Predictors of neurologic outcome after successful extracorporeal cardiopulmonary resuscitation

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ABSTRACT

Background: Extracorporeal cardiopulmonary resuscitation (ECPR) refers to use of the extracorporeal membrane oxygenation (ECMO) in cardiopulmonary arrest. Although ECPR can increase survival rate of cardiac arrest, it may also result in poor post-resuscitation neurologic status. Thus, we investigated predictors of good neurologic outcome after successful ECPR.

Methods: A total of 227 patients underwent ECPR from May 2004 to June 2013 at Samsung Medical Center. Successful ECPR was defined when a patient was alive over 24 hours after ECPR. Neurologic outcome was assessed at discharge using the Glasgow-Pittsburgh Cerebral Performance Categories scale (CPC). CPC 1 and 2 were classified as good, CPC 3 to 5 were classified as poor neurological outcomes. Patients whose neurologic status could not be assessed at discharge were excluded.

Results: One hundred fifteen patients were included. Mean age was 58 (45-66) years. Eighty patients (70%) were male. Cardiopulmonary resuscitation (CPR) was performed at non-
hospital sites for 19 (17%) patients. Bystander CPR was performed in 9 of 19 cases (47%); cardiac etiology was verified in 74 (64%) patients. Therapeutic hypothermia was performed in 9 patients (8%). Sixty-eight patients (59%) had good neurologic outcome, and 47 (41%) did not. Multivariate analysis revealed neurologic outcome was affected by hemoglobin level before ECMO ($P = 0.02$), serum lactic acid ($P < 0.001$) before ECMO insertion, and cardiac arrest to ECMO interval ($P = 0.04$).

**Conclusions:** Low hemoglobin level, high serum lactic acid before ECMO, and prolonged cardiac arrest to ECMO interval predicted poor neurologic outcome after successful ECPR. Early institution of ECMO and low threshold for blood transfusion may improve neurologic outcomes for patients who survive ECPR.

**Keywords:** Extracorporeal membrane oxygenation, Extracorporeal life support, Cardiopulmonary resuscitation, Cardiac arrest

**Background**

Extracorporeal membrane oxygenation (ECMO) is a useful intervention for refractory cardiogenic shock and respiratory failure [1, 2]. Because ECMO implementation can rapidly normalize circulation in patients under cardiac arrest, it has been used to assist cardiopulmonary resuscitation (CPR) in various situations [3-6]. Using traditional chest compression is less effective than using ECMO (extracorporeal CPR, ECPR) in CPR. ECPR has been associated with greater recovery of spontaneous circulation (ROSC). ECMO implementation takes at least several minutes when cardiopulmonary arrest is persistent
despite conventional CPR. Since brain is the most vulnerable organ to hypoxia and inadequate perfusion, ECPR can produce some patients with severe neurologic deficits if prompt ECMO implementation is not performed [7]. Beside delayed ECPR, several factors may lead to poor neurologic outcomes after ECPR. It is highly significant to achieve good neurologic outcomes as well as successful resuscitation. We investigated predictors for favorable neurologic outcome rather than survival.

Methods

This was a retrospective study performed in a cohort of patients underwent ECPR during hospitalization in Samsung Medical Center (a 1961-bed, university-affiliated, tertiary referral hospital in Seoul, South Korea). This study was performed retrospectively using a coded database, so we did not need to collect informed consent about this study. The study was approved by the Institutional Review Board of Samsung Medical Center (IRB No. SMC 2014-03-174) according to the Declaration of Helsinki to review and publish information from the patients' records. Informed consent was waived because of the retrospective nature of the study.

Patients

From May 2004 to June 2013, 227 patients underwent ECPR at Samsung Medical Center. ECPR was defined as use of venoarterial ECMO intended to treat cardiac arrest. Successful ECPR was defined when a patient was alive over 24 hours after ECPR. Some
patients never be awaken and spontaneous awakening trial was not possible. They had needed continuous sedation because of hemodynamic instability. Some patients were expired due to non-neurologic causes such as multiorgan failure, cardiac death, or uncontrolled infection. We excluded those patients were not possible to define neurologic status, because of continuous sedation and died due to unknown causes and another causes beside brain death. Thus, ninety patients were excluded and 115 patients were included in this study (Figure 1).

