “electro-pneumatic machine” developed by Harkins and Bramson (20) in 1961; the “portable pneumatic pump” developed by Nachlas and Siedband (21) in 1962; the “Beck-Rand external cardiac compression machine” developed by Safar et al. (22) in 1963; the “cardiac massage machine” developed by Bailey and Browse in 1964 (23); and the “hospital mechanical pump” developed by Nachlas and Siedband (21) in 1965 were the first MCCDs to be invented. In later years, experiments were conducted with many manually operated devices such as the “cardio-massager,” “cardio-pulsar,” pneumatic “iron heart,” and “Trafenol LR50-90” (13, 24).

Many of the first developed and tested of these devices were very complex, too heavy, or ineffective for use in CPR; therefore, they were found to be nonfunctional and unacceptable for the clinical setting. On the other hand, since the 2000s, many devices have begun to be developed and have found a clinical area of use, and the literature of the effects of these devices on CPR outcomes are steadily increasing.

Classification
These devices are different from one another in terms of their working principles, the energy they consume, and their electronic features. MCCDs used currently can be classified as follows:
1. Piston-driven CPR devices (PD-CPR),
2. Load-distributing band CPR devices (LDB-CPR).
In addition to these two fundamental groups, third-generation devices, which combine different working mechanisms and different CPR techniques, have been used in recent years, aiming to increase the hemodynamic effects of S-CPR. These are as follows:
3. Active compression–decompression CPR devices (ACD-CPR),
4. Simultaneous sterno-thoracic cardiopulmonary resuscitation devices (SST-CPR/S-CPR/X–CPR),
5. Inspiratory impedance threshold valve/devices and ResQCPR (ACD + ITD CPR),
6. Phased thoracic-abdominal active compression–decompression CPR devices (PTACD-CPR), and
7. Active compression–decompression CPR with enhanced external counterpulsation and the inspiratory impedance threshold valve (AEI-CPR) (2, 6, 13, 25-29).
The devices in these groups and their working principles have been discussed below.

1. Piston-driven devices (PD-CPR)
These are based on the “cardiac pump theory” and are first-generation mechanical devices that use a piston to exert “single-point compression” on the sternum. One of the first examples in this group was the Pneumatically Run Thumper. A more developed model of this device is the Thumper Mechanical CPR Device Model 1007 and its updated model the Life-Stat. The Life-Stat consists of a backboard attached to a column and operates pneumatically with a piston. It has a ventilator that is meant to be used in conjunction with chest compression (Figure 1a) (Michigan Instruments, USA). Mechanical piston-driven devices that are operated manually work with a lever system and are marketed under brand names such as the “Animax Mono” (Figure 1b) (AAT Alber Antiriebtechnik GmbH, Albstadt, Germany) and the “CPR RsQAssist,” which employs an audio-visual metronome (10, 13, 24, 26, 29).

2. Load-distributing band devices (LDB-CPR)
“Load-distributing band devices” are based on the “thoracic pump theory” and represent second-generation mechanical chest compression technology. These devices exert thoracic compression on the anterior-antero-lateral thorax using a wide pneumatic band that wraps around the chest, inflating and deflating at cyclically. The basic equipment in these devices consists of a backboard, a chest compression band (load-distributing Life Band), and a power system. The first example of this type of device was the “Vest-CPR.” Currently, devices that work with this mechanism are marketed under commercial names such as the pneumatic Automated CPR Vest (Reax resuscitation device) and the pneumatic or electrical AutoPulse (Figure 2). Studies show that the chest compression achieved all around the chest with the AutoPulse creates higher coronary perfusion pressure than sternal pressure (13, 24, 25, 29-33).

