Evaluation of LUCAS, a new device for automatic mechanical compression and active decompression resuscitation

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Abstract

LUCAS is a new gas-driven CPR device providing automatic chest compression and active decompression. In an artificial thorax model, superior pressure and flow were obtained with LUCAS compared with manual CPR. In a randomized study on pigs with induced ventricular fibrillation significantly higher cardiac output, carotid artery blood flow, end-tidal CO2, intrathoracic decompression-phase aortic- and coronary perfusion pressures were obtained with LUCAS-CPR (83% ROSC) compared to manual CPR (0% ROSC). In normothermic fibrillating pigs, the ROSC rate was 100% after 15 min and 38% after 60 min of LUCAS-CPR (no drug treatment). The ROSC rate increased to 75% if surface cooling to 34 °C was applied during the first 30 min of the 1-h resuscitation period. Experience with the first 20 patients has shown that LUCAS is light (6.5 kg), easy to handle, quick to apply (10-20 s), maintains a correct position, and works optimally during transport both on stretchers and in ambulances. In one hospital patient with a witnessed asystole where manual CPR failed, LUCAS-CPR achieved ROSC within 3 min. One year later the patient’s mental capacity was fully intact. To conclude, LUCAS-CPR gives significantly better circulation during ventricular fibrillation than manual CPR.

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Keywords: Active compression-decompression; Cardiopulmonary resuscitation (CPR); Coronary perfusion pressure; End-tidal carbon dioxide; Hypothermia; Return of spontaneous circulation (ROSC)

Resumo

LUCAS é um novo aparelho de RCP que funciona com gás e que faz compressão torácica automática e descompressão activa. Num estudo randomizado em porcos com fibrilação ventricular induzida foram estudados o débito cardíaco, fluxo sanguíneo da artéria carótida, CO2 no final da expiração e pressões de perfusão coronária e aórtica na fase de descompressão intratorácica, que se verificou serem significativamente mais elevadas com a RCP com LUCAS (83% ROSC) quando comparado com RCP Manual (0% ROSC). Em porcos normotérmicos em fibrilação a taxa de ROSC foi 100% ao fim de 15 min e 38% ao fim de 60 min de RCP-LUCAS (sem tratamento farmacológico). A taxa de ROSC aumentou para 75% se fosse aplicado arrefecimento superficial até aos 34 °C nos primeiros 30 min da primeira hora do período de reanimação. A experiência com os primeiros 20 doentes mostrou que o LUCAS é leve (6.5Kg), fácil de manusear, rápido de aplicar (10-20 s), mantém uma posição correcta e trabalha de forma óptima durante o transporte em macas ou em ambulâncias. Num doente hospitalar com uma assistolia testemunhada em que a RCP manual falhou a RCP-LUCAS conseguiu ROSC em 3 min. Um ano mais tarde a capacidade intelectual do doente estava intacta. Para concluir, a RCP-LUCAS dá uma circulação significativamente melhor que a RCP manual durante a fibrilação ventricular.

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Palavras chave: Compressão-descompressão activa; Ressuscitação cardíaco-pulmonar (RCP); Pressão de perfusão coronária; Dióxido de carbono tele-expiratório; Hipotermia; Retorno de circulação espontânea (ROSC)

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Resumen

LUCAS es un nuevo aparato para reanimación cardiopulmonar impulsado por gas que proporciona compresiones torácicas y descompresiones activas automáticas. En un modelo de tórax artificial, se obtuvo presión y flujo superiores con LUCAS comparado con reanimación cardiopulmonar manual. En un estudio realizado en cerdos con fibrilación ventricular inducida se alcanzaron valores significativamente mayores de gasto cardíaco, flujo de arteria coronaria, CO₂ espiratorio, presiones de perfusión coronaria y áortica en fase de descompresión con reanimación manual con LUCAS (83% ROSC) comparado con reanimación manual (0% ROSC). En cerdos normotérmicos en fibrilación ventricular, la tasa de retomo a circulación espontánea (ROSC) fue de 100% después de 15 minutos y de 38% después de 60 minutos de LUCAS-RCP (sin tratamiento con drogas). La tasa de ROSC a 75% si se aplicaba a cerdos normotérmicos en fibrilación arterial en fase de descompresión con reanimación con LUCAS (83% ROSC) comparada con reanimación manual (0% ROSC). En cirugía significativamente mayor en pacientes con un paro presenciado en asistolia, con ROSC-LUCAS consiguió ROSC en tres minutos. Un año más tarde la capacidad mental del paciente estaba intacta. Para concluir, durante la fibrilación paro presenciado en asistolia, sonde la RCP manual falló, RCP-LUCAS consiguió ROSC en tres minutos. Un año más tarde la capacidad mental del paciente estaba intacta. Para concluir, durante la fibrilación ventricular la RCP-LUCAS proporciona una circulación significativamente mejor que la RCP manual.