Endpoints and definitions

Our primary endpoint was neurologic outcome at hospital discharge. Neurologic outcome was assessed with the Glasgow-Pittsburgh Cerebral Performance Categories scale (CPC) (1 to 5 as shown in Table 1) [8]. CPC 1 and 2 were classified as good neurological outcomes, whereas CPC 3, 4, and 5 were considered poor neurological outcomes. We thoroughly reviewed the medical records, and patients were assigned to the CPC scale when two authors (JAR and YHC) agreed on definite CPC scales. ECPR was defined when ECMO was performed during CPR. CPR duration was defined as time from chest compression onset to offset. ROSC is defined for all rhythms as the restoration of a spontaneous perfusing rhythm that results in more than an occasional gasp, fleeting palpated pulse, or arterial waveform. Cardiac arrest to ECMO interval was defined as time from collapse to the point of ECMO setup and administration. The ROSC time was defined as the sum of each time of the ROSC before ECMO running.

The ECMO team consisted of cardiovascular surgeons, cardiologists, intensivists, special nurses, and perfusionists. Percutaneous cannulation was established in the femoral vein and artery by Seldinger’s technique. When it was difficult to puncture the femoral artery
percutaneously, femoral cut down procedures were performed. Our ECMO flow has been described in the previous report [6]. The institutional rapid response team called the on-call ECMO team leader when CPR was performed for more than 10 minutes, or when there were unstable vital signs or recurrent cardiac arrest. The ECMO team leader and CPR leader assessed the patient and made a decision on the ECPR. ECPR was performed when cardiopulmonary arrest is persistent despite conventional CPR over 10 minutes, witness arrest is confirmed, and the event caused the arrest is thought to be reversible. Generally ECPR was performed when there was a high probability of reversible cardiac etiology, or a respiratory cause for cardiac arrest. ECPR was deferred in cases of short life expectancy (< 6 months), uncontrolled terminal malignancy, unwitnessed collapse, limited activity, unprotected airway, and CPR time more than 60 minutes at initial contact. Age itself was not a contraindication for ECPR.

The Capiox Emergency Bypass System (Terumo, Tokyo, Japan) or Prolonged Life Support system (Maquet Cardiopulmonary AG, Hirrlingen, Germany) were used in all cases. Priming was performed with a crystalloid solution, such as normal saline or balanced solution. No patients in the study had blood primed ECMO. Anticoagulation was accomplished by unfractionated heparin bolus injection followed by continuous intravenous heparin infusion to maintain an activated clotting time between 150 and 200 seconds.

Statistical analysis

Continuous variables were expressed as medians and interquartile ranges (IQRs). Chi-square or Fisher’s Exact test were used as appropriate for categorical data, and the Mann–Whitney U test was used for continuous data. A multivariate logistic regression model
was used to identify predictors for neurologic outcome. All variables associated with neurological outcome were analyzed with a univariate analysis. Factors with \( P \)-value < 0.2 considered clinically relevant were included in a multivariate analysis. SPSS 20.0 statistical software (SPSS Inc., Chicago, IL) was used for statistical analysis. A \( P \)-value of less than 0.05 was considered statistically significant.

**Results**

**Patients’ characteristics**

Patient mean age was 58 (45-66) years, and 80 patients (70%) were male. Cardiac arrest to ECMO interval was 34 (20-53) minutes. Cardiac etiology was verified in 74 (64%) patients. Acute myocardial infarction was the cardiac arrest cause in 40 patients (35%). Thirty-eight patients (33%) had a history of ischemic heart disease. Respiratory etiology was verified in 5 patients (4%). Most patients (83%) had cardiac arrest in the hospital. Only a few patients (17%) who had cardiac arrest in a non-hospital setting had ECMO. Patient characteristics are shown in Table 2.

**ECPR and neurologic outcome**

Sixty eight patients (59%) had good neurologic outcome, but 47 patients (41%) did not (Figure 2). Therapeutic hypothermia was performed in 10 patients (5%). ECMO support
mean duration was 47.5 (18.5-101) hours. Total ICU stay was 11 (7-22.5) days. The 68 patients (59%) had good neurologic outcome and the 47 patients (41%) did not (Figure 2). The univariate analysis showed no differences between good and poor neurologic groups on age, comorbidities, bystander CPR, therapeutic hypothermia, total bilirubin, creatinine, 24 hour lactic acid clearance, ROSC before ECMO, and ROSC time (Table 2).

