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## **Mechanical chest compression devices – current and future roles**

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## ABSTRACT

**Purpose of review:** It is recognised that the quality of CPR is an important predictor of outcome from cardiac arrest yet studies consistently demonstrate that the quality of CPR performed in real life is frequently sub-optimal. Mechanical chest compression devices provide an alternative to manual CPR. This review will consider the evidence and current indications for the use of these devices.

**Recent findings:** Physiological and animal data suggest that mechanical chest compression devices are more effective than manual CPR. However there is no high quality evidence showing improved outcomes in humans. There are specific circumstances where it may not be possible to perform manual CPR effectively e.g. during ambulance transport to hospital, en-route to and during cardiac catheterisation, prior to organ donation and during diagnostic imaging where using these devices may be advantageous.

**Summary:** There is insufficient evidence to recommend the routine use of mechanical chest compression devices. There may be specific circumstances when CPR is difficult or impossible where mechanical devices may play an important role in maintaining circulation. There is an urgent need for definitive clinical and cost effectiveness trials to confirm or refute the place of mechanical chest compression devices during resuscitation.

It is estimated that approximately 600,000 sustain a cardiac arrest and receive cardiopulmonary resuscitation (CPR) in the US and Europe each year[1-2]. CPR has unequivocally been associated with improved outcomes in these patients[3]. It is becoming increasingly recognised that survival is affected not only by whether CPR is performed, but also by its quality. Recent evidence has confirmed the importance of chest compression depth[4-5], rate[6], releasing pressure between compressions[7], minimising interruptions in CPR[8], avoiding pauses prior to defibrillation[9] and not hyperventilating the patient[10] to ensure the optimal effectiveness of resuscitation. Despite the knowledge that the quality of CPR is of critical importance to outcomes, studies consistently demonstrate that the quality of CPR performed in real life by both laypersons[11] and healthcare providers[12-14] is frequently sub-optimal.

Mechanical chest compression devices which deliver automated chest compression have been available commercially for well over 20 years. These devices are attractive as they can compress the chest to specified depths/rates, compressions are uniform and efficacy does not decline due to fatigue. Despite their appeal, mechanical compression devices have yet to be universally adopted by the resuscitation community[15]. This article will review the recent evidence behind two of the more widely used mechanical chest compression devices – the LUCAS device (Jolife, Lund, Sweden) and Autopulse (Zoll Circulation, Chelmsford, MA, USA).

## LUCAS

LUCAS is a mechanical chest compression device that provides both compression and active decompression. It consists of a silicon rubber suction cup that is applied to the chest and a pneumatic cylinder mounted on two legs which are connected to a stiff back plate (figure 1). The original LUCAS device was gas driven (oxygen / air) but has been superseded by a battery driven device (LUCAS-2). This development has overcome the logistical requirement to carry compressed

gas to power the device and initial concerns about the development of high oxygen concentrations in confined spaces[16].

The device compresses the chest between 4 and 5cm at a rate of 100 compressions per minute with an equal amount of time being spent in compression and decompression. Randomised studies comparing LUCAS to manual CPR in a pig ventricular fibrillation model have shown that the device produces higher cardiac output(LUCAS vs manual 0.9(0.1) vs 0.5(0.1) l min<sup>-1</sup>, P<0.05), carotid artery blood flow (58(4) vs 32(5) ml min<sup>-1</sup>, P<0.05) end-tidal CO<sub>2</sub> (2.8(0.1) vs 2.0 (0.2) kPa, P<0.05) and coronary perfusion pressures (17(1) vs 10(2) mm Hg, P<0.05). Survival rates were also higher in the LUCAS-CPR compared to manual CPR arm (83% ROSC versus 0%)[17].