3. Active compression–decompression CPR devices (ACD-CPR)
ACD-CPR devices are third-generation devices that work on the piston principle. These devices were developed based on a news article published in 1990 about a successful resuscitation attempt of a lay person performed with a toilet plunger to his father (34, 35). As is known, in S-CPR, the return of blood to the heart is dependent only on the passive recoil of the chest wall. The principle behind this technique may be summarized as the pumping of blood outside of the thorax through positive pressure in the active compression phase and then exerting an external negative vacu-
um, creating an intrathoracic negative pressure during the active decompression phase to increase the venous return of blood to the heart. Thus, in the next compression phase, an increase is achieved in cardiac output, coronary and cerebral blood flow, and in arterial blood pressure. The most widely known and commonly used of the ACD-CPR devices is the LUCAS (Lund University Cardiac Assist System). The original LUCAS 1 was a pneumatic device that was developed in Sweden by Steen in 2002. The new model of the device, introduced in 2010 under its new name LUCAS 2 (LUCAS™ 2 Chest Compression System), runs on electricity and consists of a piston for compression, a silicone suction cup for decompression, a rechargeable battery, a backboard, and connecting legs (Figure 3a). The device allows defibrillation without interrupting compression, and its X-ray translucent capability makes cardiac catheterization possible. The ACDC Thumper is another pneumatic device. The manual devices that operate with the ACD-CPR technique are marketed under trade names such as CardioPump, ResQPump, and Ambu® Cardio Pump (Figure 3b) (6, 13, 24, 29, 36-40).

4. Simultaneous sterno-thoracic CPR devices (SST-CPR / X-CPR)

These devices were designed to benefit from both the cardiac pump and thoracic pump theories. These devices have two components: a piston (which depresses the sternum in the compression phase) and a circumferential band (which constricts the thorax simultaneously compressions). The "Life Belt" is a device that is operated manually using the SST-CPR principle. Another such device is the pneumatic "Weil Mini Chest Compressor" (Figure 4) (Resuscitation International, USA) (13, 41-44).

5. Inspiratory Impedance Threshold Device (ITD) and ResQCPR (ACD+ITD CPR)

The inspiratory ITD is a pressure-sensitive one-way valve system that can be connected to a face mask or to any developed airways equipment such as endotracheal tubing. The valve closes in the decompression phase of CPR, temporarily blocking the more than necessary passage of passive air through the open airway into the patient’s lungs, thus decreasing intrathoracic pressure and creating a small vacuum. This increases the flow of venous blood to the heart, and the increased venous return increases cardiac output in the next compression. ITD are marketed under the trademark “ResQPOD® ITD 16.” ITD can be used alone during S-CPR as well as it may be used in combination with manual ACD-CPR devices such as the CardioPump and the ResQPump. This system is known as ResQCPR. ResQCPR=ACD-CPR (ResQPUMP)+ITD (ResQPOD) (Figure 5a) (6, 27, 45-47).
6. Phased thoracic-abdominal active compression–decompression CPR devices (PTACD-CPR)

Interposed abdominal compression CPR (IAC-CPR) activates the abdominal venous reservoir by increasing abdominal pressure; this CPR technique is based on forcing venous return, thereby increasing venous return to the heart. Abdominal compression is applied to the area midpoint between the xiphoid and umbilicus in the relaxation phase of chest compression. Phased thoracic-abdominal active compression–decompression CPR constitutes the working principle behind ACD-CPR and IAC-CPR and is a new method that combines the two techniques. PTACD-CPR is applied by simultaneous chest compression (positive intrathoracic pressure) and active abdominal decompression and then following this phase, simultaneous active chest decompression (negative intrathoracic pressure) and abdominal compression. The Lifestick™ was developed for use in this technique; it is a manually controlled device. The device consists of a rigid central bar and two arms with adhesive pads that are connected to this rigid bar. The larger adhesive pad is placed over the abdomen and the smaller over the anterior chest wall. An implementer compresses the two sides of the device just like a seesaw, applying pressure both on the chest and the abdomen alternately (Figure 5b) (11, 27, 48-50).

7. Active compression–decompression CPR with Enhanced External Counterpulsation and the Inspiratory Impedance Threshold Valve (AEI-CPR)

Enhanced external counterpulsation (EECP) is a circulatory support system that achieves increased cardiac output using a method whereby cuffs applied to the lower extremities are inflated during diastole to increase coronary blood circulation and deflated at the early systole to reduce afterload and increase venous return. AEI-CPR is another experimental technique, which is a combination of active compression–decompression CPR, EECP, and Inspiratory impedance threshold valve, aiming to improve CPR hemodynamics and increase survival rates. This technique, which is still in its theoretical and experimental stage, is simply expressed as AEI-CPR=ACD-CPR+EECP+ITV (51, 52).