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Palabras clave: Compresión-descompresión activa; Reanimación cardiopulmonar (RCP); Presión de perfusión tisular; Dióxido de carbono espiratorio; Hipotermia; Retorno a circulación espontánea

1. Introduction

Cardiac arrest, either as asystole or as ventricular fibrillation (VF), is the most dramatic situation in medicine. Since Kouwenhoven and coworkers published their landmark article in 1960 [1], manual closed-chest compressions (combined with mouth-to-mouth ventilation) has been established as the initial treatment of choice for circulatory arrest, followed by defibrillation as soon as the equipment is available, if VF is the cause of the collapse. With proper training, anyone, anywhere can initiate cardio-pulmonary resuscitation (CPR). However, due to fatigue, manual CPR cannot be given for more than a few minutes before it becomes ineffective [2], and it cannot be given effectively at all during transport [3]. Most cardiac arrests occur out-of-hospital and the survival rates are very poor; in most published reports the 1-year survival rate is less than 5%. In a randomized study, Plaisance and coworkers [4] compared standard manual CPR (n = 377 patients) with active compression/decompression CPR performed manually with the CardioPump (AMBU, Copenhagen, Denmark) (n = 373 patients). The 1-year survival rate was very poor in both groups, 2 versus 5% (P = 0.03); all resuscitation efforts with either method were performed only at the scene of the cardiac arrest, and only if they were successfully resuscitated at the scene were the patients transported to hospital. To prevent fatigue, the rescuers were instructed to alternate after each 3 min of CPR. The study of Plaisance et al. demonstrates the need for a mechanical device giving adequate compressions/decompressions continuously until the patient can be delivered to a hospital with all facilities for the treatment of heart disease, including direct PTCA and heart surgery.

Most devices for mechanical chest compression in use today have operational limitations because they take too long to apply, they are cumbersome to install and operate, they are unstable on the chest, heavy, and expensive to purchase [5]. Therefore, no mechanical device for chest compression/decompression currently is used routinely in clinical practice, in spite of the obvious limitations of manual CPR. Recently, a new device named LUCAS, has been made commercially available (Figs. 1 and 2). It is designed to give automatic mechanical chest compression and active decompression. It is portable and works during transport both on stretchers and in ambulances.

The aim of the present investigation was to compare the efficacy of LUCAS with that of manual compressions on an artificial thorax model allowing exact analysis of pressure- and flow-curves, and on a pig model in which relevant physiological variables could be registered. In an earlier study using the same pig model we studied the effects of adrenaline (epinephrine) and noradrenaline (norepinephrine) on end-tidal CO₂, coronary perfusion pressure and cardiac output during cardiopulmonary resuscitation [6]. In the present study we decided to eliminate all drug therapy in order to elucidate the effects of chest compressions per se. Data from the first clinical pilot study with LUCAS are also presented.

2. Material and methods

2.1. The artificial thorax model

A 25 l plastic drum made of polyvinyl chloride (PVC) was used as an artificial thorax (Fig. 3). A soft plastic bag (150 ml), simulating a heart, was included in the
drum. Pressure (P) was continuously measured in the bag. By means of a stiff tube penetrating the tight cork of the drum, the soft bag was connected to an artificial circulatory system including two artificial heart valves for flow direction. The plastic drum was filled with 20 l water and 5 l air, and regained its original shape after deformation. During compression of the drum manually or by means of the LUCAS, the soft plastic bag ejected fluid through the outlet valve (Vo) (Carbomedics aortic valve, 25 mm Ø). To mimic the Windkessel effect of the aorta, a side tube with trapped air (C) was included in the system. Resistance in the flow system was generated using a tube compressor (R), set to give a systolic pressure of around 100 mmHg when standard manual compressions were given by a normal-sized adult male trained in CPR. (The degree of clamping was adjusted on the base of pressure measurements before and after the resistance.) The flow created by drum compression was measured continuously by a flow probe (F), (Transonic Systems Inc. HT207, New York, USA). The filling pressure of the balloon (‘ventricle’) was adjusted by letting the flow run into an open reservoir (OR) placed at an appropriate level above the soft bag. Between the reservoir and the connection to the soft bag an inlet valve (Vi) (Medtronic Hall, mitral valve, 29 mm Ø) was inserted. Flow and pressure signals were sampled on a computer.

2.2. Manual chest compressions in the pig

Manual chest compressions were given by three male surgeons trained in CPR and with clinical experience of the procedure. The surgeons were of normal size with a body weight in the range of 70–80 kg. Each surgeon worked in 3-min periods, compressing the lower one third of the sternum at a target rate of 100 compressions/min. The surgeons were instructed to give the compressions with the force they would have used on an
adult patient of normal size. The experimental protocols are depicted in Fig. 4.

