Percutaneous left ventricular assist in ischemic cardiac arrest

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**Background:** Ischemic cardiac arrest represents a challenge for optimal emergency revascularization therapy. A percutaneous left ventricular assist device (LVAD) may be beneficial.

**Objective:** To determine the effect of a percutaneous LVAD during cardiac arrest without chest compressions and to assess the effect of fluid loading.

**Design:** Totally, 16 pigs randomized to either conventional or intensive fluid with LVAD support during ventricular fibrillation (VF).

**Setting:** Acute experimental trial with pigs under general anesthesia.

**Subjects:** Farm pigs of both sexes.

**Interventions:** After randomization for fluid infusion, VF was induced by balloon occlusion of the proximal left anterior descending artery. LVAD and fluid were started after VF had been induced.

**Measurements:** Brain, kidney, myocardial tissue perfusion, and cardiac index were measured with the microsphere injection technique at baseline, 3, and 15 minutes. Additional hemodynamic monitoring continued until 30 minutes.

**Conclusions:** During VF, a percutaneous LVAD may sustain vital organ perfusion. A potential clinical role of the device during cardiac arrest has yet to be established. (Crit Care Med 2009; 37: 1365–1372)

**Key Words:** AMI; cardiac arrest; resuscitation; PCI; LVAD; tissue perfusion

Acute myocardial ischemia is a major cause of cardiac arrest (1) and urgent percutaneous coronary intervention (PCI) may improve the outcome (2–4). Current external resuscitation methods (5–10) have a high mortality (11–16) and may complicate the revascularization procedure (17–19). Assist techniques including extracorporeal life support (20) and cardiopulmonary bypass (21) have previously been used with promising results on hemodynamics but are highly invasive, limiting their use in this critical setting. The Recover LP 2.5, a novel percutaneous left ventricular assist device (LVAD), has potential for improving pre-PCI and post-PCI hemodynamics (22, 23), and its use in high-risk PCI and cardiogenic shock has been encouraging (24, 25).

In the absence of myocardial contraction, blood flow through the pulmonary circulation depends on filling pressures and thoracic volume compression and decompression as in conventional cardiopulmonary resuscitation (26–28). In addition, sequential variation of thoracic volume and pressure during mechanical ventilation may facilitate blood flow toward the left side circulation (29, 30). Blood flow through the pulmonary vasculature may also be improved by increasing central venous pressure (31). Intravenous fluid administration can increase venous return and central venous pressures augmenting blood supply to the left ventricle (32).

The aim of this study was to investigate, in an experimental model, whether blood delivery to the systemic circulation could be achieved using a percutaneous LVAD during ventricular fibrillation (VF) without simultaneous chest compressions. We also hypothesized a possible beneficial effect of fluid loading to improve delivery to the pump from the right side of the heart and therefore examined the effects of an intensified fluid regimen in a randomized comparison with conventional fluid infusion.

**MATERIALS AND METHODS**

**Animals.** Sixteen pigs (Norwegian Land Race) of either sex with a mean weight of 47 kg (range 42–55 kg) were fasted overnight but had free access to water. The animals were acclimatized for at least 7 days under controlled temperature, lighting, and humidity and were fed with a standard diet. The experimental protocol is registered and approved by the Norwegian Animal Research Authority and by the local responsible laboratory animal veterinarian, and it was conducted in accordance with national and international laws controlling experiments in live animals. A single dose of 330-mg acetylsalicylic acid was administered orally the day before the procedure to reduce the risk of coronary thrombus formation during intracoronary instrumentation.

**Anesthesia and Preparation.** After premedication with ketamine and atropine (20 mg/kg and 1 mg) IM in the neck, two ear veins were...
cannulated. Animals were placed on a warm-water blanket with continuous monitoring of rectal temperature and electrocardiogram. Ventilation (spontaneous on mask) with O2 and 3% (vaporizer setting) isoflurane (Rhodia, Bristol, United Kingdom) for 2–3 minutes allowed oral intubation. Ventilation was commenced and continued throughout the experiment with a mixture of N2O (56% to 57%) and oxygen. The mechanical ventilator (Cato M32000, Drägerwerk, Lübeck, Germany) was set to a tidal volume of 10 mL/kg and a frequency of 13–15 cycles/min; with small adjustments aiming at an end-tidal CO2 of 5%.