Studies Conducted on the Effectiveness of MCCDs

Theoretically, MCCDs appear to provide many practical advantages, such as the mechanical devices deliver compressions at the same frequency and depth which are recommended in the guidelines, as opposed to the inter-rescuer variations and fatigue factors that affect the quality of chest compression; these devices allow the rescuers to perform other tasks (cannulation, airway, etc.) and defibrillation without the need of interruption in CPR; and they provide consistent rate and depth of chest compressions during transport of the patient.

However, the main issue is to what degree these devices have an impact on survival in cardiac arrest, on hemodynamic parameters, and on the survival neurologically intact and whether they produce a significant difference in in-hospital and out-of-hospital cardiac arrests (IHCA and OHCA, respectively) compared to S-CPR. Experimental studies conducted with some mechanical chest compression devices developed in recent years present strong evidence that these devices increase the effectiveness and quality of CPR. Moreover, although they were first developed to achieve uninterrupted CPR, particularly in cases of OHCA, the studies on the use of these devices in IHCA help to expand their clinical usage area. This section will review some of the results of some MCCD-related experimental or clinical studies on in-hospital and out-of-hospital cases.

Experimental studies with LUCAS-CPR have shown that the device enables significantly higher cerebral blood circulation than S-CPR as well as higher rates of cardiac output, carotid artery blood...
Table 1. Studies with LUCAS-CPR