2.3. The properties of LUCAS

The LUCAS is a gas-driven device that provides automatic mechanical compression and active decompression. It consists of a silicon rubber suction cup similar to that used in the CardioPump and a pneumatic cylinder mounted on two legs which are connected to a stiff back plate (Figs. 1 and 2). The cover of the pneumatics, the legs, and the back plate are made of a composite material that does not conduct electricity. The system is powered by oxygen or air from a cylinder, the gas system in ambulances or the gas outlets in hospitals. The maximum compression depth is 52 mm and the maximum compression force is 500 N. The decompression force is 410 N. A regulator inside LUCAS ensures that the same force will be obtained if it is run on air or oxygen. The gas connector fits the outlets for both oxygen and air and it can be used with
gas sources with pressures ranging from 4 to 7 bar (400–700 kPa). The default setting for the compression/decompression frequency is 100 per minute. The height of the suction cup can be adjusted to fit patients with an anteroposterior thorax diameter in the range of 17–26.5 cm. The weight of the device is 6.5 kg and when stowed in a bag, its dimensions are 32 × 64 × 23 cm. When it is mounted, the dimensions are 50 × 53.8 × 22.8 cm. LUCAS is CE-marked and is commercially available in Europe since December 2001 (Jolife AB, Lund, Sweden; www.jolife.com).

### 2.4. Experimental animals

A total of 100 Swedish-bred specific pathogen free pigs with a mean weight of 22 kg (range 20–26 kg) were used. The mean external anteroposterior diameter of the thorax at the site where the chest compressions were given, i.e., at the inferior one third of the sternum, was 20 ± 1 cm (range, 18–23 cm) (Fig. 5).

All the animals received humane care in compliance with the Guide for the Care and Use of Laboratory Animals, published by the National Institutes of Health (NIH publication 85–23, revised 1985). The Institutional Review Board for animal experimentation at the University of Lund, Sweden, approved the experimental protocols. All animal experiments were designed according to the Utstein-style guidelines [7].

### 2.5. Anesthesia and preparation

The pigs had free access to water but were not allowed to eat on the day of experiment. They were anaesthetized with an induction dose of intramuscular ketamine (30 ml/kg). Sodium thiopental (5–8 mg/kg) and atropine (0.05 mg/kg) were given intravenously before tracheotomy. Anaesthesia and muscle paralysis were maintained with a continuous infusion of 30 ml/h of a 10% glucose solution containing ketamine (16 mg/ml) and pancuronium (0.6 mg/ml). In study Groups III and IV, midazolam (0.06 mg/ml) was also added to the infusion. Macrodex (up to 250 ml) was given to keep the central venous pressure within a normal range of 3–8 mmHg on a PEEP of 8 cm H2O.

For monitoring of intrathoracic aortic pressure, a catheter was introduced via a direct puncture of the left carotid artery in order to avoid ligation of the artery. The tip of the catheter was inserted into the thoracic aorta and in the same way a catheter was inserted into the right atrium via the left external jugular vein (at autopsy these positions were confirmed). Separate catheters were placed inside an artery and a vein for withdrawal of blood samples. In Group I, a Swan–Ganz catheter (7.5 F) was inserted into the pulmonary artery via the right external jugular vein. An ultrasonic blood flow probe (3 mm) connected to a flow meter (Transonic Flowmeter T201D) was placed around the right carotid artery. A Foley catheter was inserted into the urinary bladder through a suprapubic cystotomy. The temperature was measured with a temperature probe placed in the oesophagus. The animals were kept normothermic by a heating system in the operation table, if not actively cooled, as for two thirds of the animals in Group IV. The mean temperature for the pigs in Groups I–III at the end of the experiments was 37.2 ± 1.0 °C (range, 36.5–38.4 °C).

### 2.6. Experimental protocol

Fig. 4. The design of the pig experiments. The number of pigs with ROSC (return of spontaneous circulation) is indicated within the ROSC rectangle. In each case, defibrillation occurred at the end of the CPR.
intention was to identify potential physiological differences between ROSC and non-ROSC-pigs during CPR.

In Group III, the animals were randomized into eight different subgroups with 15, 20, 25, 30, 35, 40, 50 and 60 min of LUCAS-CPR before defibrillation. The aim was to determine the frequency of ROSC in each group.

In Group IV, the pigs were randomized either to normothermia or to cooling, the latter divided into two subgroups: in one VF was induced before cooling (surface cooling group), and in one after cooling to 32 °C (hypothermia group). The animals were placed within a strong plastic bag with holes for catheters, flow probe cable and the silicon rubber suction cup of LUCAS. In the surface cooling group the plastic bag was filled with ice cubes directly after induction of VF. When the oesophageal temperature reached 34 °C (after about 30 min, range 28–33 min), the ice bag was removed. The temperature continued to fall to about 32 °C at 60 min, the time at which defibrillation was attempted. For the ROSC animals, the oesophageal temperature stabilized at around 31 °C 1 h after ROSC. In the hypothermia group, cooling was done with the method described above but VF was induced at 32 °C and LUCAS-CPR was run for 60 min before defibrillation, at which time the temperature had stabilized at around 31 °C.