Anesthesia was induced by intravenous loading doses of fentanyl 0.02 mg·kg⁻¹, midazolam 0.3 mg·kg⁻¹, pancuronium 0.063 mg·kg⁻¹, and sodium pentobarbital 15 mg·kg⁻¹ and maintained with continuous infusions of fentanyl 0.02 mg·kg⁻¹·h⁻¹, midazolam 0.3 mg·kg⁻¹·h⁻¹, pancuronium 0.14 mg·kg⁻¹·h⁻¹, and pentobarbital 4 mg·kg⁻¹·h⁻¹, thus resulting in a fluid substitution of 15 mL·kg⁻¹·h⁻³ during anesthesia. Details about evaluation and the applicability of this anesthetic protocol can be found elsewhere (33).

After infiltrating the skin with 0.5% xylocaine, the femoral arteries and veins were exposed bilaterally and secured by ligatures. Although the device is provided percutaneously in humans, we used surgical cut down to optimize access site control due to anatomical features in the pig and also because of the technically complicated nature of the protocol. Arterial (13 French [F]), 6F, 5F) and venous (8F) sheaths were inserted. Arterial sampling was performed via microguide sheaths and repeated every 60 minutes for the duration of the study. A 5F-pigtail catheter was placed in the left ventricle for injection of microspheres. A 6F multipurpose hockey stick catheter served as a guide for the left coronary artery. Coronary blood velocity was measured with a coronary Doppler wire in the left circumflex artery (LCX). Right side pressures were measured with a Swan Ganz catheter in the pulmonary artery. The Recover (Abiomed, Aachen, Germany) was implanted with the inlet below and the outlet above the aortic valve (Fig. 1, a and b). Aortic pressure and pump output in liters per minute was recorded from the device module. End-tidal CO2 is a valuable tool for evaluating circulatory function in resuscitation (34–38) and was monitored from the mechanical ventilator system. Samples for arterial acid–base measurements were taken from the arterial sheaths.

**Experimental Protocol.** Animals were randomized into two groups by blinded draw. After a short period of stabilization, registrations of baseline hemodynamics and baseline microspheres injection were performed during spontaneous cardiac function. Then, a standard PCI balloon was inflated in the proximal left anterior descending artery (LAD) inducing ischemic cardiac arrest. The balloon remained inflated during the study period. The conventional fluid group received fluid as required for the anesthetic protocol only (15 mL·kg⁻¹·h⁻¹). The intensified fluid group received additional Ringers acetate 1.5 L·min⁻¹ during the 30-minute trial corresponding to 2.7 liters per 30 minutes for a 70-kg human.

Two more microsphere injections were made after 3 and 15 minutes of VF with LVAD-assisted circulation. The microsphere flows were determined during 3 minutes (i.e., from 3 to 6 minutes of VF and 15 to 18 minutes of VF).

Hemodynamic variables and end-tidal CO2 were noted at 1, 3, 10, 15, 20, and 30 minutes. The study was terminated when pump function could no longer be sustained or after 30 minutes. At the end of the experiment, the animals were euthanized by exsanguination, still in general anesthesia. A schematic presentation of the protocol (flow chart) is shown in Figure 1.

**Regional Tissue Blood Flow and Cardiac Output.** The colored microspheres (Dye-Trak VII+, Triton Technology, San Diego, CA) were injected into the left ventricle during a period of 60 seconds. Using a constant rate extraction pump, a reference blood sample was drawn from the right femoral artery for 3 minutes after the start of injections. Four different colors were used in a randomized sequence. Microspheres’ data from one animal in each group were excluded because of technical failure (reference catheter cloting). Tissue samples were collected from the right and left kidney cortex, the cerebral cortex, right ventricle, and from the LCX and LAD regions of the left ventricle. The left ventricular samples were divided into subendocardial and subepicardial halves. Tissue samples and reference blood were weighed, prepared, and tissue blood flow rate and cardiac output calculated as milliliters per minute per gram and liters per minute, respectively (39).

