Clinical paper

Cardiac arrest in the catheterisation laboratory: A 5-year experience of using mechanical chest compressions to facilitate PCI during prolonged resuscitation efforts

Henrik Wagner*, Christian J. Terkelsen, Hans Friberg, Jan Harnek, Karl Kern, Jens Flensted Lassen, Goran K. Olivecrona

Abstract

Purpose: Lengthy resuscitations in the catheterisation laboratory carry extremely high rates of mortality because it is essentially impossible to perform effective chest compressions during percutaneous coronary intervention (PCI). The purpose of this study was to evaluate the use of a mechanical chest compression device, LUCAS™, in the catheterisation laboratory, in patients who suffered circulatory arrest requiring prolonged resuscitation.

Materials and methods: The study population was comprised of patients who arrived alive to the catheterisation laboratory and then required mechanical chest compression at some time during the angiogram, PCI or pericardiocentesis between 2004 and 2008 at the Lund University Hospital. This is a retrospective registry analysis.

Results: During the study period, a total of 3058 patients were treated with PCI for ST-elevation myocardial infarction (STEMI) of whom 118 were in cardiogenic shock and 81 required defibrillations. LUCAS™ was used in 43 patients (33 STEMI, 7 non-ST-elevation myocardial infarction (NSTEMI), 2 elective PCIs and 1 patient with tamponade). Five patients had tamponade due to myocardial rupture prior to PCI that was revealed at the start of the PCI, and all five died. Of the remaining 38 patients, 1 patient underwent a successful pericardiocentesis and 36 were treated with PCI. Eleven of these patients were discharged alive in good neurological condition.

Conclusion: The use of mechanical chest compressions in the catheterisation laboratory allows for continued PCI or pericardiocentesis despite ongoing cardiac or circulatory arrest with artificially sustained circulation. It is unlikely that few, if any, of the patients would have survived without the use of mechanical chest compressions in the catheterisation laboratory.

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Cardiac arrest in the catheterisation laboratory during percutaneous coronary interventions (PCIs), although a rare event, is often treated rapidly and successfully with cardiopulmonary resuscitation (CPR) and DC conversions.1 In a minority of these patients, the initial resuscitation efforts are not immediately successful, and extended periods of manual chest compressions are needed to maintain coronary and cerebral circulation. In patients who require prolonged CPR, mortality rates are very high.2 Frequently, a continued uninterrupted PCI procedure of, for example, a suddenly occluded left main artery is often the best option for attaining return of spontaneous circulation (ROSC). However, continued cardiac compressions with simultaneous PCI, which requires fluoroscopy, is exceedingly difficult to perform effectively.

A novel mechanical chest compression device, LUCASTM (Jolife AB, Lund, Sweden), (Fig. 1), has been shown to sustain both coronary and cerebral circulation despite ongoing cardiac arrest.3,4 The device is mostly radio translucent, enabling continued PCI with maintained circulation despite ongoing cardiac arrest, which has been documented in several reports.5–9 Although LUCASTM has been shown to be successfully used in the catheterisation laboratory to maintain circulation,5–9 a recent report found abysmal survival rates in patients who were brought to the catheterisation laboratory...
in cardiac arrest but with circulation maintained with mechanical chest compressions. Our experience since 2004 with the chest compression device LUCASTM is similar. Essentially, all patients admitted to our catheterisation laboratory already in cardiac arrest, but with circulation maintained through mechanical chest compressions, do not survive. However, patients who arrive at the catheterisation laboratory with intact circulation and who then suffer cardiac arrest in the catheterisation laboratory, often during the procedure of angiography or PCI, may have a benefit of LUCASTM, especially if there is concomitant PCI performed.

1. Methods

The study is a retrospective registry analysis of all patients who suffered a prolonged resuscitation episode while scheduled for any procedure in the coronary catheterisation laboratory at the Lund University Hospital during 2004–2008, to which the hospital’s cardiac arrest team was alerted. The database of the cardiac arrest team was used to find the patients in the study.

Prolonged resuscitation was defined as an episode of cardiac arrest necessitating a period of several minutes of manual chest compressions, followed by the use of a mechanical chest compressions and then tracheal intubation. The other selection criterion was that the patients had to arrive alive to the catheterisation laboratory before the episode of cardiac arrest. We also evaluated similar patients (requiring >1 min of manual chest compressions and requiring tracheal intubation) in which no mechanical chest compression device was used in the same time span.