VF was induced with a 5–20 mA, 6 Hz and 30 V alternating current delivered to the epicardial surface via a needle electrode. Circulatory arrest was confirmed by a fall in arterial blood pressure and end-tidal CO2 concentration and an ECG showing VF. Chest compressions were started after an interval of 90 s (Group I and II) or 30 s (Group III and IV).

Defibrillation was attempted as soon as possible (within 10 s) after the interruption of chest compressions with a direct current (DC) countershock of 300 J. In case of persistent VF, DC countershocks of 360 J were administered up to 3 times if necessary. If VF or asystole

Fig. 5. Transverse section of a 23 kg pig just distal to processus xiphoideus. The heart ventricles are not compressed directly between the sternum and the spine during chest compressions due to the central position of the ventricles within the thoracic cavity. The anteroposterior diameter in this pig was 19.5 cm. Normal ventilated lungs (upper left), atelectatic lungs after disconnection from the ventilator (upper right), lungs extirpated (lower left), and manual forceful compression without direct compression of the ventricles between sternum and the spine (lower right).
performed after four countershocks (with short periods of manual chest compression between each shock), resuscitation was defined as unsuccessful. The pigs with ROSC were monitored for 2 h, after which they were euthanized and autopsied. The position of the aortic and central venous catheters was especially checked, as were the heart valves, heart septum and ductus arteriosus. The aortic and pulmonary valve function was investigated by testing for leakage in a vertical position, and the tricuspid and mitral valves were tested by a quick injection of saline in a Foley-catheter placed intraventricularly via a stab wound through the ventricular wall. The autopsies did not show any pathology (special care was taken when the hearts of the non-survivors were investigated).

2.7. Haemodynamic measurements

Pressure and blood flow signals were sampled 50 times/s and the mean value for each variable was recorded every 5 s during the whole experiment, using a computer supplied with a data acquisition system (TestPoint, Capital Equipment Corporation, Billerica, MA). Coronary perfusion pressure was continuously calculated by the computer as the difference between the intrathoracic aortic and right atrial pressure during the decompression phase.

2.8. Blood gas analysis

Blood gases and electrolytes were analyzed directly after the sample had been obtained using a blood gas analyzer (ABL 505, Radiometer, Copenhagen). Arterial and mixed venous O2-saturations (SaO2, SvO2) and total haemoglobin concentration were analyzed with a multi-wave-length oximeter (OSM3, Radiometer, Copenhagen) using the pig mode. In the hypothermic animals, the blood gas apparatus was adjusted to measure at the same temperature as the pig.

2.9. Ventilatory settings and measurements

Pressure-regulated, volume-controlled ventilation (Servo Ventilator 300, Siemens, Solna, Sweden) was used to obtain a stable minute volume. Ventilatory support was continued throughout all the experiments with a minute volume of 5 l/min at 20 breaths/min and a PEEP of 8 cm H2O in Group I and II, and a minute volume of 7.5 l/min at 25 breaths/min and a PEEP of 8 cm H2O in Group III and IV. An inspired oxygen concentration (FiO2) of 0.21 was used throughout, except during the periods of chest compressions, when it was set to 1.0.

The ventilation was not synchronized with the chest compressions. A prototype infra-red CO2 analyzer (Servotek AB, Arlöv, Sweden) was used, which has a function similar to that of the Servo 930 CO2 analyzer, using an infra-red source and a detector placed astride the Y-tubing (main stream). The analyzer has a response time of 5 ms, a low noise level and a full-scale deflection of 10% CO2. It was calibrated to zero with air and also calibrated with gas containing 5.05 ± 0.010% CO2 in air (Alfax AB, Arlöv, Sweden). End-tidal CO2 was monitored continuously and the value was recorded once a minute on a computer during the course of the experiment.

2.10. Anteroposterior thorax diameter in humans

The anteroposterior thorax diameter of 50 men and 15 women was measured at the level where external chest compressions should be given. This was done by placing the subjects on their backs close to a wall, lowering a stiff plate angled at 90° to the wall and attached to the wall until it just touch a point between the middle and lower third of the sternum, and measuring the distance from the lower edge of the plate to the floor.

2.11. Clinical pilot study with LUCAS

Permission for a clinical pilot study with LUCAS, including 20 patients, was given by the Medical Ethics Committee at the University of Lund. The study was designed to see if the device was easy and safe to use. The test was done when standard cardiopulmonary resuscitation had failed, as a last extra chance to save the patient’s life. The device used on the first patients in this pilot study was a prototype with the same pneumatic properties as in the later model of LUCAS, but with an aesthetically less attractive appearance.