The **Recover LP 2.5.** This is a minimally invasive ventricular assist device designed to be inserted percutaneously via the femoral artery (Fig. 2, A and B). The diameter is 4 mm (12F). Up to 2.5 L/min of blood can be delivered with a maximal rotational speed of 55,000 rounds per minute. The pump is advanced by means of a 0.14’ guide wire under fluoroscopy. The distal end is pigtail shaped and deployed in the apex of the left ventricle. Outflow is located in the proximal ascending aorta. A pressure sensor is used to verify correct placement of outflow in the aorta. Pump (LVAD) output was measured in liter per minute or as total output (L) during 39 minutes or to cessation of pump function. Chest compressions were not performed at any time during this experiment.

**Statistical Analysis.** Results are presented as mean ± s.e. Two-sample Student’s t tests were used for between-groups comparisons. Area under the curve analysis was performed for difference in total pump output between the groups during the 30-minute study period. Exact chi-square tests were used to compare proportions of animals completing the 30-minute protocol. The difference in pump out-

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**Figure 1. Protocol flow chart. LAD, left anterior descending artery; VF, ventricular fibrillation.**

**Figure 2. A. The Recover LP 2.5. B. The Recover LP 2.5 in situ, fluoroscopic image. a. Soft pigtail-shaped tip deployed in apex of left ventricle; b, inlet deployed in left ventricle; c, outlet above aortic cusps; 1, coronary wire in the left anterior descending artery; 2, pigtail catheter in left ventricle; 3, Swan-Ganz in pulmonary artery; 4, coronary catheter in left main stem; 5, flow wire in the left circumflex artery; 6, left ventricular assist device, coronary, and pigtail catheter shafts in descending aorta.**
RESULTS

Cardiac Index and Tissue Perfusion. No substantial differences between the intensive fluid and the conventional fluid groups could be demonstrated at baseline for the general hemodynamic variables (Table 1) or tissue blood flow rates. The mean time from balloon occlusion of the LAD before start of VF was 11 minutes with a range of 1–20 minutes. Cardiac index measured with microspheres did not differ between the groups at any time point. Baseline values averaged 4.2 ± 1.4 (sd) L·min⁻¹·m⁻² for the 14 subjects with completed data from the microsphere measurements (Fig. 3). Three minutes after onset of VF and start of LVAD, the corresponding value was 1.2 ± 0.6 L·min⁻¹·m⁻² (28% of baseline; p < 0.05), and after 15 minutes, cardiac index averaged 1.2 ± 0.7 L·min⁻¹·m⁻² (28% of baseline).

No differences in blood flow rate could be demonstrated between the groups at 3 or 15 minutes, either in cerebral tissue or kidneys or in the different regions of the myocardium. Tissue blood flow rate in cerebral tissue averaged 0.42 ± 0.11 mL·min⁻¹·g⁻¹ at baseline declining to 0.28 ± 0.18 mL·min⁻¹·g⁻¹ (65% of baseline) at 3 minutes (p < 0.05) and 0.24 ± 0.16 mL·min⁻¹·g⁻¹ (57% of baseline) at 15 minutes of VF with LVAD-assisted circulation. A very low tissue flow rate found in the myocardium supplied by LAD was consistent with the angioplasty balloon occlusion. Blood flow in the region supplied from the epicardial LCX region at baseline was 0.96 ± 0.39 mL·min⁻¹·g⁻¹ and declined significantly to 0.71 ± 0.40 mL·min⁻¹·g⁻¹ (74% of baseline, p < 0.005) at 3 minutes and 0.56 ± 0.40 mL·min⁻¹·g⁻¹ (58% of baseline) at 15 minutes. Renal blood flow rate deteriorated to 14% of baseline at 3 minutes (p < 0.05) and further to only 9% at 15 minutes (Fig. 3).

