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8.1 Introduction

Cardiac arrest represents a dramatic clinical event that can occur suddenly and often without premonitory signs. This condition is characterized by sudden loss of consciousness caused by the lack of cerebral blood flow, which occurs when the heart ceases to pump. Indeed, it represents a leading cause of death in the Western world, with as many as 350,000–700,000 people in the United States, Canada, and Europe sustaining cardiac arrest each year [1, 2]. Cardiopulmonary resuscitation (CPR), including chest compression, often in conjunction with electrical defibrillation, has the potential of re-establishing spontaneous circulation (ROSC). Despite major efforts to improve outcomes from cardiac arrest, average survival rate remains dismal and presents a large variation with a spread between 2 and 39 % [3, 4]. Both in heavily populated larger cities and in sparsely populated rural communities, delayed response by emergency medical services compromises outcomes such that survival is more disappointing.

During cardiac arrest, coronary blood flow ceases, accounting for a progressive and severe energy imbalance. Intramyocardial hypercarbic acidosis is associated with depletion of high energy phosphates and correspondingly severe global myocardial ischemia [5]. The ischemic left ventricle becomes contracted ushering in the stone heart [6]. After onset of contracture, the probability of ROSC becomes remote. There is now evidence that the highest priority of intervention is to re-establish systemic blood flow promptly by external chest compression, and thereby achieve and maintain threshold levels of coronary and cerebral perfusion.

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Accordingly, effective, consistent, and uninterrupted chest compression is now designated as the primary intervention for management of cardiac arrest. Both survival and neurological recovery are contingent upon initiating chest compression within <5 min [7, 8]. Accordingly, bystander initiated chest compressions by minimally trained, nonprofessional rescuers subsequently supported by well-organized professional emergency medical providers have significantly increased survival from out-of-hospital cardiac arrest.

8.2 Quality of CPR

In addition to the benefits of prompt intervention, it is also the quality of chest compressions delivered in both in- and out-of-hospital settings that has proven to be a determinant of outcomes. Indeed, blood flows generated by chest compression are dependent on the pressure gradient between the aortic and the venous pressures. Coronary perfusion pressure (CPP), defined as the difference between simultaneously measured minimal aortic pressure and right atrial pressure during compression diastole, is highly correlated with coronary blood flow during cardiac resuscitation and is currently recognized as the best single indicator of the likelihood of ROSC [9]. Based on both experimental and clinical observations, ROSC can be predicted when CPP is maintained above 15 mmHg during compressions [9, 10]. Resuscitative strategies that increase CPP, including high quality chest compressions as well as the use of vasopressor, have been therefore supported and considered more effective in restore circulation. In fact, although chest compression produces less than 50 % of prearrest stroke volumes, threshold levels of myocardial and cerebral blood flows are restored such as to minimize ischemic myocardial and cerebral ischemic injury [11].

The evidence is secure that the quality of chest compression is a major determinant of successful resuscitation. A good quality CPR should be performed with: adequate chest compression depth and rate; duty cycle; minimal interruptions; and complete chest recoil [12]. Yet, there is persuasive evidence that conventional manual chest compressions are often performed ineffectively. Indeed, whereas 23 % of victims were resuscitated after what Wik et al. defined as “good CPR,” only 1 % were resuscitated with “not good CPR” [13]. In both in-hospital and out-of-hospital settings, the quality of CPR, and specifically chest compressions, was also the major determinant of the ROSC. Based on 176 victims of out-of-hospital cardiac arrest, only 28 % of rescuers performed competent chest compressions in which the anterior–posterior diameter was decreased by approximately 5 cm so as to conform to the international guidelines [14]. An inadequate depth of chest compressions was also reported in 67 instances of in-hospital cardiac arrest [15]. Human observational studies also showed that interruptions of chest compressions are common, averaging 24–57 % of the total arrest time [13].

However, even well-trained professional providers cannot maintain effective chest compression for intervals that exceed 2 min [16, 17]. This limitation is in addition to the documented inconsistency of depth and rate of compressions [14–18]. The challenges are even greater during evacuation and transport of victims. Therefore, the option of using mechanical devices is attractive. Mechanical chest compression potentially overcomes operator fatigue, slow rates of compression, and inadequate depth of compression. A mechanical compressor would also allow for the delivery of an electrical shock without interruption of manual compression for the protection of the rescuer.

8.3 Mechanical Compression

Several new devices have recently been introduced to facilitate mechanical chest compression [19]. Both the AutoPulse (ZOLL Medical Corporation, USA) and the Lund University Cardiac Arrest System (LUCAS) (LUCAS, PhysioControl, Sweden) have demonstrated equivalency and potentially even greater effectiveness than manual chest compression.