During the period 1 January 2004 to 31 December 2007, the mechanical chest compression device used was LUCAS V1 (European version). During the period 1 January 2008 to 31 December 2008, the mechanical chest compression device used was LUCAS V2 (US version), which has the same operating parameters as LUCAS V1 except for the decompression force which is set to a maximum of 13 N.

All charts and autopsy reports of our cohort of 43 patients were examined. The predefined endpoints were mortality status on departure from the catheterisation laboratory, successful PCI and discharge from hospital in the Cerebral Performance Categories (CPCs) 1 or 2, representing a good neurological outcome.

The use of LUCASTM was stopped at the discretion of the attending physician either because the patient achieved ROSC or because further treatment with LUCASTM was deemed futile.

### Table 1

Patient characteristics and outcomes.

<table>
<thead>
<tr>
<th>Patient history (n=43)</th>
<th>In hospital death</th>
<th>Discharged alive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>24 (56%)</td>
<td>16</td>
</tr>
<tr>
<td>Diabetes</td>
<td>11 (25.5%)</td>
<td>10</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>15 (35%)</td>
<td>10</td>
</tr>
<tr>
<td>Smoking/X-smoke</td>
<td>22 (51%)</td>
<td>16</td>
</tr>
<tr>
<td>Previous MI</td>
<td>12 (28%)</td>
<td>8</td>
</tr>
<tr>
<td>Previous PCI</td>
<td>5 (11.3%)</td>
<td>5</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>6 (14%)</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indication for cath lab procedure (n=43)</th>
<th>In hospital death</th>
<th>Discharged alive</th>
</tr>
</thead>
<tbody>
<tr>
<td>STEMI</td>
<td>33 (77%)</td>
<td>27</td>
</tr>
<tr>
<td>NSTEMI</td>
<td>7 (16.1%)</td>
<td>3</td>
</tr>
<tr>
<td>Elective PCI</td>
<td>2 (4.6%)</td>
<td>0</td>
</tr>
<tr>
<td>Tamponade</td>
<td>1 (2.3%)</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Culprit lesion in coronary patients (n=42)</th>
<th>In hospital death</th>
<th>Discharged alive</th>
</tr>
</thead>
<tbody>
<tr>
<td>LM</td>
<td>9 (21%)</td>
<td>6</td>
</tr>
<tr>
<td>LAD</td>
<td>25 (60%)</td>
<td>20</td>
</tr>
<tr>
<td>LCx</td>
<td>2 (4.7%)</td>
<td>0</td>
</tr>
<tr>
<td>RCA</td>
<td>6 (14.3%)</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Initial rhythm at cardiac arrest (n=43)</th>
<th>In hospital death</th>
<th>Discharged alive</th>
</tr>
</thead>
<tbody>
<tr>
<td>VF/VT</td>
<td>6 (14%)</td>
<td>2</td>
</tr>
<tr>
<td>PEA</td>
<td>28 (65%)</td>
<td>25</td>
</tr>
<tr>
<td>Asystole</td>
<td>9 (21%)</td>
<td>4</td>
</tr>
</tbody>
</table>

Background information of the 43 patients, the indication for the procedure in the cath lab, the culprit coronary artery and the initial rhythm at cardiac arrest. AMI: acute myocardial infarction, PCI: percutaneous coronary intervention, CABG: coronary artery bypass grafting, STEMI: ST elevation myocardial infarction, NSTEMI: non-ST elevation myocardial infarction, LM: left main artery, LAD: left anterior descending artery, Cx: left circumflex artery, RCA: right coronary artery, VT: ventricular tachycardia, VF: ventricular fibrillation, PEA: pulseless electrical activity.
During the study period, a total of 6350 PCIs were performed of which 3058 patients were treated for acute ST-elevation myocardial infarctions (STEMI). A total of 118 patients were in cardiogenic shock and 81 required defibrillations due to VF or ventricular tachycardia (VT).

Mechanical chest compressions were used in 43 patients of which 31 (72%) were males. Patients were between ages 31 and 86 years (mean age: 73.3 years). The patients were admitted for STEMI, non-ST-elevation myocardial infarct (NSTEMI), elective PCI and pericardiocentesis (Table 1). The culprit artery was the left anterior descending artery (LAD) or the left main in 81% of the cases and 65% of the patients had PEA as the initial rhythm during cardiac arrest (Table 1).