Effects of Fluid Loading. All animals in the intensified fluid group completed the study period maintaining assisted circulation during the 30 minutes study period. Only three of the eight animals in the conventional fluid group reached 30 minutes, resulting in a significant difference between the groups for this prespecified end point (p = 0.026). The intensified fluid group maintained a better performance and achieved significantly higher total pump output, measured as area under the curve, 49.3 ± 15.0 vs. 24.4 ± 14.1 L (p = 0.004). Figure 4 shows the changes in mean and individual pump output in the two groups. The overall decline was significantly lower with intensified fluid regimen (p = 0.002 for interaction). Pump output was significantly higher in this group compared with the conventional fluid supplement group at 15, 20, and 30 minutes (p ≤ 0.013). As a sensitivity analysis, the model was rerun with pump output equal to zero when pump output was not measurable. In this analysis, there was a swifter decline in the minimal fluid supplement group from 1 minute to 10–20 minutes after VF, resulting in somewhat larger group differences in this period of time. The output in the intensified fluid supplement group was virtually unchanged in this model.

Additional Hemodynamics. The end-tidal CO₂ and mean aortic pressure declined after induction of VF with the larg-
indicating no significant blood loss or hemoglobin concentration was seen from groups. No significant change in blood pressures no statistically significant differences were seen between the two groups, and further to 30 minutes in the intensified fluid group. Doppler blood flow velocity in the LCX and pulmonary pressure also decreased (p \leq 0.008), with no significant group differences (p \geq 0.319 for interaction) (Fig. 5). Thus, for end-tidal CO\(_2\), mean aortic pressure, LCX flow, and pulmonary artery pressures no statistically significant differences were seen between the two groups. For arterial pH, the decline was significant from baseline to 30 minutes (p \leq 0.005 in both groups); values did not differ between the groups at 30 minutes (7.43 \pm 0.01 to 7.20 \pm 0.01 in the conservative group and 7.45 \pm 0.02 to 7.24 \pm 0.20 in the intensified fluid group).

Table 2 shows the results from arterial and venous blood gas analysis. No significant differences were seen between the groups. No significant change in blood hemoglobin concentration was seen from baseline to study end in either group indicating no significant blood loss or hemodilution. Table 3 shows systemic oxygen delivery and consumption data, and also cerebral and myocardial oxygen delivery, calculated from microspheres flow measurements.

**DISCUSSION**

To our knowledge, there are no data regarding the use of percutaneous LVADs in the setting of ischemic cardiac arrest either in humans or in animal subjects. Our protocol was performed in a closed chest model with mechanical ventilation and a percutaneous LVAD with or without fluid loading. This study indicates that vital organ perfusion can be maintained although at a reduced level during acute ischemic cardiac arrest using a percutaneous LVAD.

In severe heart failure, surgical LVADs have previously been shown to improve hemodynamics, reduce symptoms, and also enhance the survival over time (41–43). Continuous flow devices have been used and results have been favorable (44) suggesting that pulsatile flow is not a requirement for maintaining long-term tissue blood supply and metabolism with low cardiac output. Various other approaches have been used for improving outcomes in the setting of cardiac arrest. Cardiopulmonary bypass (21), extracorporeal membrane oxygenation, extracorporeal life support (20), and open chest cardiac massage (45) have been investigated and results have indicated possible clinical potential. However, these methods are technically complicated; some require major surgical intervention and thus are less suited for rapid use in a critical situation.

In contrast, even though the Recover LP 2.5 requires specialized personnel and equipment, and therefore is not suitable for out-of-hospital use, this device can rapidly be advanced to the left ventricle via a 12F femoral artery sheath. During ongoing manual resuscitation, deployment would be expected to be more complicated than in a stable clinical setting. In particular, a short interruption of CPR may be necessary for optimal control when introducing the device into the ventricle. However, the device can be rapidly delivered into the left ventricle over a guidewire during CPR, and clinically relevant interruption of chest compressions during deployment is not likely to be required. The time for preparation of the console and device before implantation is within the range of few minutes, well within the time for transfer of patients with cardiac arrest to the cath lab.