Several case series demonstrate the feasibility of using the device in the out of hospital[18] and in-hospital[19-20] settings. Axelsson *et al* reported their experience with introducing LUCAS into two emergency medical service (EMS) systems in Sweden[21]. The LUCAS device was alternated between one of four advanced life support units. Patients were included in the study if they sustained a witnessed out of hospital cardiac arrest of presumed cardiac aetiology. Patients were assigned to control or LUCAS arms depending upon whether the attending ALS unit had a LUCAS device or not. The groups were reasonably well matched although there were some differences in the aetiology of arrest between the two groups. Although there was evidence of higher end-tidal CO<sub>2</sub> in the LUCAS arm[22] there was no difference in outcomes between groups (survival to hospital discharge rate 5% for LUCAS 5.9% for control)[21]. The study showed that the average time to apply the LUCAS device was 12 minutes after arrival of EMS (18 minutes after the patient collapsed). It is unknown if outcomes would have been different had the device been deployed earlier.

There have not been any large, prospective randomised controlled trials reported with the LUCAS device to date. There are currently two large trials in progress. The LINC trial (NCT00609778) is a

randomised comparison of a LUCAS optimised resuscitation algorithm (which includes the use of blind shocks without ECG analysis) compared to current European Resuscitation Council guidelines. The primary outcome is four-hour survival and secondary outcomes include survival and neurological status (CPC score) at hospital discharge, 1 and 6 months. A UK based study, the PARAMEDIC trial (Prehospital Randomised Assessment of a Mechanical Compression Device In Cardiac Arrest (ISRCTN: 08233942) is a multi-centre, cluster randomised controlled clinical and cost effectiveness study. In this study the LUCAS device is being used simply as a comparator to manual chest compressions. The primary outcome of this study is survival 30 days after hospital admission. Secondary outcomes include other measures of clinical effectiveness (ROSC rate, survival at 12 months, neurological outcomes and cost effectiveness). These studies should provide definitive evidence for the use of the LUCAS device in out of hospital cardiac arrest.

## Autopulse

The AutoPulse device comprises a backboard and load distributing band (LDB) that is placed around the chest. The device contains a motor which tightens and loosens the band around the chest after which the chest is allowed to passively decompress. The device adjusts the LDB to the size of the subject being resuscitated and distributes the compressive load over the anterior chest (figure 2). Porcine and human studies comparing AutoPulse to standard CPR showed improved myocardial flow[23], blood pressure[23-25], coronary perfusion pressure (CPP)[23-24] and cerebral blood flow[24].

Two landmark studies reporting the results of Autopulse in the out of hospital setting were published in the same edition of JAMA in 2006. The first study was a before and after cohort study examining the effects of introducing Autopulse into a single EMS system[26]. The study found a significant improvement in return of spontaneous circulation (35% Autopulse versus 20% manual

CPR and survival to hospital discharge (10% versus 3%). There was no difference in neurological status at discharge between the groups.

The second study was a cluster randomised trial comparing Autopulse to standard CPR in 5 EMS systems in the US and Canada (the ASPIRE trial)[27]. The study enrolled 1071 patients with out of hospital cardiac arrest. It was terminated prematurely at the recommendation of the Data Monitoring and Safety Committee due to a lack of benefit in the primary outcome (survival to 4 hours) and apparent harm in secondary, longer-term, patient-centred outcomes.

There are several possible explanations for the disparity in the outcomes of these studies. The design of the studies was different. The Ong study was a before / after study which may be biased by changes in practice through the passage of time. Although the ASPIRE study should be protected from this bias, there remains the potential that the EMS providers would behave differently whilst under the focus of a trial for example it has been suggested the quality of CPR in the control arm may have improved or that a culture of belief that the device was doing good led to enrolment of patients that might otherwise not have normally had resuscitation commenced. A post hoc analysis of the ASPIRE trial reports that one of the five sites (site C) outcomes were significantly worse for the Autopulse group than in the other four sites where outcomes were trending in favour of the intervention[28].

Care should be taken when planning trials to ensure that the introduction of the new technology is uniform and does not reduce existing performance. In the two clinical Autopulse studies above deployment of the device took longer in the ASPIRE study than in the Ong study (12 versus 4.7 minutes). In the build up to the CIRC trial (a new multicentre RCT of Autopulse <http://circtrtrial.com/>) Tomte piloted the effect of introducing autopulse to different ambulance services. The study found that in the best performing site, the introduction of Autopulse increased the no flow fraction, whilst at the poorer performing sites, the no flow fraction improved[29]. The finding that the application of mechanical CPR devices may impair CPR performance has also been observed in clinical studies

emphasising the importance of adequate training not only on use of the device but also in the context of the overall CPR attempt[30].

