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Clinical paper

Continuous mechanical chest compression during in-hospital cardiopulmonary resuscitation of patients with pulseless electrical activity☆

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ABSTRACT

Survival after in-hospital pulseless electrical activity (PEA) cardiac arrest is poor and has not changed during the last 10 years. Effective chest compressions may improve survival after PEA. We investigated whether a mechanical device (LUCAS™-CPR) can ensure chest compressions during cardiac arrest according to guidelines and without interruption during transport, diagnostic procedures and in the catheter laboratory.

Methods: We studied mechanical chest compression in 28 patients with PEA (pulmonary embolism (PE) $n = 14$; cardiogenic shock/acute myocardial infarction; $n = 9$; severe hyperkalemia; $n = 2$; sustained ventricular arrhythmias/electrical storm; $n = 3$) in a university hospital setting.

Results: During or immediately after CPR, 21 patients underwent coronary angiography and or pulmonary angiography. Successful return of a spontaneous circulation (ROSC) was achieved in 27 out of the 28 patients. Ten patients died within the first hour and three patients died within 24 h after CPR. A total of 14 patients survived and were discharged from hospital (13 without significant neurological deficit). Interestingly, six patients with PE did not have thrombolytic therapy due to contraindications. CT-angiography findings in these patients showed fragmentation of the thrombus suggesting thrombus breakdown as an additional effect of mechanical chest compressions. No patients exhibited any life-threatening device-related complications.

Conclusion: Continuous chest compression with an automatic mechanical device is feasible, safe, and might improve outcomes after in-hospital-resuscitation of PEA. Patients with PE may benefit from effective continuous chest compression, probably due to thrombus fragmentation and increased pulmonary artery blood flow.

1. Introduction

The incidence of pulseless electrical activity (PEA) after in-hospital cardiac arrest (IHCA) is unchanged for the last 10 years (29–37%), and similar to the incidence of asystole (30–39%). Both PEA and asystole have similar rates of survival to hospital discharge (about 10%).[1–4] Ventricular fibrillation (VF) accounts for 23–40% of IHCAs and has higher rates of survival (30–40% to hospital discharge) due to effective treatment with defibrillation. Survival from PEA and asystole depends on treating the underlying cause of cardiac arrest and this often requires a longer period of chest compressions (CC). Studies show that high quality CC is difficult to achieve on manikins and real patients during long periods of resuscitation even when performed by hospital staff.[5–9]

Pulseless electrical activity is often seen after pulmonary embolism (PE) or coronary artery thrombosis (e.g., main-stem occlusion) and is associated with poor survival.[10] Thrombolytic treatment during CPR for PE induced cardiac arrest has been shown to have good survival in small case series but larger case series have not shown this.[11,12]

Chest compressions are important for the defibrillation success and survival from VF, both in humans[13,14] and animals.[15] We aimed to evaluate if effective continuous chest compression

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Table 1
Consecutive patients with PEA undergoing CPR with LUCAS for IHCA.

	Gender	Age	Underlying diagnosis for PEA	LUCAS compression (min)	Outcome
1.	Female	47yo	Fulminant pulmonary embolism	50	ROSC
2.	Male	60yo	STEMI/main stem thrombosis	85	ROSC
3.	Male	68yo	Fulminant pulmonary embolism	100	Survival
4.	Female	74yo	Fulminant pulmonary embolism	20	Survival
5.	Female	81yo	Fulminant pulmonary embolism	35	Survival
6.	Male	66yo	Fulminant pulmonary embolism	25	Survival
7.	Female	60yo	Fulminant pulmonary embolism	10	Survival
8.	Male	64yo	STEMI/stent-thrombosis prox. LCx	15	Survival
9.	Male	72yo	STEMI/thromb. occlusion prox. LAD	120	Survival
10.	Male	54yo	Fulminant pulmonary embolism	20	Survival
11.	Female	60yo	Fulminant pulmonary embolism	10	Survival
12.	Female	35yo	Fulminant pulmonary embolism	180	ROSC
13.	Male	69yo	Severe hyperkalemia/cardiomyopathy	40	Survival
14.	Male	64yo	ICD-testing during CRT-ICD-implantation	60	ROSC
15.	Male	70yo	STEMI/stent-thrombosis prox. LAD	30	Survival/N
16.	Female	34yo	Fulminant pulmonary embolism	70	ROSC
17.	Male	82yo	PAVR/ during balloon-occlusion	45	ROSC
18.	Male	66yo	Amiodarone during incess. VT	60	Survival
19.	Female	78yo	Fulminant pulmonary embolism	20	ROSC
20.	Male	71yo	Cardiogenic shock/AMI	45	ROSC
21	Male	59yo	STEMI/left main-stem occlusion	75	Survival
22.	Male	71yo	NSTEMI/thrombolytic CABG-occlusion	20	ROSC
23.	Male	80yo	Fulminant pulmonary embolism	25	Deceased
24.	Female	59yo	Electric storm/ICD patient with DCM	40	ROSC
25.	Male	77yo	Fulminant pulmonary embolism	30	ROSC
26.	Female	51yo	Fulminant pulmonary embolism	60	ROSC
27.	Male	62yo	Severe hyperkalemia/patient on dialysis	10	Survival
28.	Male	69yo	Cardiogenic shock/AMI	25	ROSC