External mechanical compression–decompression devices have been shown to improve survival when compared with manual CPR in pigs, and there have been reports of effective use in humans (46, 47). In these studies, changes in intrathoracic pressure created by external compression and those resulting from mechanical ventilation are considered important for maintaining systemic output. The filling of the left ventricle in cardiac arrest is affected by right side filling pressures and may be improved by IV fluid supplementation, and in addition, thoracic volume inflation/deflation during ventilation supports blood flow through the pulmonary vasculature (26–32). Fluid supplementation did have an effect in our study; although all animals receiving intensified fluid maintained pump output from 15 to 30 minutes, only three animals in the conventional group reached 30 minutes. The declining circulation over time also in the IV fluid supplement group in our study indicates that...
blood supply from the right to the left deteriorates over time in VF. In a clinical situation, some patients with chronic pulmonary disease and secondary pulmonary hypertension, or acidosis and hypoxia in the severely ill, could in theory have a reduced therapeutic effect of the device. High oxygen delivery from the ventilator system, and correction of acidosis and pulmonary vasodilators like nitric oxide and iloprost could have therapeutic potential and may be a focus for further studies.

Surgically implanted right ventricular assist devices may improve hemodynamics compared with LVAD alone in heart failure (48, 49). A percutaneous right ventricular assist device (right ventricular assist device) may also be helpful in this situation. Such a device is, to our knowledge, currently under development but not yet available for use. A percutaneous right ventricular assist device could improve filling from the right and could be of benefit compared with the intensive fluid loading used in our protocol. High pressure mechanical ventilator therapy may make fluid loading feasible in a clinical setting, but the regimen used could have a detrimental effect if used for a longer duration. An effective right ventricular assist device could in theory resolve this issue. Currently, only short-term application of the percutaneous LVAD may be practically feasible during VF.

Coronary and cerebral perfusion and end-tidal CO₂ levels are useful in assessing the prognostic effect of hemodynamic and respiratory interventions in cardiac arrest. Microsphere injection is a recognized method for measuring cardiac out-