8.4 AutoPulse

The AutoPulse is a battery powered load-distributing band, mechanical CPR device. Its functioning is based on the concept that distributing force over the entire chest through a band improves the effectiveness of chest compressions by delivering more total energy to the torso. The device adjusts automatically to the size and shape of each patient and is constructed around a backboard that contains a motorized rotating shaft. It utilizes a load-distributing band, which is connected to the rotating shaft to compress the chest. The band is tightened or relaxed around the chest rhythmically to provide a “squeezing” effect during the compression phase [20]. AutoPulse-CPR features are reported in Table 8.1.

Earlier animal studies of pigs and clinical studies in the setting of in-hospital cardiac arrest have reported better hemodynamics during mechanical compression with AutoPulse in comparison to standard manual CPR [21, 22]. Subsequently, nonrandomized human series have reported increased rates of sustained ROSC

Table 8.1 AutoPulse-CPR features

Compression depth: 20 % of chest anterior–posterior diameter

Rate of compression: 80/min

Duty cycle: equal compression/decompression time

Compression/ventilation ratio: 30/2 or continuous compressions

Full chest recoil allowed

[23] and increased survival to hospital discharge following out-of-hospital cardiac arrest with AutoPulse in comparison to standard CPR (9.7 versus 2.9 %) [24]. Based on these results, the ASPIRE trial (AutoPulse Assisted Prehospital International Resuscitation) was initiated [25]. It was a multicenter, randomized trial of patients experiencing out-of-hospital cardiac arrest in the United States and Canada. The trial compared mechanical cardiopulmonary resuscitation with AutoPulse device (AutoPulse-CPR) to traditional manual CPR (manual-CPR). The primary end point was survival to 4 h after the 911 call, while secondary end points were survival to hospital discharge and neurological outcome. Enrollment was suspended early due to safety concerns, after approximately 1,000 patients. No difference existed in the primary end point of survival to 4 h. However, survival to hospital discharge was 9.9 % in the manual-CPR group and 5.8 % in the AutoPulse-CPR. More importantly, a significantly worse neurologic outcome was observed when AutoPulse-CPR was compared with manual-CPR (3.1 versus 7.5 %, $p = 0.006$). However, a subsequently posthoc analysis of this study revealed significant heterogeneity among study sites [26]. Indeed, one site (site C) made a potentially important protocol change midtrial, and enrollment at that site was noted to be independently associated with outcome. The protocol change at site C also appeared to have resulted in a delay in application of AutoPulse-CPR. Before and after the protocol change survival in patients receiving AutoPulse-CPR decreased from 19.6 to 4 %. At the time the trial was suspended, the outcomes of patients at the other sites appeared to have been trending in favor of the intervention.

A more recent prospective cohort evaluation compared resuscitation outcomes before and after switching from manual CPR to AutoPulse-CPR in a multicenter emergency department trial, enrolling 1,011 patients (459 in the manual CPR and 552 patients in the AutoPulse-CPR) [27]. The rate of survival to hospital discharge tended to be higher in the AutoPulse-CPR phase (3.3 %) versus the manual one (1.3 %). There were also more survivors in the AutoPulse group with cerebral performance category 1 compared to the manual group ($p = 0.01$).

Finally, another multicenter randomized clinical trial, the “Circulation Improving Resuscitation Care (CIRC)” trial was conducted by Dr. Wik [28] and compared AutoPulse versus “high quality” manual CPR in over 4,000 out-of-hospital cardiac arrest patients in the USA and Europe. The trial had unique features: [1] training of all EMS providers in a standardized deployment strategy and continuous monitoring for protocol compliance; [2] a pre-trial simulation study of provider compliance with the trial protocol; [3] three distinct study phases (in-field training, run-in, and statistical inclusion) to minimize the Hawthorne effect and other biases; [4] monitoring of the CPR process using either transthoracic impedance or accelerometer data; [5] randomization at the subject level after the decision to resuscitate is made to reduce selection bias; [6] use of specific statistical tests with sufficient power to determine superiority, inferiority, or equivalence. Although the full article has not been published yet, preliminary results have shown equivalency in ROSC, 24 h survival, survival to hospital discharge, and hands-off fraction between the two CPR approaches.

8.5 LUCAS

The LUCAS chest compression system is a portable piston device composed of: a back plate, which is positioned underneath the patient as a support for the external chest compressions; an upper part mounted on two arms, which contains the battery and the compression mechanism with a disposable suction cup; a stabilization strap which helps to secure the position of the device in relation to the patient. Two versions of the device have been produced. The original LUCAS 1, is a pneumatically driven device that requires no electrical supply, but is powered by compressed air from a portable compressed air cylinder or wall outlet. The LUCAS 2, instead is a battery powered device. LUCAS CPR features are reported in Table 8.2.