A total of 36 PCIs were attempted in the 42 patients with coronary disease, of which 27 PCIs were considered ‘technically successful’ procedures defined as a residual stenosis <50% at the site of the target lesion and achieving TIMI 2 or TIMI 3 blood flow with ongoing mechanical chest compressions (Table 2). Reasons for unsuccessful PCIs were primarily complex lesions, distal embolisation or no reflow in the coronary arteries and not primarily due to the use of LUCAS™. A total of 39 patients were treated with LUCAS™, and mean treatment time was 28.15 min (SEM ± 3.4, range: 1–90 min). Seventeen patients were discharged from the catheterisation laboratory, one with ongoing treatment for the 11 survivors was 16.5 min (SEM ± 3.8, range: 1–50 min).

All 12 patients, who were discharged from hospital, suffered rib fractures and, possibly, sternal fractures. One of the survivors suffered a ruptured spleen and gastric ventricle due to user error. Six autopsies were performed on the remaining thirty-one patients who died, and rib fractures were seen in all six patients and sternal fractures in five patients. One autopsy showed a small bleeding around the aortic arch. No other potential injuries of consequence due to mechanical chest compressions were found.

During the study period, only four patients with a lengthy cardiac arrest were treated with manual chest compressions alone and all four patients died.

### 3. Discussion

We have retrospectively analysed the effect of using a mechanical chest compression device as an adjunctive treatment for patients who suffer extended periods of cardiac arrest following arrival in the catheterisation laboratory and found that >25% of the patients could eventually be discharged to their homes in good neurological condition (CPC 1).

Although approximately 2,000,000 patients undergo PCI yearly worldwide, during the procedure, only a very small minority of patients will suffer a protracted cardiac arrest episode that results in death. However, performing effective manual chest compressions during a PCI in the catheterisation laboratory is linked with obvious culprit lesion. All five patients with myocardial rupture and tamponade died. Three patients were referred for emergency cardiac surgery, of which two died during surgery and one postoperatively. In 31 patients, the PCI procedures were performed with ongoing mechanical chest compressions and in four patients, only an angiogram was performed during mechanical chest compressions. Of the 17 patients who were discharged from the catheterisation laboratory, 12 were eventually discharged from the hospital with 11 of them in CPC 1 (Fig. 2). The initial rhythm in the 12 patients discharged alive was VF in four patients, PEA in three patients and asystole in five patients. One of the discharged patients had incurred a serious hypoxic brain injury and later died at the referral hospital. One patient, who was successfully treated for cardiac tamponade, died at the oncology ward due to cancer, and one patient died post-procedurally in the ward due to therapy-resistant VF.

Of the 11 patients who were discharged in good condition, all underwent successful PCIs, and in eight of the cases the PCI was performed with ongoing LUCAS™, while in two cases, LUCAS™ was used during the angiography. The mean treatment time of LUCAS™ for the 11 survivors was 16.5 min (SEM ± 3.8, range: 1–50 min).

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### 2. Results

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extreme difficulties. Thus, it would be attractive to have a chest compression device that could deliver effective compressions while still allowing for continued PCI due to its radio-translucent design. LUCASTM is one of two available mechanical chest compressions devices, the other being AutopulseTM (Zoll Medical Corporation, Chelmsford, MA, USA), which has been used in the catheterisation laboratory. With LUCASTM, due to the anterior–posterior (AP) design of the piston, essentially all views (such as cranial, caudal, RAO and LAO) except the straight AP are available during the time LUCASTM is in place. Fortunately, the available views are almost always the preferred angiographic views during PCIs even without LUCASTM. Maneouviring the X-ray detector around LUCASTM is easily performed with a monoplane catheterisation laboratory and with more difficulty in a biplane laboratory. Although LUCASTM produces an excessive movement of the chest and is somewhat bulky, it should still always be feasible, although not always easy, to perform a PCI. But if a high-precision manoeuvre is required, such as positioning a coronary stent, LUCASTM can be temporarily paused for a few seconds, which remedies the situation of excessive chest movement during stenting.