Overall, there remains uncertainty about the health and economic impact of the routine use of Autopulse for cardiac arrest. The results of the CIRC trial should help to answer some of these outstanding questions.

### **Injuries associated with mechanical compression devices**

Chest compressions whether undertaken manually or mechanically are associated with a risk of skeletal, soft tissue and internal injuries[31]. Reports on conventional CPR in adults suggest an incidence of rib fractures ranging from 13 to 97%, and of sternal fractures from 1 to 43%[32-33].

Soft tissue injuries such as skin abrasions / erythema are also seen frequently. More severe injuries such as splenic, hepatic or gastric rupture; aorta rupture, air embolism fortunately occur less frequently but are nevertheless present in 0-2% of cases[31].

A growing number of case reports of skeletal, soft tissue and organ damage have been appearing in the literature in association with mechanical chest compression devices[34-36]. This is perhaps not surprising as the injuries that can be sustained during CPR can be dramatic. In the context of new pieces of equipment, the extremes of death or survival are likely to create an element of publication bias. As the injury patterns described to date with mechanical devices have also been seen in association with manual CPR it is difficult to clearly separate expected from novel injuries.

Smeakal *et al* compared autopsy findings in 85 patients unsuccessfully resuscitated by either manual or mechanical (LUCAS) CPR. Injuries were seen in 22/38(58%) in the LUCAS group compared to 21/47 (45%) in the manual group ( $p = 0.28$ ). Fracture of the sternum was present in 21% in the manual group and 29% in the LUCAS group ( $p = 0.46$ ) and  $\geq 3$  rib fractures) 44% versus 27.7% ( $p = 0.12$ ). Internal bleeding was found in 36% of the LUCAS group compared to 17% in the manual group. There was one ruptured abdominal aortic aneurysm in the LUCAS group and one thoracic

aortic dissection in each group, all of which were considered by the pathologist to be the primary cause of cardiac arrest and not injuries from treatment. Although the study concludes that injury patterns were similar, the study lacked sufficient power for this endpoint and it was not designed as an equivalence study.

It is important to note that virtually all of the studies reporting injury patterns in association with CPR have been derived from autopsy studies of non-survivors of cardiac arrest. In this scenario, the number of injuries found among patients who die is of little importance to the effectiveness of mechanical CPR, unless they translate into a reduction in mortality. Injuries in survivors and the consequences thereof are much more important. Survival and disability are the relevant endpoints that should be addressed in properly designed clinical and cost effectiveness trials rather than reliance on further post mortem studies which do not take into account the potential clinical effectiveness of mechanical chest compression devices.

### **Use in special circumstances**

As has been discussed above, the routine, everyday use of mechanical chest compression devices as an alternative to CPR cannot currently be recommended. However there are situations where manual CPR may difficult or impossible to perform effectively where the use of mechanical compression devices may be the only option. The use of mechanical chest compression devices for such interventions as percutaneous coronary intervention; diagnostic imaging, organ transplantation; ambulance transport may prove invaluable.

#### *Percutaneous coronary intervention*

In adults, the commonest cause of cardiopulmonary arrest is cardiac in origin. Acute coronary occlusion causing myocardial ischaemia is a common substrate for the development of ventricular

fibrillation. There is good evidence to demonstrate the efficacy of percutaneous coronary intervention (PCI) in patients with an acute ST elevation myocardial infarction (STEMI)[37]. There is also evidence of benefit if PCI is performed after return of spontaneous circulation[38-39]. There are a growing number of case reports of patients surviving after being taken to the catheter lab in cardiac arrest exist, however it is currently unknown if PCI could be routinely extended to include appropriately selected patients in cardiac arrest[40].