Table 2. Arterial and venous blood gas analysis before and during ventricular fibrillation (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>3 Min</th>
<th>15 Min</th>
<th>30 Min</th>
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<tbody>
<tr>
<td><strong>Arterial SO₂, %</strong></td>
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<tr>
<td>Intensified</td>
<td>99.9 (0.1)</td>
<td>97.9 (2.2)</td>
<td>88.2 (5.3)</td>
<td>80.3 (18.8)</td>
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<tr>
<td>Conventional</td>
<td>99.8 (0.4)</td>
<td>87.8 (13.7)</td>
<td>99.4 (—)</td>
<td>16.0 (1.6)</td>
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<td><strong>Arterial HCO₃, kPa</strong></td>
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<tr>
<td>Intensified</td>
<td>28.2 (2.2)</td>
<td>22.8 (3.4)</td>
<td>17.0 (3.9)</td>
<td>22.0 (—)</td>
</tr>
<tr>
<td>Conventional</td>
<td>28.2 (1.8)</td>
<td>25.1 (2.8)</td>
<td>21.9 (3.3)</td>
<td>11.4 (10.6)</td>
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<tr>
<td><strong>Arterial PO₂, kPa</strong></td>
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<tr>
<td>Intensified</td>
<td>38.7 (8.3)</td>
<td>14.4 (12.0)</td>
<td>10.8 (5.3)</td>
<td>2.2 (—)</td>
</tr>
<tr>
<td>Conventional</td>
<td>40.9 (10.8)</td>
<td>17.0 (15.8)</td>
<td>15.4 (1.0)</td>
<td>11.4 (10.6)</td>
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<tr>
<td><strong>Arterial PCO₂, kPa</strong></td>
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<tr>
<td>Intensified</td>
<td>5.6 (0.4)</td>
<td>5.8 (3.1)</td>
<td>6.7 (4.5)</td>
<td>7.9 (4.8)</td>
</tr>
<tr>
<td>Conventional</td>
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<td>4.6 (1.6)</td>
<td>5.4 (2.8)</td>
<td>14.2 (—)</td>
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<td><strong>Arterial pH</strong></td>
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<tr>
<td>Intensified</td>
<td>7.44 (0.04)</td>
<td>7.36 (0.18)</td>
<td>7.18 (0.23)</td>
<td>7.15 (0.24)</td>
</tr>
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<td>Conventional</td>
<td>7.47 (0.04)</td>
<td>7.49 (0.13)</td>
<td>7.42 (0.22)</td>
<td>7.6 (—)</td>
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<td><strong>Venous SO₂, %</strong></td>
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<tr>
<td>Intensified</td>
<td>78.3 (3.8)</td>
<td>68.0 (—)</td>
<td>76.0 (—)</td>
<td>72.0 (—)</td>
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<tr>
<td>Conventional</td>
<td>77.2 (8.7)</td>
<td>65.0 (—)</td>
<td>65.0 (—)</td>
<td>65.0 (—)</td>
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<tr>
<td><strong>Venous HCO₃, kPa</strong></td>
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<tr>
<td>Intensified</td>
<td>29.4 (3.1)</td>
<td>21.7 (4.7)</td>
<td>19.5 (6.3)</td>
<td>15.0 (6.8)</td>
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<td>29.7 (1.7)</td>
<td>27.3 (1.4)</td>
<td>25.0 (2.3)</td>
<td>25.0 (2.3)</td>
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<tr>
<td><strong>Venous PO₂, kPa</strong></td>
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<tr>
<td>Intensified</td>
<td>5.0 (0.5)</td>
<td>2.5 (0.6)</td>
<td>2.6 (0.4)</td>
<td>2.5 (1.1)</td>
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<tr>
<td>Conventional</td>
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<td>2.2 (0.6)</td>
<td>2.1 (0.2)</td>
<td>2.1 (0.2)</td>
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<tr>
<td><strong>Venous PCO₂, kPa</strong></td>
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<tr>
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<td>6.9 (0.4)</td>
<td>8.4 (4.0)</td>
<td>8.7 (4.3)</td>
<td>7.5 (2.0)</td>
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<tr>
<td>Conventional</td>
<td>6.7 (0.4)</td>
<td>6.6 (0.9)</td>
<td>7.8 (1.1)</td>
<td>7.8 (1.1)</td>
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<tr>
<td><strong>Venous pH</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Intensified</td>
<td>7.38 (0.04)</td>
<td>7.20 (0.15)</td>
<td>7.14 (0.20)</td>
<td>7.03 (0.24)</td>
</tr>
<tr>
<td>Conventional</td>
<td>7.40 (0.04)</td>
<td>7.37 (0.06)</td>
<td>7.27 (0.04)</td>
<td>7.27 (0.04)</td>
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Figure 5. Vital parameters as mean values for each group during the study period. p = not significant between the groups. n = 8 at all times in the intensive fluid group. In the conventional fluid group, n = 7 at 1, 3, 10 and 15 minutes, n = 4 at 20 minutes, and n = 3 at 30 minutes.
put and tissue perfusion and has been previously used in cardiac arrest (7, 39, 50). Thermodilution was not performed during the study as the infusion of high-dose IV saline would be expected to cause temperature differences in particular between the groups and thereby make assessment of cardiac output unreliable. Experience from pilot studies indicated that stable pump function measurements could not be predictably maintained in a low output state in a setting with cardiac arrest. On this background, a pump delivery during VF of less than 30% of the output recorded immediately after cardiac arrest was chosen as a cut-off point.