2.12. Statistical analysis

All results are expressed as the mean ± standard error of the mean (S.E.M.). For statistical analysis the unpaired Student’s t-test was used.

3. Results

3.1. Manual CPR vs. LUCAS-CPR in the artificial thorax model

Typical pressure-flow curves for the artificial thorax model are presented in Fig. 6; in the left panel the rescuer performs manual CPR as he would have done in a clinical situation, and in the middle panel his performance during 5 s of maximal effort is shown. As seen in the right panel, LUCAS-CPR creates pressure-flow curves quite different from those seen during manual CPR, i.e. the area under the curves produced.
by LUCAS is greater, with a corresponding increase in mean pressure and flow. The explosivity of the gas-driven pneumatics in LUCAS creates an instant increase and decrease in pressure, with a 50% duty cycle regarding both time and flow. The high peak pressures caused by maximal manual CPR cannot be maintained during the compression phase and therefore can not compete in efficiency with the LUCAS-CPR, despite lower peak pressures with the latter.

3.2. Manual CPR vs. LUCAS-CPR in the pig (Group I)

There was no return of spontaneous circulation (ROSC) with manual CPR, whereas five of six animals had ROSC with LUCAS-CPR (Fig. 7). The diastolic and mean arterial pressures were significantly higher with LUCAS-CPR (Table 1). In Fig. 8 the pressure curves obtained from the intrathoracic aorta and the right atrium are superimposed. The areas between the curves in the decompression phase are greater during LUCAS-CPR than during manual CPR, indicating a higher myocardial perfusion pressure during LUCAS-CPR. The coronary artery perfusion pressure was around 10 mmHg with manual CPR and around 15 mmHg with LUCAS-CPR (Fig. 9).

The values obtained after 5 min of CPR are presented in Table 1. The cardiac output, end-tidal CO₂, right carotid arterial blood flow and coronary perfusion pressure were significantly higher with LUCAS-CPR. There was no significant difference in the blood gas values (except for a slightly higher P<sub>V</sub>O₂ value in the LUCAS-CPR group), which were within normal ranges in both groups. The five pigs with ROSC in the LUCAS-CPR group were followed for 2 h before being euthanized and autopsied. At the end of this observation period, the arterial pressure, carotid flow and blood gases were not significantly different from the baseline values obtained before induction of VF.

3.3. ROSC vs. non-ROSC after 30 min of LUCAS-CPR (Group II)

There was a 50% ROSC rate in this group of 16 animals. Pressure-flow curves during 30 min of LUCAS-
CPR without and with ROSC are shown in Fig. 10, upper and lower panels, respectively. The coronary perfusion pressure is shown in Fig. 11 and the end-tidal CO2 values in Fig. 12. No significant difference in any variables measured was seen after 5 and 15 min of LUCAS-CPR. In Table 2 values after 25 min of LUCAS-CPR are shown. Coronary perfusion pressure, end-tidal CO2 and right carotid arterial flow were significantly higher in the ROSC group. There was no significant differences in the blood gases except for PVCO2 at 25 min, 5.6 ± 0.4 vs. 7.7 ± 0.7 (P < 0.05) in the ROSC and non-ROSC pigs, respectively. The corresponding values for SvO2 at 25 min were 54 ± 6 and 34 ± 9% (P = 0.093). The animals with ROSC were followed for 2 h before being euthanized and autopsied. At the end of this observation period, the arterial pressures, blood gases and carotid blood flow were not significantly different from the baseline values obtained before the induction of VF.

3.4. ROSC after 15–60 min of LUCAS-CPR (Group III)

All animals achieved ROSC after 15 min of LUCAS-CPR, whereas beyond 15 min there was an increased rate of animals without ROSC without any obvious association with CPR time (Fig. 4). The mean coronary perfusion pressure and the end-tidal CO2 values of the last 5 min of the resuscitation period were calculated for each pig. For all animals with ROSC (n = 27), the mean coronary perfusion pressure was 15 ± 5 mmHg, compared with 2 ± 5 mmHg (P < 0.01) for the non-ROSC pigs. The corresponding values for end-tidal CO2 were 2.6 ± 0.7 and 2.0 ± 1.0% (P < 0.01), respectively. At the end of the observation period of 2 h, all ROSC pigs had blood pressure, end-tidal CO2, and carotid flow values that were not significantly different from the values obtained before the induction of VF.

3.5. LUCAS-CPR in normothermia versus hypothermia (Group IV)

The results obtained in the Group IV pigs are shown in Figs. 13–15 and in Table 3. The coronary perfusion pressure started to decrease after 20 min of CPR in the normothermic animals. The end-tidal CO2 values in the hypothermic group were stable throughout CPR whereas a decline over time was seen in the normothermic group. In the surface cooling group, the end-tidal CO2 value and the oesophagus temperature at 50 min were the same as in the hypothermic group, reflecting the reduced metabolism and CO2 production in these two groups at this point. The metabolic acidosis (base excess in Table 3) measured after 50 min of CPR was more pronounced in the normothermic pigs. The reactive hyperaemia after ROSC was higher in the normothermic group (Figs. 13–15).