Currently, there are no published multicenter randomized human studies comparing LUCAS CPR with standard CPR. Nevertheless, animal studies and clinical observations have reported better hemodynamics during LUCAS CPR in comparison to manual CPR. A single study of pigs with VF showed that LUCAS CPR improved hemodynamic and short-term survival rates compared with standard CPR [29]. Indeed, after 5 min of untreated VF, animals were subjected to 20-min CPR with either LUCAS or manual CPR. Significantly higher CPP was observed during LUCAS CPR. All the pigs in the mechanical group achieved ROSC compared with only 37 % in the manual group. Another animal study randomized pigs to a 15-min CPR with either LUCAS or manual compression [30]. During CPR, the cortical cerebral blood flow was significantly higher in the group treated with LUCAS ($p = 0.041$). End-tidal CO₂, an indirect measurement of the achieved cardiac output during CPR, was also significantly higher in the group treated with the LUCAS device ($p = 0.009$). Greater EtCO₂ was also consistently measured over a 15-min interval of LUCAS CPR in comparison with manual CPR in a prospective clinical study including 126 out-of-hospital cardiac arrest patients [31].

Six case series involving overall approximately 200 patients have reported variable success in use of the LUCAS device when implemented after an unsuccessful period of manual CPR [32–37]. One study, in particular, was a good quality case series including 100 patients [33]. Of the 43 witnessed cases treated with LUCAS within 15 min from ambulance call: 24 had VF and 15 (63 %) of these cases achieved ROSC and 6 (25 %) of them survived with a good neurological

Table 8.2 Lucas CPR features

Compression depth: 5 cm
Rate of compression: 100/min
Duty cycle: equal compression/decompression time
Compression/ventilation ratio: 30/2 or continuous compressions
Full chest recoil allowed

recovery after 30 days; 19 patients, instead, were found in asystole at rescuers' arrival and 5 (26 %) of them achieved ROSC and 1 (5 %) survived for over 30 days. Another study using concurrent controls in witnessed out-of-hospital cardiac arrest was unable to show benefit in ROSC, survival to hospital, and survival to hospital discharge with the use of the LUCAS device over the use of manual CPR [38].

More recently, LUCAS versus manual CPR have been compared in a randomized prospective pilot study, enrolling 149 patients with out-of-hospital cardiac arrest in two Swedish cities [39]. This pilot study reported no difference in ROSC, 4 h survival, and survival to hospital discharge between the two CPR approaches. Nevertheless, this was only a pilot investigation and data were used for power calculation in one of the two currently underway randomized multicenter trials [40, 41].

Indeed, the role of LUCAS CPR will be clarified after completion and publication of the results of the following two ongoing studies: (1) The prehospital randomized assessment of a mechanical compression device in cardiac arrest (PARAMEDIC) trial, that enrolls 4,000 patients in England, Wales, and Scotland, in order to assess effects of LUCAS CPR over manual CPR on 30-day survival [40]; and (2) The LUCAS in Cardiac arrest study (LINC): a study comparing conventional adult out-of-hospital cardiopulmonary resuscitation with a concept with mechanical chest compressions and simultaneous defibrillation that enrolls 2,500 patients with the intent to compare the effects of LUCAS CPR over manual CPR on 4-h survival [41].

8.6 Conclusions

At the moment, there are insufficient data to support or refute the use of a mechanical compressor instead of manual CPR. Currently undergoing multicenter randomized clinical trials comparing mechanical versus manual CPR will clarify the role of mechanical chest compression. There is some low-quality evidence that mechanical CPR can improve consistency and reduce interruptions in chest compressions. Nevertheless, it has to be recognized that mechanical devices for CPR warrant: continuous high quality CPR; compression during transport; no interruptions in CPR; no rescuer fatigue; and more importantly hands free for other procedures. It may be reasonable therefore to consider mechanical CPR to maintain continuous chest compression while undergoing CT scan or similar diagnostic studies, or interventional procedures treatments, i.e., primary angioplasty, when provision of manual CPR would be difficult. Finally, in order to ensure the best result from the use of a mechanical CPR it is important to consider the implementation of cardiac arrest teams specially trained in applying and starting mechanical compressor, i.e., with a "pit-crew" protocol, so as to reduce the interruption in CPR [20]. Interruptions in chest compressions to apply a mechanical device, in fact, can be as low as 20 s, but are often much longer, i.e., almost 2 min [42].

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