Survival = hospital discharge without significant neurological deficits (CRC 1 + 2).
Survival/N = hospital discharge with significant neurological deficits (CRC $\geq$ 3).
AMI = acute myocardial infarction. CABG = coronary artery bypass grafting. DCM = dilative cardiomyopathy. ICD = implantable cardioverter defibrillator. PAVR = percutaneous aortic valve replacement. NSTEMI = non-ST-segment elevation myocardial infarction. STEMI = ST-segment elevation myocardial infarction.

(rate of 100 min^{-1}, compression depth of 50 mm, 50/50 duty cycle and adequate recoil) using a mechanical chest compression device (LUCAS^{TM}) is safe and feasible during treatment of patients with PEA cardiac arrest.

2. Methods

Patients were enrolled from August 2006 to August 2008 in three European university hospitals (Lübeck and Dresden, Germany and Lund, Sweden). Resuscitation events were studied among patients that experienced cardiac arrest, defined by the documented loss of a pulse and respirations as well as the delivery of (initially manual) chest compressions. Out of hospital cardiac arrest (OHCA) cases were not included. Only patients with PEA as the underlying rhythm were investigated. Other cardiac arrest treatments included were: diagnostic imaging using coronary angiography, pulmonary angiography and CT-angiography during mechanical chest compressions. Imaging was followed by treatment with percutaneous coronary intervention (PCI) and thrombolysis when indicated. Following return of spontaneous circulation (ROSC) comatose patients were treated with hypothermia in the intensive care unit according to local protocols.

Continuous chest compressions were delivered mechanically using LUCAS^{TM} CPR (Jolife, Sweden). This device can be used to deliver chest compression according to the current guidelines[16,17] without interruptions during prolonged resuscitation, patient transport, acute diagnostic procedures, and during coronary angiography.[18–21] The use of LUCAS^{TM} for IHCA was left to the discretion of the resuscitation team, however, in all three centres the use of the LUCAS^{TM} device was already established for patients with ongoing CPR on the wards, the coronary care units, the intensive care units, and the catheterization laboratory for more than 12 months before the initiation of the present study. Following the intervention, all patients were intensively screened for life-threatening device-related complications and some of the deceased patients underwent a forensic necropsy.

The predefined endpoints were: ROSC, 24 hour survival, hospital discharge with Cerebral Performance Category (CPC) 1 or 2 and device-related complications.

3. Results

A total of 28 patients with PEA were included in the study. Most patients were enrolled in the university hospital Lübeck where 21 consecutive patients with PEA cardiac arrests on internal medicine wards were enrolled. Within the enrolment period there were 215 in-hospital resuscitations in the university hospital Lübeck including 52 patients with PEA. Between January 2008 and August 2008 a total of 4 non-consecutive patients were enrolled in the heart center Dresden, and 3 patients in the department of cardiology of the Lund university.

The 28 patients with PEA in the study included 10 were females, 18 males and the mean age was 64.4 $\pm$ 12 (mean $\pm$ SD) years (range 34–82 years). The underlying cause of PEA was: PE (n = 14), cardiogenic shock/acute myocardial infarction (n = 9), severe hyperkalemia (n = 2) and sustained ventricular arrhythmias/electric storm (n = 3). LUCAS^{TM} CC were performed for a median duration of 37.5 min (range 10 and 180 min) (Table 1).

During or directly after CPR, 21 patients underwent coronary angiography/pulmonary angiography. Initial ROSC was achieved in 27 out of 28 patients. Ten patients died within the first hour, another three patients died within 24 h after CPR. A total of 14 patients survived and were discharged from hospital (13 without significant neurological deficits – CPC 1 and 2). Six of the 14 patients with PE did not undergo thrombolytic therapy because they had contraindications. CT-angiography in these patients showed fragmentation of the thrombus even though thrombolytic therapy was not was given (Fig. 1). None of the patients exhibited significant or serious injuries