Multivariate analysis revealed neurologic outcome was affected by hemoglobin level, serum lactic acid before ECMO insertion, and cardiac arrest to ECMO interval (Figure 3). However, age, gender, out of hospital cardiac arrest, hemoglobin level after ECMO, acute coronary syndrome, initial shockable rhythm, and CPR duration were not independent predictors of neurologic outcome (Table 3).

**Discussion**

Ninety patients were excluded whose neurologic outcome could not be assessed or died due to unknown causes or another cause beside brain death. A few patients’ neurologic status could not be assessed because of poor medical record. Death due to unknown causes or another cause beside brain death was not possible to define as CPC 5 in this study, because this study was about ECPR and neurologic outcome rather than morbidity and mortality. Thus, 115 patients were included in this study.

Chest compression results in a cardiac output of 25–30% of normal when performed under optimal conditions [4]. Prolonged CPR duration is related to low ROSC chance and
cerebral damage [9]. However, ECMO can rapidly normalize both blood flow and oxygenation. If the procedure is successful, ECMO can effectively protect major organs and often reverse the underlying cause of cardiac arrest [4, 5, 10, 11].

However, there are two major limitations of ECPR. One is the need for anticoagulation. ECPR should not be performed in patients with contraindications of anticoagulation, such as intracranial bleeding. The other is limited availability and time for preparation and insertion. Thus, devices must be kept ready for use. Leaders should be familiar with both surgical and catheter techniques. The ECMO procedure in patients undergoing CPR takes longer and carries higher procedural failure and complications risks. A long time lag between cardiac arrest and ECMO “pump on” may be tolerable for many major organs except brain, which is most sensitive to hypoxia. Thus, poor neurologic outcomes with preserved function of other vital organs can result from ECPR. Liberal use of ECMO as a CPR method carries the risk of increasing the number of patients who have severe neurologic deficits requiring large amounts of familial and social resources. Although several factors, including initial rhythm, resuscitation duration, underlying causes, and initial resuscitative effort are associated with both survival and neurologic outcomes after ECPR [11], we focused on favorable neurologic outcome predictors after successful ECPR.

Hemoglobin level before ECMO insertion was an important factor for neurologic outcome after successful ECPR. However, we could not find any studies that investigated the relationship between hemoglobin level and neurologic outcome after ECPR. Initial hemoglobin level was reported as important for neurologic outcome after conventional CPR. The SOS-KANTO study group showed higher hemoglobin level at hospital arrival was associated with favorable short-term neurologic outcomes among post-cardiac arrest patients [12]. Previous animal studies also showed that the penumbral brain is much more vulnerable
than the normal brain, with oxygen delivery and cerebral metabolic rate progressively declining as hemoglobin concentration decreased [13, 14]. All these mechanisms appear to be physiologic neuro-protective mechanisms against ischemia caused by anemia. In the case of ECPR, most patients had prolonged cardiac arrest with bolus infusion of crystalloid and colloid fluids. Because the crystalloid solution for ECMO priming dilutes the blood, hemoglobin level can be decreased acutely. Efforts to correct anemia before or during ECMO administration may enhance oxygen delivery, and be neuro-protective.

Lactic acid is a useful marker of tissue hypoxia. Lactic acid level and its clearance are associated with mortality and neurologic outcome after CPR [15]. Kliegel et al. showed that persistent hyperlactemia for 48 hours predicted mortality and a poor neurologic outcome after conventional CPR [16]. There was a significant correlation between lactic acid level and cardiac arrest to ECMO interval [17]. Sawamoto et al. reported there was a significant difference in serum lactic acid level between patients with good and poor neurologic outcomes in primary hypothermic cardiac arrest resuscitated with ECPR [18]. This study showed neurologic outcome was affected by serum lactic acid level before ECMO insertion. Several studies showed CPR duration was associated with survival rate and neurological outcomes [9, 19].