<table>
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<th>Study design</th>
<th>Results</th>
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<tr>
<td>1. Rubertsson S (2005). Increased cortical cerebral blood flow with LUCAS: a new device for mechanical chest compressions compared to standard external compressions during experimental cardiopulmonary resuscitation (9)</td>
<td>Experimental: pigs: VF was induced (n=14), L-KPR (n=7), S-KPR (n=7)</td>
<td>Mean cortical cerebral blood flow and ETCO2 was significantly higher in L-CPR than in S-CPR (p&lt;0.01).</td>
<td>L-CPR generated higher cerebral blood flow and cardiac output than S-CPR. The results strongly support prospective randomized studies in patients to evaluate the effects of this device in clinical practice.</td>
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<tr>
<td>2. Steen S (2002). Evaluation of LUCAS, a new device for automatic mechanical compression and active decompression resuscitation (36)</td>
<td>Experimental: In an artificial thorax model and pigs: VF was induced</td>
<td>In thorax model: Superior pressure and flow were obtained with L-CPR compared with S-CPR. In pigs: higher CO, carotid artery blood flow, ETCO2, and CPP were obtained with L-CPR (83% ROSC) than with S-CPR (0% ROSC).</td>
<td>LUCAS gave significantly better circulation during ventricular fibrillation than manual chest compressions.</td>
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<tr>
<td>3. Liao Q (2010). Manual versus mechanical cardiopulmonary resuscitation. An experimental study in pigs (53)</td>
<td>Experimental: pigs: VF was induced (n=16), L-KPR (n=8), S-KPR (n=8)</td>
<td>ROSC: L-CPR (n=8), S-CPR (n=3). The mean CPP: L-CPR 20, S-CPR 5 mmHg, p&lt;0.01, ETCO2: higher in the L-CPR group (p&lt;0.05).</td>
<td>L-CPR generated higher CPP than S-CPR.</td>
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<td>4. Larsen A (2007). Cardiac arrest with continuous mechanical chest compression during percutaneous coronary intervention. A report on the use of the LUCAS device (54)</td>
<td>Clinical study: IHCA: LUCAS has been used During PCI (n=13)</td>
<td>The device allowed visualization of the coronary arteries in all patients, PCI was successfully performed in eight patients.</td>
<td>Coronary angiography and coronary intervention may be successfully performed in patients with cardiac arrest using the LUCAS device.</td>
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<tr>
<td>5. Bonnemeier H (2009). Automated continuous chest compression for in-hospital cardiopulmonary resuscitation of patients with pulseless electrical activity: A report of five cases (55)</td>
<td>Clinical study: IHCA, LUCAS has been used During PCI (n=5)</td>
<td>The device allows for uninterrupted chest compressions during angiography and angioplasty. CT evidences show that L-CPR may also provide additional therapeutic effects in those patients with PEA due to PE, mechanical thrombus fragmentation, and increase pulmonary artery flow after LUCAS-compression.</td>
<td>LUCAS may significantly improve the chain of survival and clinical outcome in patients with IHCA.</td>
</tr>
<tr>
<td>6. Bonnemeier H (2011). Continuous Mechanical chest compression during in hospital cardiopulmonary resuscitation of patients with pulseless electrical activity (56)</td>
<td>Clinical study: IHCA, L-CPR, patients with PEA (n=28) L-CPR, During coronary angiography and pulmonary angiography (n=21),</td>
<td>ROSC: n=27, Dying within the first hour (n=10), 24 h (n=3) after CPR, Discharged from hospital CPC 1 and 2: n=13, PE (n=14), did not undergo thrombolytic therapy (n=6/14), CT angiography in these patients showed fragmentation of the thrombus.</td>
<td>Continuous chest compression with LUCAS seems to be feasible, safe, and might improve outcomes after IHCA of PEA cardiac arrest. Patients with PE may benefit probably because of thrombus fragmentation and increased pulmonary artery blood flow.</td>
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<tr>
<td>7. Wagner H (2010). Cardiac arrest in the catheterization laboratory: a 5-year experience of using mechanical chest compressions to facilitate PCI during prolonged resuscitation efforts (57)</td>
<td>Clinical study: Retrospective 2004-2008, IHCA: During PCI using LUCAS (n=43)</td>
<td>The PCI procedures were successfully performed during mechanical chest compressions (n=36) and pericardiocentesis (n=1).</td>
<td>Discharge CPC 1: &gt;25% (n=11) Mechanical chest compressions devices enable continued chest compressions during PCI with maintained circulation, which may reduce mortality in patients with cardiac arrest, requiring lengthy CPR, in the catheterization laboratory.</td>
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<td>8. Fidler R (2014). Three modes of cardiac compressions in a single patient: A comparison of usual manual compressions, automated compressions, and open cardiac massage (58)</td>
<td>Case report: Post-CABG patient receiving three modes of cardiac compressions S-CPR, L-CPR, and open cardiac massage</td>
<td>S-CPR (8 min): Average arterial pressures=65/10 mmHg L-CPR (10 min): Average arterial pressures=100/60 mmHg, Open cardiac massage: Average arterial pressures=70/15 mmHg</td>
<td>LUCAS-2 could provide superior arterial blood pressure compared to S-CPR and open cardiac massage.</td>
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flow, end tidal CO₂ (ETCO₂), aortic and coronary perfusion pressure, and ROSC (9, 36, 53). The LUCAS device has been used in IHCA situations, in cardiac catheterization laboratories, and intensive care units. Studies show that the LUCAS device is functional during percutaneous coronary interventions (54-58). The findings reported in the mentioned studies have been summarized in Table 1. The use and effectiveness of mechanical chest compression devices such as LUCAS in organ transplants from non-heart-beating donors, in situations where a decision to terminate life and execute an organ transplant has been made, and where CPR is continued until the start of extracorporeal oxygenation (ECMO) are the subjects of ongoing studies (59).

Details of LUCAS-CPR studies with nontraumatic adult OHCA patients are shown in Table 1. Axelsson et al. (60) and Smekal et al. (61) have reported no significant differences in their studies when compared with S-CPR, whereas the same team in another study (62) revealed that ETCO₂ values, which are a prognostic value for cardiac output and survival, were significantly higher in the LUCAS group.
than in the S-CPR group. In two large randomized studies ("LINC-LUCAS in Cardiac Arrest" and "PARAMEDIC-The Prehospital Randomised Assessment of a Mechanical Compression Device In Cardiac Arrest"), no significant difference was found between LUCAS-CPR and S-CPR in terms of ROSC and survival with good neurological outcomes (63, 64). In another study in which the skills of healthcare personnel in using LUCAS were evaluated, it was determined that the rate and depth of compressions applied to a manikin using LUCAS were inadequate compared with S-CPR (65).