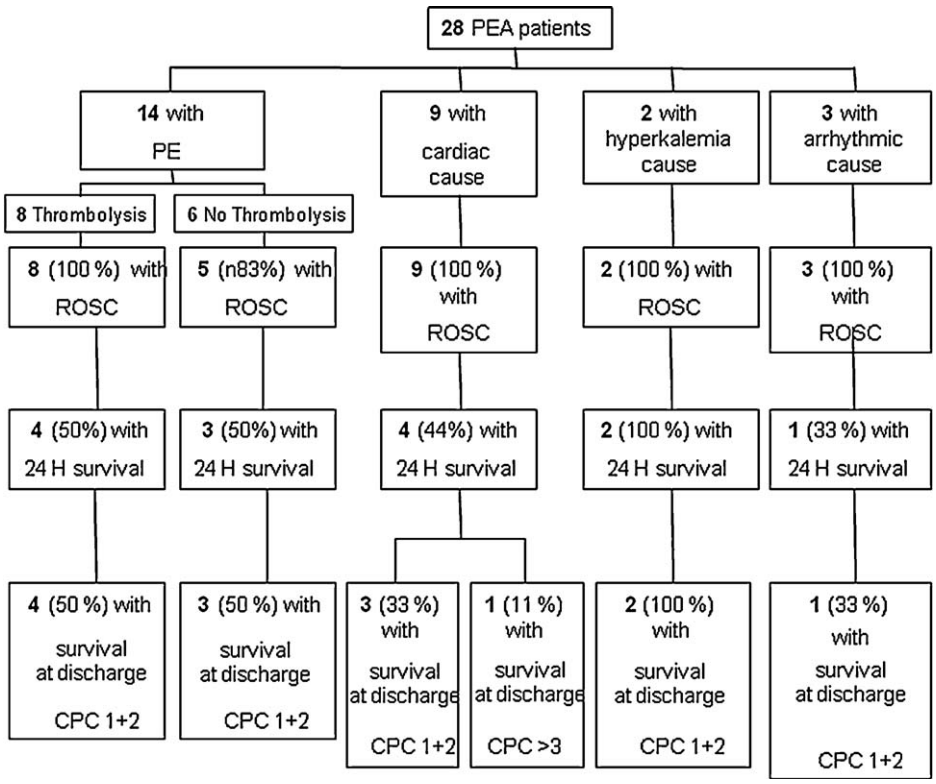

Fig. 1. Flow chart of treatment and outcome data for all 28 PEA patients studied, according to the predefined endpoints: ROSC, 24 h survival, hospital discharge with good Cerebral Performance Category (CPC 1 + 2).

associated with LUCAS™ CC. The deceased patients that underwent forensic necropsy showed no evidence of a device related injury.

4. Discussion

We report a case series of good outcomes after continuous chest compression using LUCAS™ and early imaging and intervention for IHCA due to PEA. We found that almost 50% of the patients survived to discharge to their homes with good or moderate neurological function (CPC 1 and 2).

Neurologically intact survival rates have not improved in more than a decade, and overall survival rates of in-hospital-cardiopulmonary resuscitation are still alarmingly low for patients with PEA. Cardiopulmonary resuscitation of IHCA has been shown to be inconsistent and often does not meet guideline recommendations, even when performed by well-trained hospital staff.[9,23] Studies show that even experienced rescuers produce shallower and slower compressions over time, without being aware. In an effort to improve manual CPR, several mechanical devices are available and mainly in use by out-of-hospital-emergency medical services. The setting and environment, the response time, the medical and diagnostic equipment, and the patient population are all different for CPR after IHCA, compared to OHCA. Resuscitation from IHCA would be expected to be more successful, but even with experienced hospital staff and CPR training programs, resuscitation skills deteriorate over time. Furthermore, translation from training to actual cardiac arrest settings and rescuer fatigue during CPR limit IHCA CPR quality.[23] Mechanical CPR devices offer new opportunities for IHCA resuscitation as they help to sustain circulation with consistent compressions according to the guidelines during prolonged resuscitation efforts, transportation, and during interventional procedures such as PCI. There is clinical evidence that mechanical CPR devices provide chest compressions more reliably at a set rate and depth and thus generate better hemodynamic characteristics than manual chest compressions.[24–26] Furthermore, using mechanical CPR it is possible to "buy time" in an effective manner ensuring adequate circulation and allowing interventional procedures treatments – i.e., primary angioplasty or computed tomography. In addition to these practical benefits, experimental data show significantly increased flow and ROSC levels with mechanical CPR devices compared to manual chest compression.[27,28]

Another significant benefit of mechanical chest compression for the clinical management of IHCA is becoming clearer: in the catheter laboratory, one of the pivotal points of IHCA, interventions are not possible without interrupting manual chest compressions. Usually, CPR is difficult in the catheter laboratory because effective manual chest compressions are difficult due to the gantry around the patient's chest and the height of the table. Furthermore, interventions are hindered during manual compressions there is significant radiation exposure to the staff performing CPR. Our experience from several IHCA cases treated with the LUCAS device in the catheter laboratory supports previous observations that this device is feasible, safe and highly effective in this setting. Mechanical chest compressions are also useful during emergency computer tomography.[22]

Besides the significant advantages of continuous CPR, effective external chest compression may also provide additional therapeutic effects in patients with PEA due to PE. After long-term LUCAS-compression we found considerable CT evidence of mechanical thrombus fragmentation as a surrogate marker of increased pulmonary artery flow (Figs. 2 and 3).