Our analysis showed cardiac arrest to ECMO interval was prognostic, but CPR duration was not. CPR duration did not include ROSC time. Since ROSC does not mean stable vital signs with good oxygen delivery, a patient could be still hypotensive and hypoxic due to stunned myocardium and pulmonary problems. Therefore, brain perfusion was affected by cardiac arrest to ECMO interval rather than CPR duration in ECPR. Thus, ECMO use without hesitation may save brain when a patient is still unstable after ROSC.
There are several limitations in this study. This is retrospective review of medical records. CPC scale was retrospectively determined based on medical records. We excluded patients whose neurologic status could not be assessed due to deterioration followed by death. However, we included patients who had a diagnosis of brain death. Therapeutic hypothermia may be associated with neurologic outcomes after cardiac arrest [20]. Therapeutic hypothermia was performed for a limited number of patients in our study. Although there were a few studies which investigated ECPR and neurologic outcomes, most included all ECPR patients [4, 10, 11]. They failed to suggest prognostic factors for favorable neurologic outcome. Those studies also might have included patients who died on ECMO, or quickly after ECMO weaning. Because there is no definition for ECMO procedural success or salvage, we excluded patients who did not survive for more than 24 hours after ECMO initiation.

Conclusions

In conclusion, low hemoglobin level, high serum lactic acid, and prolonged cardiac arrest to ECMO interval were independent predictors for poor neurologic outcome after successful ECPR. Early ECMO institution, low blood transfusion threshold, and a sophisticated EMCO program which enabled accurate ECPR decisions and fast ECMO procedures may improve patient neurologic outcome for those who survive cardiac arrest.

Abbreviations
ECMO, Extracorporeal membrane oxygenation; CPR, Cardiopulmonary resuscitation; ECPR, Extracorporeal cardiopulmonary resuscitation; ROSC, Recovery of spontaneous circulation; CPC, Cerebral performance categories scale; IQR, Interquartile range.

Competing interests

The authors declare that they have no competing interests.

Authors’ contributions

JAR participated in the study design, collection of data, drafting of the manuscript and statistical analysis. YHC participated in conception of the study, the study design, collection of data and drafting of the manuscript. KS participated in statistical analysis and helped draft the manuscript. SHC participated in statistical analysis and helped draft the manuscript. JHY\textsuperscript{1,3} participated in the design of the study and performed statistical analysis and drafted the manuscript. JHC participated in the study design and coordination and helped to draft the manuscript. JHY\textsuperscript{2} participated in conception of the study, the study design, collection of data and drafting of the manuscript. All authors read and approved the final manuscript.

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**Legends to figures**

**Figure 1** Population inclusion and exclusion criteria; ECPR, extracorporeal cardiopulmonary resuscitation.

**Figure 2** Cerebral performance category (CPC) score distribution in good and poor neurologic outcomes.

**Figure 3** Independent predictors of good neurologic outcome; Pre-lactic acid, lactic acid level before ECMO; Pre-Hb, hemoglobin level before ECMO; ECMO, extracorporeal membrane oxygenation; CI, confidence interval.
Table 1 Glasgow-Pittsburgh Cerebral Performance Categories

Cerebral Performance Categories*

1. Good Cerebral Performance
Conscious: Alert, able to work and lead a normal life. May have minor psychological or neurological deficits (mild dysphasia, nonincapacitating hemiparesis, or minor cranial nerve abnormalities).

2. Moderate Cerebral Disability
Conscious. Sufficient cerebral function for part-time work in sheltered environment or independent activities of daily life (dressing, traveling by public transportation, and preparing food). May have hemiplegia, seizures, ataxia, dysarthria, dysphasia, or permanent memory or mental changes.

3. Severe Cerebral Disability
Conscious. Dependent on others for daily support because of impaired brain function (in an institution or at home with exceptional family effort). At least limited cognition. Includes a wide range of cerebral abnormalities from ambulatory with severe memory disturbance or dementia precluding independent existence, to paralytic and able to communicate only with eyes, as in the locked-in syndrome.

4. Coma, Vegetative State
Not conscious. Unaware of surroundings, no cognition. No verbal or psychological interactions with environment.