Table 2 displays the findings of studies conducted on the effectiveness of AutoPulse in CPR (A-CPR). A study conducted with IHCA patients where AutoPulse was used reported that A-CPR produced higher coronary perfusion pressure than S-CPR (66). In two non-randomized studies using AutoPulse in OHCA patients, it was shown that A-CPR produced better results than S-CPR (32, 67). In a multi-center randomized study, the results of the ASPIRE Trial (Assisted Prehospital International Resuscitation Research) indicated that survival to hospital discharge and good neurological outcomes were lower in A-CPR than in S-CPR (68). The results of two nonrandomized, small sample size studies support the effectiveness of A-CPR (69, 70). Another large, randomized study (30) reports that survival rates with ROSC and satisfactory neurological outcomes were better with A-CPR than with S-CPR. A review of the results of the "Circulation-Improving Resuscitation Care (CIRC) Trial," another multicenter randomized study conducted with AutoPulse, revealed that A-CPR is equal to S-CPR in terms of ROSC and survival rates (71).

Table 3 presents the results of some studies that have reported on other MCCDs and techniques. Some trials on ResQCPR (ACD+ITD CPR) have reported short- and long-term survival rates to be higher than those for S-CPR (47, 72, 73). The results of trials with simultaneous sterno-thoracic CPR devices (SST-CPR/X-CPR) reveal hope for the future of these devices. In a study that consisted of a small series of cases, X-CPR produced higher coronary perfusion pressure than S-CPR (42). In a study with the Lifestick, a phased thoracic-abdominal active compression–decompression CPR device, no difference was detected compared with S-CPR in terms of ROSC, but it was reported that this technique could be advantageous for patients with asystole or pulseless electrical activity (48). Another study conducted with a small sample (49) did not report any statistically significant difference between using the Lifestick and applying S-CPR.

In the literature, there are also simulation studies where CPR devices and techniques were compared. Zhang et al. (11) used a circulation computer model in an experimental study to compare five ITD-supported techniques (S-CPR, ACD-CPR, IAC-CPR, Lifestick-CPR, and EEEP-CPR) in terms of their hemodynamic effects, and they found Lifestick-CPR to be the most effective. A similar simulation study of five CPR techniques (S-CPR, ACD-CPR, IAC-CPR, ACD-CPR+External counterpulsation (ECP), and S-CPR+ECP) had made a comparison and found that cardiac output, cerebral blood flow, coronary blood flow, and mean coronary perfusion pressure to be the lowest in S-CPR and highest in IAC-CPR, with ACD-CPR+ECP exhibiting values close to this (51).

**MCCDs in the Guidelines**

An evaluation was made of the recommendations for use and the levels of evidence cited in the AHA 2010 and 2015 guidelines based on large randomized trials. In the case of automatic ACD-CPR devices such as LUCAS and LDB-CPR devices such as AutoPulse, the guidelines state that the evidence to support or reject the routine use of these devices in the treatment of cardiac arrest is not sufficient and that manual chest compressions remain as the standard treatment of cardiac arrest; however, these devices may be a reasonable alternative for use by properly trained personnel (AHA 2015: Class IIb, LOE B-R). Furthermore, the guidelines state that the use of mechanical piston devices may be considered in specific settings where the delivery of high-quality manual compressions may be challenging or dangerous for the provider (e.g., limited rescuers available and prolonged CPR during hypothermic cardiac arrest, in a moving ambulance, in the angiography suite, and during preparation for extracorporeal CPR (ECPR)), provided that rescuers strictly limit interruptions in CPR during deployment and removal of the devices (AHA 2015: Class IIb, LOE C-E0) (6, 74).

The AHA 2010 guideline states with regard to manual ACD-CPR devices that there is no adequate evidence to either recommend or reject the routine use of these devices and that the use of the devices may be considered in the event of properly trained personnel. The AHA 2015 guideline has not made any revision with regard to these devices, maintaining the same recommendations and evidence level specified in the 2010 guideline (Class IIb, LOE B) (6, 74).