Thus, from our point of view, the integration of an automatic mechanical compression device into the in-hospital chain of survival, significantly improves IHCA resuscitation management and infrastructure, and, above all, seems also to increase clinical outcome (compared to data from IHCA registries).

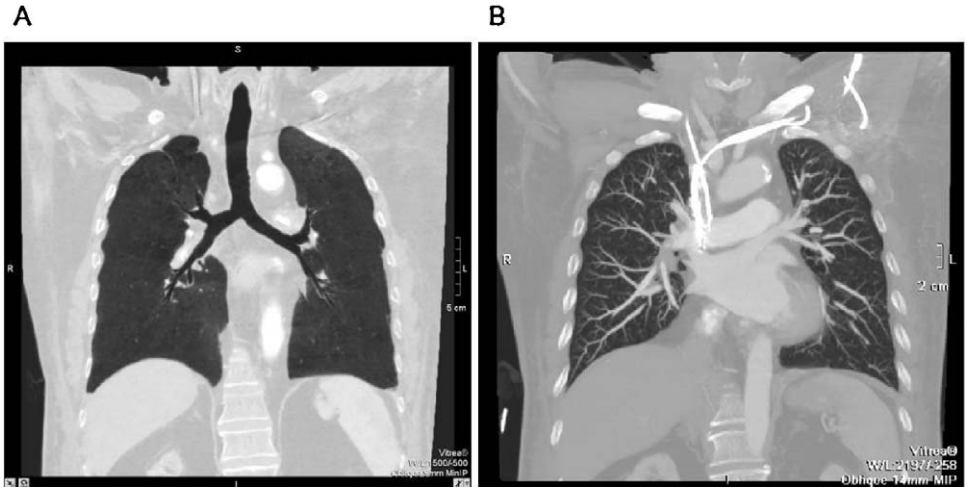

Fig. 2. Frontal reconstructed CT images in lung-window (A) and pulmonary angiography (B) setting demonstrating no injuries of thoracic and abdominal organs after long-term LUCAS-compression.

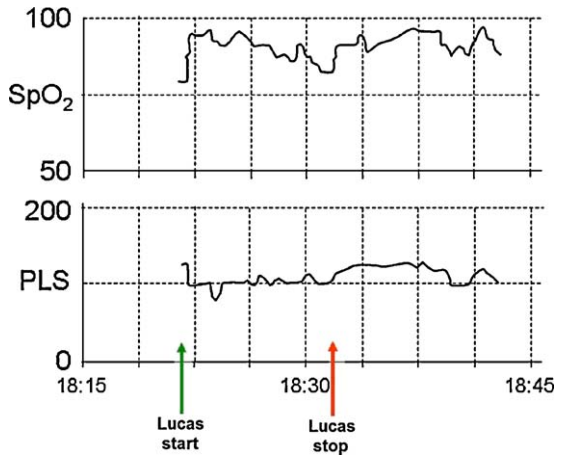

Fig. 3. The upper panel shows the increase in SpO_2 on pulse oximetry during LUCAS™ chest compression rising from approximately 55% to approximately 90%. The lower panel shows the pulse rate during LUCAS™ chest compressions (stable around 100/min). The green arrow shows were LUCAS™ chest compressions starts and the red arrow shows were the patient regains circulation (ROSC).

Our study has a number of weaknesses. We have presented a small number of cardiac arrests that represent only a small proportion of all cardiac arrests occurring over the time period. Most of the cases came from one centre (Lübeck) and selection bias will have contributed to the good outcomes. We do not report the overall outcomes for all cardiac arrest patients in the study centres during the time of the study. There is no formal control group to make a comparison with standard CPR. We cannot say for certain which aspect of care resulted in the good outcomes we report.

Ongoing multi-centre randomized controlled studies will provide more evidence about the role of compression devices in CPR. Our findings do however suggest that CPR for IHCA with a mechanical device is safe and feasible, and can help improve the care of IHCA patients.

5. Conclusion

Continuous chest compression with an automatic mechanical device seems to be feasible, safe, and might improve outcomes after in-hospital-resuscitation of PEA cardiac arrest. Patients with PE may benefit from effective continuous chest compression, probably due to thrombus fragmentation and increased pulmonary artery blood flow.

Conflict of Interest statement

There are no potential conflicts of interest to disclose.

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