3.6. Anteroposterior chest diameter in 65 adult humans

Both the mean and median anteroposterior diameter was 21 cm (range 17–26 cm).

3.7. Clinical experience with LUCAS

The pilot study, where LUCAS was used as a last resort in 20 cases where standard advanced CPR had failed, documented that LUCAS is easy to apply and easy to use. In most cases it took less than 20 s to apply. The staff appreciated the fact that one person could be used for other purposes during CPR.

In one clinical case the efficacy of LUCAS was demonstrated. A 55-year-old diabetic man undergoing peritoneal dialysis due to renal failure suddenly suffered a witnessed collapse in a nephrology ward. Two nephrologists started manual chest compressions and
ventilation with a self-inflating bag, after confirming that the patient had no palpable pulses and no spontaneous respiration. The CPR-team (hospital team consisting of one cardiologist assisted by one specially trained cardiology nurse and one anaesthesiologist assisted by one specially trained anaesthesiology nurse) was called and arrived after 4 min. The ECG showed asystole. The patient was intubated and the heavily built male cardiologist in the resuscitation team continued manual chest compressions. Atropine and adrenaline were given intravenously. After 9 min of standard CPR without signs of ROSC, the cardiologist agreed that LUCAS could be applied to the patient, as a last effort to save the patient’s life. The assistant nurse, who had been trained in the use of LUCAS-CPR, quickly applied the device and immediately after the start of LUCAS, strong pulses could be palpated. After 3 min of chest

Table 1

<table>
<thead>
<tr>
<th>Physiological variables in experiments comparing manual CPR with LUCAS-CPR (Group I)</th>
<th>Baseline values</th>
<th>Values after 5 min of CPR</th>
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<tr>
<td></td>
<td>Manual CPR</td>
<td>LUCAS-CPR</td>
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<tr>
<td>Aortic pressure (mmHg)</td>
<td>Mean 68 ± 2</td>
<td>77 ± 4</td>
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<tr>
<td></td>
<td>Systolic 90 ± 2</td>
<td>95 ± 3</td>
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<tr>
<td></td>
<td>Diastolic 56 ± 3</td>
<td>64 ± 4</td>
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<tr>
<td>Right atrial pressure (mmHg)</td>
<td>Mean 6 ± 1</td>
<td>7 ± 1</td>
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<tr>
<td></td>
<td>Systolic 9 ± 1</td>
<td>10 ± 1</td>
</tr>
<tr>
<td></td>
<td>Diastolic 6 ± 1</td>
<td>6 ± 1</td>
</tr>
<tr>
<td>Pulmonary arterial pressure (mmHg)</td>
<td>Mean 52 ± 3</td>
<td>58 ± 5</td>
</tr>
<tr>
<td></td>
<td>Systolic 2.9 ± 0.3</td>
<td>3.3 ± 0.4</td>
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<tr>
<td></td>
<td>(%) 100</td>
<td>100</td>
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<tr>
<td></td>
<td>End-tidal CO2 (%)</td>
<td>4.2 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>Carotid arterial blood flow (ml/min)</td>
<td>189 ± 24</td>
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<tr>
<td></td>
<td>(%) 100</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>Mixed venous blood gas (kPa)</td>
<td>PrO2 7.3 ± 0.6</td>
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<tr>
<td></td>
<td>(kPa)</td>
<td>PrCO2 5.6 ± 0.3</td>
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<tr>
<td></td>
<td>pH 7.40 ± 0.03</td>
<td>7.43 ± 0.02</td>
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<td></td>
<td>SvO2 80 ± 4</td>
<td>84 ± 5</td>
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<td>Arterial blood gas (kPa)</td>
<td>PaO2 54 ± 6</td>
<td>57 ± 3</td>
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<td></td>
<td>(kPa)</td>
<td>PaCO2 4.4 ± 0.2</td>
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<td>pH 7.48 ± 0.03</td>
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<td>(%)</td>
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Inspired oxygen fraction = 1.0, blood gas apparatus set on the pig mode.

Fig. 8. Typical pressure curves obtained in a 20 kg pig during manual CPR and during LUCAS-CPR. The area between the curves for intrathoracic aortic pressure and right atrial pressure gives a picture of the coronary perfusion pressure. Note the biphasic positive curves and greater area between the curves during LUCAS-CPR.

Fig. 9. The coronary perfusion pressure obtained during manual CPR vs. LUCAS-CPR in pigs (Group I). The coarse line shows the mean value. S.E.M. (thin line) is shown only on one side for the sake of clarity. n = 6 in both groups.
compressions/decompressions with LUCAS, the patient regained spontaneous circulation. He was transferred to the intensive care unit and was treated on a ventilator for 1 week. Blood cultures showed severe sepsis. After appropriate antibiotic therapy, the patient was weaned from the ventilator, recovered, and left the hospital. At a follow-up visit 1 year later, his mental capacity was fully intact.