With respect to the sole use of the ITD-CPR device, the AHA 2010 guideline's recommendation and evidence level places the device in Class IIb, LOE B, whereas the AHA 2015 guideline has changed the recommendation and evidence level, placing it in the category of "Not recommended for routine use S-CPR" (Class III: No benefit, LOE A) (6, 74).

There appears to be no evaluation in the AHA 2010 guideline for ITD+ACD-CPR (RESQCPR). In the 2015 guideline, however, it is stated that this combination is not recommended for routine use as an alternative for S-CPR but may be considered as an alternative only in the presence of available equipment and trained personnel (Class IIb, LOE C-LD) (6, 74).

It can be seen that the AHA 2010 and 2015 guidelines do not include information and data on CPR devices and combinations such as the SST-CPR, PTACD-CPR, and AEI-CPR because these are still in the experimental stage and are not supported by adequate clinical research (6, 74).

**Conclusion**

In a general assessment, it may be stated that although large randomized trials have as yet highlighted the superiority of these devices over S-CPR in OHCA, they have at the same time not produced any evidence to prove their failure or harm.

The possibilities to be created by the harmonious cooperation of the disciplines of mathematics, biology, medicine, engineering, and the physical sciences in “the process of developing biomedical equipment technologies” and the role technology will play in constructing the future cannot be ignored. Therefore, an increase in the number of experimental and clinical research on CPR technologies and the evaluations & revisions performed according to the results of these studies will pave the way for the development of changes with respect to the application methods and areas of these devices. Ensuring that these devices become more functional, effective, and reliable will improve the effectiveness of CPR and may reduce the incidence of morbidity and mortality accompanying cardiac arrest.
Table 2. Studies with Autopulse