In order to perform PCI during cardiac arrest, CPR must be continued. However undertaking manual CPR in parallel with PCI is difficult. The overhead gantry around the patient's chest often gets in the way and the height of the catheterisation table makes CPR more difficult. CPR is frequently interrupted during imaging. Staff are also exposed to potentially high doses of radiation ( $100 \text{ Gy cm}^{-2}$ ) [19]. Mechanical chest compression devices can provide chest compressions during PCI. The overhead gantry can be angled to allow the device to be put in place and images acquired. The base plates of Autopulse and LUCAS are partially radiolucent and so allow diagnostic imaging to continue during CPR.

A growing number of case reports / series demonstrate the feasibility of conducting PCI during mechanical chest compression[20, 41-43]. Mechanical CPR with LUCAS can maintain adequate coronary perfusion pressures ( $>15 \text{ mm Hg}$ ) and TIMI grade 3 flow[44]. The largest series of cases to date where mechanical CPR has been used in the cardiac catheter lab comprises 43 patients at Lund University Hospital in Sweden. The indication for cardiac catheterisation in these patients were STEMI (n=33), non STEMI (n=7); elective PCI (n=2) and tamponade (n=1). Initial imaging demonstrated 5 patients had sustained myocardial rupture. All these patients died. Of the remaining 38; 1 had a cardiac tamponade drained successfully and 36 underwent PCI for coronary occlusion (no culprit lesion was found during diagnostic angiography in the remaining patient). Twentyseven (75%) of the PCIs were considered 'technically successful' procedures defined as a residual stenosis  $<50\%$  at the site of the target lesion and achieving TIMI 2 (slow velocity flow to the distal part of the artery) or

TIMI 3 (normal blood flow) with ongoing mechanical chest compressions. The average duration of LUCAS use was approximately 30 minutes (range 1-99 minutes). Sixteen patients achieved return of spontaneous circulation in the catheter lab. One patient was transferred from the catheter lab with on-going LUCAS-CPR to the operating theatre. Twelve of these patients were finally discharged from hospital (27%). Most (11/12) had cerebral performance categories of 1 (neurologically intact).

Without these interventions these patient would almost certainly have died.

This study is important as it clearly demonstrates the technical possibilities and outcomes from using mechanical chest compression devices for facilitating PCI during cardiac arrest when manual chest compression is difficult or impossible to perform. What is now needed are prospective clinical studies on patients who do not achieve ROSC with traditional ALS in out-of-hospital cardiac arrest to investigate if this approach is feasible and may save even more lives[40].

#### *Mechanical CPR during diagnostic imaging*

Resuscitation algorithms recommend that potentially reversible causes of cardiac arrest are sought during resuscitation[45]. However it can be difficult to establish the cause of cardiac arrest in many patients based on history and clinical examination findings alone. Ultrasound (including echocardiography) has been used with some success to identify reversible causes of cardiac arrest[46-47]. The ability to undertake computed tomography (CT) scanning has been limited by the inability to perform manual chest compression during image acquisition due to the physical constraints placed by the CT scan tunnel. Wirth *et al* [48] recently completed a pilot study of CT scanning during mechanical chest compression. In this study the feasibility of image acquisition during mechanical CPR was first assessed on a chest / heart phantom model. Mechanical CPR had to be stopped during image acquisition due to the presence of movement artefact. In addition, the battery plate had to be removed from the Autopulse device during the acquisition of CT brain images. Space limitations meant that the compression piston had to be disconnected from the back plate when the LUCAS device was used. For both devices this means that periods of “no-flow” time

are inevitable during the acquisition of CT images. The study goes on to report their experience of using this experimental protocol in 3 patients in sustained cardiac arrest (2 with Autopulse, 1 LUCAS). In each case new diagnostic information was obtained from the CT images (2 x pulmonary embolus; 1 x massive intracerebral haemorrhage). Interruptions in CPR were not measured, but the authors estimate that they were no greater than 20 seconds during image acquisition. With advances in technology, they hypothesise that it should be possible by integrating data from the Autopulse device with the CT scan software to allow uninterrupted image acquisition during the relaxation phase of chest compression. This is an important study in that it demonstrates the feasibility of CT imaging during mechanical chest compression. However further studies confirming the safety, efficacy and cost effectiveness of using CT scanning during CPR are required before this approach can be routinely recommended.