Fig. 10. The pressure- and carotid flow curves in pigs with ROSC vs. pigs without ROSC (Group II). Data shown as mean ± S.E.M., n = 8 in each group. VF = induction of ventricular fibrillation. Def = defibrillation.

Fig. 11. The coronary perfusion pressure in pigs with ROSC vs. pigs without ROSC (Group II). Data shown as mean ± S.E.M., n = 8 in each group. S.E.M. is shown only on one side for the sake of clarity.

Fig. 12. End-tidal CO₂ in pigs with ROSC vs. pigs without ROSC (Group II). Data shown as mean ± S.E.M., n = 8 in each group. * P < 0.05.

Fig. 13. LUCAS-CPR during 1 h of ventricular fibrillation (VF) in normothermia. Temperature, systolic, mean and diastolic (SAP, MAP, DAP) intrathoracic aortic pressure, coronary perfusion pressure, and right carotid arterial blood flow are shown as mean ± S.E.M. n = 8 (n = 3 after defibrillation (Def)).
4. Discussion

The animal experiments in this study were performed and reported according to the Utstein guidelines for laboratory CPR research [7]. These recommend use of swine weighing 20–25 kg. The anteroposterior chest diameter of pigs this size will be similar to that of average sized adult humans. Our measurements of 65 adult humans confirmed this. Swine have the advantage of being uniform in size and shape at similar ages and weights and there are many similarities in metabolic and cardiovascular function between swine and humans.
The coronary vascular anatomy is also similar to that of humans, with the exception of the left azygos vein, which in the pig enters the coronary sinus rather than a precaval vein. An important difference is that in pigs, the ventricles are positioned in the center of the thoracic cavity, surrounded by lung tissue on all sides (see Fig. 5). During the compression phase of CPR, the ventricles of a pig is not compressed by the sternum and spine, but are compressed indirectly by the pressure increase inside the chest. This mechanism is known as the 'thoracic pump theory', in contrast to 'the heart pump theory', in which it is thought that chest compression causes a direct compression of the heart against the spine [5,10,11]. The circulation created by the chest compressions in our study was probably caused by a 'thoracic pump' rather than a 'heart pump' mechanism.

In the present study the ventilation was kept constant throughout, with the intention to use end-tidal CO₂ values as an indication of the efficiency of the chest compressions. Due to the reduced cardiac output during CPR, the animals were relatively hyperventilated, resulting in respiratory alkalosis that compensated for the metabolic acidosis that also developed during prolonged CPR (Table 3). Thus, we think that buffer therapy with this experimental design would not have added any benefits for the animals. The use of drugs to increase the coronary perfusion pressure might have raised the ROSC, but were excluded in order to be able to judge the efficacy of chest compressions/decompressions per se.

In a clinical study comparing manual compression with manual compression/decompression, the latter approach significantly improved long-term survival rates among patients who had cardiac arrest out-of-hospital [4]. What role did the active decompression play in our study? The suction cup of LUCAS was too wide (13.5 cm in diameter) to fit snugly with the precordial chest in the pigs used. The upper thorax is too narrow for real vacuum to be created during the compression phase. This was confirmed by the fact that no suction mark could be seen after CPR. However, in the pigs resuscitated for longer than 20 min, the thorax softened and became more flat. A vacuum was then created, and after the CPR, a suction mark was seen. In a pilot study elucidating the efficacy of manual compressions for 30 min using this pig model, the end-tidal CO₂ fell to zero after about 20 min of manual compressions. At that time the pig thorax had lost its elastic recoil, and the anteroposterior diameter had diminished significantly and no ROSC was obtained. We think that in such a situation active decompression may be of value, if thereby an increase in venous return can be accomplished. As long as the thorax is intact, with normal elastic recoil of the chest in each decompression phase, we think active decompression is of less importance. In the Group II experiments we observed that after about 15–20 min with CPR, the non-ROSC animals started to lose coronary perfusion pressure while the ROSC-pigs did not (Fig. 11). Paradis et al. measured the coronary perfusion pressure in 100 patients with cardiac arrest [12]. In their study 24 patients had ROSC. Initial coronary perfusion pressure was 1.6±8.5 mmHg in patients without ROSC and 13.4±8.5 mmHg in patients with ROSC, whereas the maximal coronary pressure measured was 8.4±10.0 mmHg in those without ROSC and 25.6±7.7 mmHg in those with ROSC. Only patients with maximal coronary perfusion pressures of 15 mmHg