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<tr>
<th>Title</th>
<th>Study design</th>
<th>Results</th>
<th>Conclusion</th>
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<tr>
<td>1. Timerman S (2004). Improved hemodynamic performance with a novel chest compression device during treatment of in-hospital cardiac arrest (66)</td>
<td>IHCA (at ICU), Brazil, 2000-2001, A-CPR (n=8), S-CPR (n=8)</td>
<td>Peak aortic pressure: A-CPR=153, S-CPR=115 mmHg, p&lt;0.0001 Peak right atrial pressure: A-CPR=129, S-CPR=83 mmHg, p&lt;0.0001 CPP: A-CPR=20 mmHg, S-CPR=15 mmHg, p&lt;0.015</td>
<td>A-CPR demonstrated a clinically significant improvement in hemodynamics compared to manual chest compressions.</td>
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<td>2. Hock Ong ME (2006). Use of an automated, load-distributing band chest compression device for out-of-hospital cardiac arrest resuscitation (32)</td>
<td>OHCA, observational, A-CPR (n=284), (2003-2005) S-CPR (n=499) (2001-2003) device was used in only 210 cases</td>
<td>ROSC: A-CPR 34.5%, S-CPR 20.2% Survival to hospital admission: A-CPR 20.9%, S-CPR 11.1% Survival to hospital discharge: A-CPR 9.7%, S-CPR 2.9% No difference in CPC (p=0.360) (device was used in only 210 cases)</td>
<td>A-CPR is better. AutoPulse was improved survival to hospital discharge when compared to S-CPR</td>
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<td>4. Hallstrom A (2006). Manual chest compression vs. use of an automated chest compression device during resuscitation following out-of-hospital cardiac arrest: A randomized trial (ASPIRE) (68)</td>
<td>OHCA, randomized, multicenter (US, Canada) 2004-2005 A-CPR (n=554), S-CPR (n=517)</td>
<td>Survival to 4 h: A-CPR 29.5%, S-CPR 28.5%, p=0.74 Survival to hospital discharge: A-CPR 5.8%, S-CPR 9.9%, p=0.60 CPC 1–2 at hospital discharge: A-CPR 3.1%, S-CPR 7.5%, p&lt;0.006</td>
<td>Use of an automated LDB-CPR device as implemented in this study was associated with worse neurological outcomes and a trend toward worse survival compared with manual CPR.</td>
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<tr>
<td>5. Krep H (2007). Out-of-hospital cardiopulmonary resuscitation with the AutoPulse™ system: A prospective observational study with a new load-distributing band</td>
<td>OHCA, prospective, Germany, 2004-2005 A-CPR (n=46), ACD-CPR (n=48)</td>
<td>ROSC: 54.3% (n=25/46), chest compression device (69) Admitted to ICU: 39.1% (n=18/46), Discharged from ICU: 21.8% (n=10/46) Discharged CPC 1=2; CPC 2=1; CPC 3=7 patient (n=10) ROSC ACD-CPR with use cardio pump: 52% (n=48)</td>
<td>The AutoPulse is an effective and safe mechanical CPR device useful in OHCA</td>
</tr>
<tr>
<td>6. Duchateau F-X (2010). Effect of the AutoPulse™ automated band chest compression device on hemodynamics in out-of-hospital cardiac arrest resuscitation (70)</td>
<td>OHCA, prospective, France (2008) A-CPR (n=29) (first S-CPR and then A-CPR same groups)</td>
<td>Median diastolic BP: A-CPR 23 mmHg, S-CPR 17 mmHg, p&lt;0.001 Median systolic BP: A-CPR 106 mmHg, S-CPR 72 mmHg, p&lt;0.02 Mean BP: A-CPR 36 mmHg, S-CPR 29 mmHg, p&lt;0.002, ETCO₂; did not increase with Autopulse (from 21 to 22 mmHg, p=0.80)</td>
<td>The use of the AuToPulse is associated with increased diastolic BP compared to S-CPR.</td>
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<td>7. Jennings PA (2012). An automated CPR device compared with standard chest compressions for out-of-hospital resuscitation (25)</td>
<td>OHCA, retrospective, Australia, 2006-2010 A-CPR (n=66), S-CPR (n=220)</td>
<td>Survival to hospital : A-CPR 26% (17/66), S-CPR 20% (43/220), p=0.23 Survived to hospital discharge: A-CPR 3% (n=2/66), S-CPR 7% (15/220), p=0.38</td>
<td>Further research is warranted, which involves randomization and larger number of cases to investigate the potential benefit of A-CPR, including survival to hospital discharge.</td>
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<td>8. Hock Ong ME (2012). Improved neurologically intact survival with the use of an automated, load-distributing band chest compression device for cardiac arrest presenting to the emergency department (30)</td>
<td>OHCA, multicenter, randomized, Singapore S-CPR (n=459, 2004-2007) A-CPR (n=522, 2007-2009)</td>
<td>ROSC: A-CPR 35.3% (n=195), S-CPR 22.4% (n=103) Survival to hospital discharge: A-CPR 3.3%, S-KPR 1.3% CPC 1 -2 at hospital discharge: A-CPR 81.3% (n=13/16), S-CPR 33.3% (n=2/6).</td>
<td>The AutoPulse improved survival with intact neurological status on discharge in adults with non-traumatic cardiac arrest.</td>
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</table>

CO: cardiac output; CPP: coronary perfusion pressure; IHCA: in-hospital cardiac arrest; OHCA: out-hospital Cardiac arrest; ROSC: return of spontaneous circulation; PEA: pulseless electrical activity; CPC: cerebral performance category; ETCO₂: end-tidal CO₂; A-KPR: AutoPulse CPR; ICU: intensive care unit; BP: blood pressure
Table 3. Studies with other devices