How well does LUCASTM work? From animal data, LUCASTM has been shown to maintain a positive coronary perfusion pressure and restore at least 60% of the cerebral blood flow in pigs.3,4 In one particular patient, LUCASTM maintained circulation for 25 min, during which time the patient was in VF. Following PCI and defibrillation, the patient regained consciousness in the catheterisation laboratory and was later discharged in CPC 1. 15 In patients with cardiac arrest and ongoing treatment with LUCASTM, Larsen et al. found that a mean systolic blood pressure of 70 mmHg (range: 60–110 mmHg, n = 11) could be attained,8 which mirrors our own experience. However, all of the patients in the study by Larsen et al. were already in cardiac arrest before arrival to the catheterisation laboratory and all these patients died. On the contrary, all the patients in our study arrived to the catheterisation laboratory with intact circulation and of these, 11 of 43 patients survived to discharge.

The main issue for patients in cardiac arrest is to restore circulation to the brain and only thereafter to attempt to remedy the cause of the cardiac arrest. Other means to solve restoration of blood flow in cardiac arrest in the catheterisation laboratory has been through percutaneous cardiopulmonary bypass (PCPB) or percutaneous left ventricular assist devices (PLVADs) such as Impella (Abiomed Inc., Danvers, MA, USA) or Tandem HeartTM (CardiacAssist Inc., Pittsburgh, PA, USA). Several small studies using PCPB in intractable cardiac arrest have been reported with good results. 13–15 PCPB and PLVADs seem to be effective but are usually take longer to initiate and can require extra staff with special skills, such as a cardiovascular surgeon and a perfusionist. LUCASTM may, on the other hand, be applied quickly (<1 min) and may also be used as a bridge to a PLVAD or PCPB.

There are several limitations to our study. First, we were unable to find any remaining recordings of continuous blood pressure measurements or coronary perfusion pressures during the periods of mechanical chest compressions, but in our experience, the circulation attained mirrors that found by Larsen et al. Second, two surviving patients had use of mechanical chest compressions for a limited time, less than 5 min. However, both patients had undergone treatment with CPR and manual compressions for several minutes prior to the application of LUCASTM. Third, this is a retrospective registry study and is limited in size. Still, our centre is the first one to ever use LUCASTM during PCI in the catheterisation laboratory and we have collected a large cohort of patients, mainly from our STEMI population.

During the analysed period, mechanical chest compressions during prolonged CPR were not used in four patients who were either very old or suffering from severe co-morbidities.

Another issue of importance is if mechanical chest compressions cause more injuries to patients than manually performed chest compressions. Manual chest compressions are well known to cause traumatic complications 16,17 and this is also likely to occur with mechanical chest compression devices to at least a similar degree.18 Reports of injuries caused by manual chest compressions are, however, difficult to compare due to different methodologies used,19 but a large ongoing randomised trial comparing manual chest compressions to mechanical chest compressions in out-of-hospital cardiac arrest will address this issue (the LINC study, see www.clinicaltrials.gov).

Among our patients, only one patient suffered severe injuries due to treatment with LUCASTM, but this was due to misapplication, as the LUCASTM device was placed too low and started performing compressions in the upper abdomen, which caused traumatic rupture of both the gastric ventricle and the spleen. Both the gastric bleeding as well as the ruptured spleen was revealed and successfully treated during abdominal surgery following PCI. All survivors showed evidence of likely suffering from non-debilitating rib fractures and possibly sternal fractures. Similar injuries were seen in the limited number of autopsies performed on the deceased patients.

One patient suffered an anoxic brain injury. It is unclear why this occurred, but it is likely due to a delay in applying LUCASTM, as this was one of the early patients that LUCASTM was used in, and this delay caused an insufficient cerebral perfusion leading to the injury.

Finally, one great advantage of mechanical chest compressions during cardiac arrest emergencies in the catheterisation laboratory is the ability to provide continuous chest compressions. Any interruption of chest compressions compromises both heart and brain blood flow. Such interruptions are a leading cause of poor outcome from cardiac arrest wherever it occurs. In circumstances where PCI or pericardiocentesis can correct the underlying cause of cardiac arrest, the provision of continued perfusion during such emergent procedures can result in not only lives being saved, but normal functioning neurological outcomes being achieved as well.20

4. Conclusions

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 catheterisation laboratory, especially in patients with an initial rhythm of VF.

Conflict of interest statement

Dr. Olivecrona and Dr. Friberg have received lecture honorariums from Jolife AB, Lund, Sweden.

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References