In this study, flow measured by microspheres to the cerebral and cardiac tissues was sustained at 65% and 74%, respectively, of the baseline values, whereas total output rapidly decreased to 28% of baseline, suggesting autoregulation in vital organs. An early reference study of regional blood flow during CPR using microspheres in dogs reported flow to brain at 90%, heart at 35%, and cardiac output at 27% compared with baseline after 5 minutes of CPR (50). Another study compared hemodynamic effects of a novel active compression–decompression device with manual compression in pigs reporting superior results with the device. After 5 minutes of VF, flow in the carotid artery was 58 mL/min compared with 201 mL/min at baseline (29%), and at similar time points, the coronary perfusion pressure was 17 vs. 58 mm Hg (29%) and cardiac output was 0.9 vs. 3.3 L (27%).

End-tidal CO₂ was 2.0% with manual compression and 2.8% with the device at 5 minutes compared with 4.2 and 4.1 at baseline (46). Our findings correspond well with these findings although different models have been used and direct comparison to our study is not feasible. In general, the relative changes in vital organ perfusion, hemodynamics, and end-tidal CO₂ levels during cardiac arrest seen in our study are numerically reasonably in line with other available data. A direct comparison with standard cardiopulmonary resuscitation was not an objective of this study, and therefore we cannot infer any clinical implications from our results alone.

Severe renal hypoperfusion was seen in this study as has been reported previously with cardiopulmonary resuscitation (50). This has been shown not to be associated with adverse outcomes after cardiac arrest in the absence of preexisting renal failure (51). The drop in pH was not severe in either group and although this may reflect the effectiveness of the device, it may be related to the relatively short ischemic time (30 minutes or less) and the ability of skeletal muscle to compensate for acid–base disturbances in this time frame (52).

Effects of the Device on Different Clinical Parameters Need Further Investigation. A protocol including subjects with chronic ischemic heart failure and induced VF could be useful for further determining the possible clinical role of the LVAD. The current model for persistent VF is supported by an experimental study that indicates chronic ischemic heart failure does not reduce the chance of successful defibrillation but primarily affects outcomes with regard to postresuscitation cardiac function (53). Other studies have suggested that ischemically induced cardiac arrest with LAD occlusion (54) or stenosis (55) are clinically relevant and that persistent ischemia due to impaired LAD flow markedly reduces the success rate of defibrillation compared with other mechanisms for VF and thus increases the risk of persistent VF. A model with persistent severe LAD ischemia thus seems valid for the setting of persistent VF.

Limitations. The use of an LVAD in cardiac arrest over a longer period of time if revascularization has not restored sinus rhythm broaches ethical issues similar to other resuscitation methods. The effect on clinically important outcomes such as return of spontaneous circulation, functional status, or even survival is unknown, and the effect of the device on postresuscitation clinical end points should be the focus on further studies. We are unaware of similar interventions in earlier studies of cardiac arrest.

Anesthesia included the use of neuromuscular blockade as this was deemed relevant in a setting of critical cerebral ischemia; furthermore, this protocol has been evaluated and used in our institution (33) and is considered safe and predictable in hypothermia, which may be relevant for further studies of cardiac arrest.

Intensive fluid loading is potentially deleterious in patients with severely compromised left ventricular function because of acute myocardial infarction. During the first 15 minutes of VF, there was no obvious advantage of the intensive fluid regimen. Based on the steady fall in cardiac output, it is a reasonable interpretation that gradually less blood volume passed through the lungs to the left ventricle during the study period. Intensive fluids prolonged effective pumping of the Impella, but interpretation of any favorable hemodynamic effect of fluids after 15 minutes is hampered by the low number of survivors in the control group because measurements were done at fixed time points and not at arbitrary time points before an end point was reached. Even if fluid loading might have less detrimental effects during mechanical ventilation, the regimen used needs further evaluation before fluid loading can be applied clinically in the setting of cardiac arrest.

**CONCLUSION**

Our results demonstrate the potential of a percutaneous LVAD to sustain perfusion to the brain and myocardium during ischemic cardiac arrest in a porcine model. Further assessment and validation of the findings are required before conclusions with regard to clinical applications can be made.
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