### *Transplantation*

Non-heart beating organ donation programmes have been developed in an attempt to increase the donor pool for organ transplantation[49]. Following confirmation of cardiac death (in accordance with local guidelines/laws) patients may be eligible for non-heart beating organ donation. Non-heart beating organ donation can be classified as controlled or uncontrolled according to the circumstances surrounding their death[50]. Controlled donors are those that sustain a cardiac arrest after planned withdrawal of treatment due to non-survivable injuries / illnesses. Uncontrolled donors are patients that are either brought in dead to the emergency department; or who either arrest in hospital or are brought to the emergency department with on-going CPR but despite resuscitation efforts a spontaneous circulation cannot be restored.

The duration of warm ischaemia time from cessation of cardiac output until organ preservation is an important predictor of subsequent graft function. In controlled organ donation, withdrawal of treatment is planned and there is time prior to cardiac arrest to obtain informed consent, prepare the donor for organ retrieval for example by moving to the operating procedure or pre-cannulation

to allow organ preservation can be commenced immediately after cardiac arrest. In these situations it is practical to limit warm ischaemia times to less than 30 minutes[51]. By contrast, in the situation of uncontrolled donors, as cardiac arrest has already occurred there is a much greater pressure on time to undertake the necessary ethical, legal and practical procedures required to retrieve organs. Mechanical chest compression devices have proved to be a valuable tool for maintain effective circulation after confirmation of cardiac death while the necessary steps are undertaken[51-53].

### *Transportation*

Transportation from the scene of a cardiac arrest to hospital with on-going CPR has several risks. Continuing CPR in the back of a moving ambulance is challenging. In order to perform compressions at least one member of the ambulance crew must remain standing and un-restrained next to the victim. A systematic examination of ambulance crash data identified that travelling with emergency lights and sirens increases the risk of an accident and standing, un-restrained in the back of the ambulance vehicle as important risk factors for injury and death[54]. Ambulance crews may also sustain back strain injuries as a consequence of prolonged chest compression in cramped circumstances[55-56]. Movement of the CPR provider and patient in the back of the vehicle may also make it difficult to perform effective CPR.

Studies examining the quality of CPR during ambulance transfer have produced mixed results. One study found the quality of CPR was poor both at the scene of the arrest and during ambulance transfer. No flow fraction (the proportion of the resuscitation attempt when no chest compressions are performed) was close to 0.5 before and during transport and compressions were of inadequate depth over 50% of the time. Ventilation rates were slightly higher in the transport group (median 15 (IQR 12-18) versus 10 (IQR 9-10,  $P < 0.05$ ). In a similar study by Olasveengen *et al* the quality of CPR at the scene of the arrest was better than in the former study (no flow fraction 0.19) but deteriorated during ambulance transfer (no flow fraction 0.27,  $P=0.002$ )[57].

Mechanical chest compression devices allow uninterrupted chest compressions to be undertaken during transfer. They would reduce the need for ambulance staff to travel standing up and unrestrained during the transfer. Crash tests involving the LUCAS device demonstrate that the device remained attached at impact speeds of 30 km/h[18]. Mechanical chest compression is also able to overcome the reduced quality of CPR that may be seen during ambulance transfer[57].

## Conclusions

Mechanical chest compression devices address many of the current limitations of manual CPR. However at this time there is insufficient evidence to recommend their routine use in out of hospital and in hospital cardiac arrest. There may be specific circumstances where CPR is difficult or impossible when mechanical devices can usefully maintain circulation. Examples include during ambulance transport to hospital, en-route to and during cardiac catheterisation, during diagnostic imaging and prior to organ donation. There is an urgent need for definitive clinical and cost effectiveness trials to confirm or refute the routine use of mechanical chest compression devices during resuscitation.

*Figure legends*

Figure 1: The LUCAS device (reproduced from Steen S Resuscitation 2002 with permission)

Figure 2: The Autopulse device (reproduced from Morozumi J et al Resuscitation 2009 with permission)

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(survival to hospital discharge 9.9% in the manual CPR group and 5.8% in the LDB-CPR group (P = .06). Neurologically intact survival 7.5% in the manual CPR group and in 3.1% of the LDB-CPR group (P = .006).

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