<table>
<thead>
<tr>
<th>Title</th>
<th>Study design</th>
<th>Results</th>
<th>Conclusion</th>
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<tr>
<td>Plaisance P (2004). Evaluation of an impedance threshold device in patients receiving active compression decompression cardiopulmonary resuscitation for out of hospital cardiac arrest (72)</td>
<td>OHCA, multicenter, randomized, prospective, France, 1999-2000 ACD-KPR+ active ITD (n=200) ACD-KPR + sham ITD (n=200)</td>
<td>24-h survival: ACD-KPR+ active ITD 32%, ACD-KPR+sham ITD 22%, p = 0.02 ROSC: ACD-KPR+active ITD:48%, ACD-KPR+sham ITD:39%, p=0.05 Survival ICU admission: ACD-CPR+active ITD 40%, ACD-CPR+sham ITD 29% (p=0.02) Hospital discharge: ACD-CPR+active ITD: 5%, ACD-KPR+sham ITD:4% (p=0.02)</td>
<td>ACD-KPR+active ITD significantly improved 24-h survival rates.</td>
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<tr>
<td>Frascionea RJ (2013). Treatment of non-traumatic out-of-hospital cardiac arrest with active compression decompression cardiopulmonary resuscitation plus an impedance threshold device (47)</td>
<td>ResQTrial: OHCA, randomized, prospective, multicenter (US, 2005-2009) ResQCPR (n=1403) S-CPR (n=1335)</td>
<td>Survival with good neurologic outcomes: ResQCPR 7.9%, S-CPR 5.7%, p:0.027 1-year survival: ResQCPR 7.9, S-CPR 5.7%, p:0.026</td>
<td>ResQCPR showed significant increase in survival to hospital discharge with favorable neurological function compared with S-CPR</td>
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<tr>
<td>Cha KC (2014). Hemodynamic Effects of an Automatic Simultaneous Sternothoracic CPR Device in Patients with Cardiac Arrest (42)</td>
<td>OHCA, X-CPR (n=11) S-CPR (n=14)</td>
<td>Right atrial pressures during compression and relaxation and ETCO2 were not different between two groups. Femoral arterial pressures during relaxation and CPP were higher in X-CPR (p=0.017).</td>
<td>X-KPR demonstrated higher coronary perfusion pressure than standard CPR</td>
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<td>Yang Z (2014). Similar Hemodynamic Efficacy Between 30-mm and 50-mm Compression Depth During Mechanical Chest Compression with Weil Mini Chest Compressor (43)</td>
<td>Experimental, pigs: VF was induced, MCC compression depth: 30 mm (n=5) and 50 mm (n=5)</td>
<td>There were no differences in CPP, ETCO2, carotid blood flow between the two groups. Significantly less rib fracture was observed in the 30-mm group, p&lt;0.05.</td>
<td>Similar hemodynamic efficacy was observed between 30- and 50-mm compression depth with the Weil Mini Chest Compressor.</td>
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<tr>
<td>Chen W (2012). The effects of a newly developed miniaturized mechanical chest compressor on outcomes of cardiopulmonary resuscitation in a porcine model (44)</td>
<td>Experimental, pigs: VF was induced (n=30) MCC and (LUCAS or Thumper)</td>
<td>MCC generated significantly greater CPP, ETCO2, carotid blood flow, and intrathoracic negative pressure, with significantly lower compression depth and fewer rib fractures than both the LUCAS and Thumper devices</td>
<td>MCC may provide a new option for cardiopulmonary resuscitation.</td>
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<tr>
<td>Amtz HR (2001). Phased Chest and Abdominal Compression–Decompression Versus Conventional Cardiopulmonary Resuscitation in Out-of-Hospital Cardiac Arrest (48)</td>
<td>OHCA, Germany, Lifestick (n=24), S-CPR (n=28),</td>
<td>ROSC: S-CPR 50% (13/26), Lifestick-CPR 38% (9/24), p:0.55, ROSC at VF: S-CPR 68% (13/19), Lifestick 44% (4/9), p:0.43, ROSC at NEA/ asystole: S-CPR 0%, Lifestick-CPR 33% (5/15), p:0.23 Survival 1h: S-CPR 46% (12/26), Lifestick 25% (6/24) Hospital discharge: S-CPR 7/26, Lifestick 0 Autopsy: Sternal or rib fractures were found more frequently with S-CPR, p&lt;0.035</td>
<td>Lifestick resuscitation is feasible and safe and may be advantageous in patients with asystole or pulseless electric activity.</td>
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<td>Havel C (2008). Safety, feasibility, and hemodynamic and blood flow effects of active compression–decompression of thorax and abdomen in patients with cardiac arrest (49)</td>
<td>OHCA, Prospective, single-center, phase II study, Lifestick (n=20) Thumper (n=11)</td>
<td>Although Lifestick seemed to improve hemodynamic effects compared with the Thumper device, they were not significantly different between Lifestick and Thumper in resuscitations.</td>
<td>Lifestick is safe and beneficial. The small number of patients included in the study limits the conclusions about the hemodynamic effects of the Lifestick.</td>
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</table>

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