5. Death
Certified brain dead or dead by traditional criteria.
Table 2 Baseline characteristics of successful ECPR group and univariate analysis for factors associated with good and poor CPC group

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Good CPC group</th>
<th>Poor CPC group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>58.0 (45.0-66.0)</td>
<td>58.0 (44.0-66.0)</td>
<td>58.5 (44.5-69.5)</td>
<td>0.32</td>
</tr>
<tr>
<td>Gender Male (%)</td>
<td>80 (70)</td>
<td>47 (69)</td>
<td>33 (70)</td>
<td>0.90</td>
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<tr>
<td>Height (cm)</td>
<td>164.5 (157.0-170.0)</td>
<td>168.0 (157.0-173.0)</td>
<td>163.0 (155.5-169.5)</td>
<td>0.59</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>62.0 (55.1-69.7)</td>
<td>62.0 (54.9-71.0)</td>
<td>63.2 (55.9-69.6)</td>
<td>0.82</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DM (%)</td>
<td>51 (44)</td>
<td>30 (44)</td>
<td>21 (45)</td>
<td>0.95</td>
</tr>
<tr>
<td>HTN (%)</td>
<td>45 (39)</td>
<td>27 (40)</td>
<td>18 (38)</td>
<td>0.88</td>
</tr>
<tr>
<td>Smoking (%)</td>
<td>49 (43)</td>
<td>29 (43)</td>
<td>20 (43)</td>
<td>0.99</td>
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<tr>
<td>CKD (%)</td>
<td>8 (7)</td>
<td>6 (9)</td>
<td>2 (4)</td>
<td>0.47</td>
</tr>
<tr>
<td>PAOD (%)</td>
<td>4 (4)</td>
<td>2 (3)</td>
<td>2 (4)</td>
<td>0.71</td>
</tr>
<tr>
<td>Dyslipidemia (%)</td>
<td>11 (10)</td>
<td>8 (12)</td>
<td>3 (6)</td>
<td>0.52</td>
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<tr>
<td>Acute coronary syndrome</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STEMI (%)</td>
<td>22 (19)</td>
<td>17 (25)</td>
<td>5 (11)</td>
<td>0.12</td>
</tr>
<tr>
<td>NSTEMI (%)</td>
<td>18 (16)</td>
<td>9 (13)</td>
<td>9 (19)</td>
<td></td>
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<tr>
<td>Unstable angina (%)</td>
<td>4 (4)</td>
<td>4 (6)</td>
<td>0 (0)</td>
<td></td>
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<tr>
<td>Location of arrest</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Category</td>
<td>Hospital (%)</td>
<td>Out of hospital (%)</td>
<td>P-value</td>
<td></td>
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<tr>
<td>---------------------------------------------</td>
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<td></td>
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<tr>
<td>In hospital (%)</td>
<td>96 (83)</td>
<td>61 (90)</td>
<td>35 (75)</td>
<td>0.03</td>
</tr>
<tr>
<td>Out of hospital (%)</td>
<td>19 (17)</td>
<td>7 (10)</td>
<td>12 (26)</td>
<td></td>
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<tr>
<td>Bystander CPR (%)</td>
<td>9 (8)</td>
<td>6 (9)</td>
<td>3 (6)</td>
<td>0.74</td>
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<tr>
<td>Therapeutic hypothermia (%)</td>
<td>9 (8)</td>
<td>5 (7)</td>
<td>4 (9)</td>
<td>0.82</td>
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<tr>
<td>Shockable rhythm (VF/VT) (%)</td>
<td>48 (42)</td>
<td>33 (49)</td>
<td>15 (32)</td>
<td>0.08</td>
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Laboratory data

<table>
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<th>Parameter</th>
<th>Hospital</th>
<th>Out of hospital</th>
<th>P-value</th>
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<tbody>
<tr>
<td>Hemoglobin before ECMO (g/dL)</td>
<td>11.9 (10.3-14.3)</td>
<td>11.9 (10.2-14.8)</td>
<td>10.7 (9.2-12.3)</td>
</tr>
<tr>
<td>Hemoglobin after ECMO (g/dL)</td>
<td>10.4 (9.2-12.9)</td>
<td>10.0 (8.6-12.4)</td>
<td>9.7 (8.6-12.2)</td>
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<tr>
<td>T-bil (mg/dL)</td>
<td>0.9 (0.6-1.5)</td>
<td>0.8 (0.6-1.5)</td>
<td>0.9 (0.7-1.2)</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>17.4 (12.5-24.6)</td>
<td>16.9 (13.8-28.0)</td>
<td>17.0 (11.4-22.1)</td>
</tr>
<tr>
<td>Cr (mg/dL)</td>
<td>1.1 (0.9-1.5)</td>
<td>1.2 (0.9-1.5)</td>
<td>1.2 (0.9-1.7)</td>
</tr>
<tr>
<td>Lactic acid (mmol/L)</td>
<td>8.5 (4.4-13.2)</td>
<td>8.0 (3.9-11.7)</td>
<td>13.6 (6.7-16.5)</td>
</tr>
<tr>
<td>CPR duration (min)</td>
<td>23.0 (15.0-41.0)</td>
<td>19.0 (15.0-28.0)</td>
<td>29.0 (21.0-51.0)</td>
</tr>
<tr>
<td>ROSC before ECMO insertion (%)</td>
<td>50 (44)</td>
<td>31 (46)</td>
<td>19 (40)</td>
</tr>
<tr>
<td>ROSC time (min)</td>
<td>0 (0-5.0)</td>
<td>0 (0-11.0)</td>
<td>0 (0-0)</td>
</tr>
<tr>
<td>Cardiac arrest to ECMO interval (min)</td>
<td>34.0 (20.0-53.0)</td>
<td>24.5 (18.0-36.0)</td>
<td>40.0 (25.0-60.0)</td>
</tr>
</tbody>
</table>