<table>
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<tr>
<th>Table 3</th>
<th>Blood gas values in LUCAS-CPR pigs with normothermia vs. surface cooling during CPR and CPR in hypothermia (Group IV)</th>
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<tr>
<td></td>
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<tr>
<td></td>
<td><strong>End-tidal CO₂ (%)</strong></td>
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<td></td>
<td><strong>Base</strong></td>
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<tr>
<td>Normothermia</td>
<td>4.2±0.2</td>
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<tr>
<td>Surface cooling</td>
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<tr>
<td>Hypothermia</td>
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<tr>
<td>SvO₂ (%)</td>
<td><strong>Base excess-arterial</strong></td>
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<tr>
<td>Normothermia</td>
<td>84±3</td>
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<tr>
<td>Surface cooling</td>
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<td>Hypothermia</td>
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<td>pH-arterial</td>
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</tr>
<tr>
<td>Hypothermia</td>
<td>7.52±0.02</td>
</tr>
</tbody>
</table>

All baseline values were obtained at normothermia. All blood gas values were obtained with FiO₂ = 1.0; the blood gas apparatus was adjusted to measure at the same temperature as the pig and with the pig mode.
or more had ROSC. These data correspond well with those obtained on the ROSC and non-ROSC pigs in the present study, attesting to the relevance of this model.

End-tidal CO₂ levels reflect cardiac output during CPR. Levine and coworkers monitored end-tidal CO₂ during CPR in 150 consecutive victims of cardiac arrest out-of-hospital [13]. A 20-min end-tidal CO₂ value of 10 mmHg (1.3 kPa) or less was associated with a lack of (ROSC) in their study. In the Group I pigs in our study, manual CPR was able to produce end-tidal CO₂ values of 2 kPa, but the coronary perfusion pressure in those pigs was only around 10 mmHg, and no ROSC was obtained. Thus, in successful CPR, it is not enough to obtain a critical cardiac output (adequate end-tidal CO₂ values); an adequate coronary perfusion pressure is equally essential for ROSC.

Early defibrillation is the most important single factor to influence survival after sudden circulatory arrest, if it can be accomplished within 4 min, according to a study published by Cobb and coworkers [14]. In their study, survival improved if 90 s of external chest compressions were given prior to defibrillation in the group of patients where defibrillation could not be given within 4 min of circulatory arrest. In an experimental study by Sato and coworkers, they describe the adverse effects of interrupting chest compressions during CPR [15]. In their model, the coronary perfusion pressure during CPR was 26 ± 2 mmHg, but 10 s after interruption of CPR, it had decreased to 6 ± 3 mmHg, and after 20, 30 and 40 s it was 4 ± 2, 4 ± 4 and 4 ± 4 mmHg, respectively, i.e. close to zero. If defibrillation was done under ongoing CPR, the 24 h-survival rate was 80%. If it was delayed by 10 s, the 24 h survival rate was reduced to 40%, and if it was delayed by 20 s or more, there were no survivors. With these results in mind, it is easy to understand why defibrillation after 4 min of circulatory arrest is not likely to be successful. After that time, there has been minimal or no coronary circulation for at least 3 min. The advice given by Cobb et al. for routine provision of 90 s of CPR prior to a defibrillation seems most logical. In addition, defibrillation has greater chance of success if it can be delivered under ongoing CPR, as shown by Sato et al., i.e. with blood circulation through the heart muscle tissue [15]. Defibrillation during manual CPR cannot be done for safety reasons, but it would be one obvious advantage of mechanical CPR. The exterior of LUCAS is made of a non-conducting material, and by using electrode pads on the patient, defibrillations can be given safely during CPR.

As the Group IV study indicates, surface cooling as soon as possible after mechanical CPR is initiated may be of great advantage for several reasons. The coronary perfusion pressure increased promptly, probably due to redistribution of the blood volume and increased systemic vascular resistance. The metabolism will be reduced by about 6–7%/°C that the body temperature is lowered [16,17], with the consequence that less circulation will be needed to ensure an adequate organ perfusion. Hypothermia will also protect the brain [18–20].

Preliminary reports from the clinical pilot study with LUCAS are promising. It has been easy to handle, it can be applied to the patient within 10–20 s, it fits on stretchers, the suction cup helps to maintain a correct position and it fits and works well within ambulances. Defibrillation may be delivered during ongoing chest compressions. Several prospective randomized studies within or out-of-hospital are being planned. The most critical factor for successful CPR out-of-hospital is to initiate adequate chest compressions and oxygenation as quickly as possible after cardiac arrest, before the brain has been irreversibly injured. Traditional manual CPR will lose none of its importance with the introduction of mechanical CPR, quite the opposite. Knowing that a machine is under way to take over the chest compressions should only give the rescuer(s) added strength to maintain forceful manual CPR until the ambulance team arrives.

To conclude, gas-driven compressions and active decompressions with LUCAS give significantly better circulation during ventricular fibrillation compared to manual chest compressions.

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References