ECPR, extracorporeal cardiopulmonary resuscitation; CPC, cerebral performance category; DM, diabetes mellitus; HTN, hypertension; CKD, chronic kidney disease; PAOD, peripheral arterial occlusive disease; STEMI, ST elevation myocardial infarction; NSTEMI, non-ST elevation myocardial infarction; CPR, cardiopulmonary resuscitation; VF, ventricular fibrillation; VT, ventricular tachycardia; Hb, hemoglobin; T-bil, total bilirubin; BUN, blood urea nitrogen; Cr, creatinine; ROSC, return of spontaneous circulation; ECMO, extracorporeal membrane oxygenation.
<table>
<thead>
<tr>
<th>Variable</th>
<th>OR</th>
<th>(95% CI)</th>
<th>P-value</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>0.965</td>
<td>(0.931-1.001)</td>
<td>0.06</td>
</tr>
<tr>
<td>Gender Male (%)</td>
<td>0.576</td>
<td>(0.139-2.358)</td>
<td>0.45</td>
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<tr>
<td>Pre-lactic acid (mmol/L)*</td>
<td>0.764</td>
<td>(0.660-0.885)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Out of hospital CPR (%)</td>
<td>0.141</td>
<td>(0.017-1.187)</td>
<td>0.07</td>
</tr>
<tr>
<td>Hemoglobin before ECMO (g/dL)*</td>
<td>1.502</td>
<td>(1.070-2.108)</td>
<td>0.02</td>
</tr>
<tr>
<td>Hemoglobin after ECMO (g/dL)</td>
<td>0.932</td>
<td>(0.720-1.207)</td>
<td>0.59</td>
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<tr>
<td>Acute coronary syndrome (%)</td>
<td>3.387</td>
<td>(0.677-16.938)</td>
<td>0.14</td>
</tr>
<tr>
<td>Shockable rhythm (VF/VT) (%)</td>
<td>3.679</td>
<td>(0.881-15.360)</td>
<td>0.07</td>
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<td>CPR duration (min)</td>
<td>1.003</td>
<td>(0.967-1.041)</td>
<td>0.86</td>
</tr>
<tr>
<td>Cardiac arrest to ECMO interval (min)*</td>
<td>0.960</td>
<td>(0.922-0.999)</td>
<td>0.04</td>
</tr>
</tbody>
</table>

*Variables with P value < 0.05, OR, odds ratio; CI, confidence interval.
E-CPR
n=227

Excluded
Did not survive > 24 hour: 22

Successful E-CPR
n = 205

Excluded
Death before formal neurologic assessment: 90

Neurologic status could be assessed
n = 115
Figure 2

The bar chart shows the distribution of CPC categories with the following counts:
- CPC 1: 42
- CPC 2: 26
- CPC 3: 10
- CPC 4: 13
- CPC 5: 24
Figure 3

**Odds Ratio (95% CI)**

- **Pre-lactic acid (mmol/L)**
  - 0.764 (0.660-0.885)
  - *p* value <0.001

- **Hemoglobin before ECMO (g/dL)**
  - 1.502 (1.070-2.108)
  - *p* value 0.02

- **Cardiac arrest to ECMO interval (min)**
  - 0.960 (0.922-0.999)
  - *p* value 0.04

**Odds ratio**

- Poor neurologic outcome
- Good neurologic outcome