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Saving life and brain with extracorporeal cardiopulmonary resuscitation: A single-center analysis of in-hospital cardiac arrests.

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Saving Life and Brain with Extracorporeal Cardiopulmonary Resuscitation (E-CPR). A Single Center Analysis of In-Hospital Cardiac Arrests.

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Abstract

Objective: Despite advances in medical care, survival to discharge and full neurological recovery after cardiac arrest remains < 20% following CPR. An alternate approach to traditional CPR is extracorporeal cardiopulmonary resuscitation (E-CPR), which places patients on ECMO during CPR, and provides immediate cardiopulmonary support when traditional resuscitation has been unsuccessful. Here, we report results from E-CPR at our institution.

Methods: Between 2010 and June 2014, a total of 107 adult ECMO procedures were performed at our institution. Patient demographics, survival to discharge, and neurological recovery of patients that underwent E-CPR were retrospectively analyzed with IRB approval.

Results: 23 patients (15 males and 8 females, mean age 46 ± 12 years) underwent E-CPR. All patients who met criteria were placed on 24-hour hypothermia protocol (target temperature 33 ^{0}C) with initiation of ECMO. The mean duration of ECMO support was 6.2 ± 5.5 days. Nine patients died while on ECMO from the following causes: anoxic brain injury (4), stroke (4), and bowel necrosis (1). Two patients with anoxic brain injury on E-CPR donated multiple organs for transplant. The survival to discharge rate was 30% (7/23 patients) with ~100% full neurological recovery.

Conclusions: The E-CPR procedure provided reasonable patient recovery. E-CPR also allowed for neurological recovery and made multi-organ procurement possible. Based on the above survival rates, E-CPR should be considered when determining the optimal treatment path for patients who need cardiopulmonary resuscitation. The proper use of E-CPR improved hospital outcomes for the in-hospital cardiac arrested patients.

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Title: Saving Life and Brain with Extracorporeal Cardiopulmonary Resuscitation (E-CPR). A Single Center Analysis of In-Hospital Cardiac Arrests.

Central Message: E-CPR offers means of resuscitation for patients refractory to traditional CPR. E-CPR improved hospital outcomes for patients who suffered an in-hospital cardiac arrest.

Perspective: Results from E-CPR at our institution show a ~30% hospital discharge rate with no major neurological consequence. E-CPR also made multi organ procurement possible in non-survivors through ECMO support of end-organ function. Based on these statistics, E-CPR should be considered when determining a treatment path for patients who suffer an in-hospital cardiac arrest.

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List of abbreviations

ALT: alanine aminotransferase.

AMI: acute myocardial infarction.

APACHE II: acute physiology and chronic health evaluation II.

AST: aspartate aminotransferase.

CPR: cardiopulmonary resuscitation.

ECMO: extracorporeal membrane oxygenation.

ICU: intensive care unit.

E-CPR: extracorporeal cardiopulmonary resuscitation.

MELD: model for end-stage liver disease.

SAPS II: simplified acute physiology score II.

SOFA: sequential organ failure assessment score.

VTach/VFib: ventricular tachycardia or ventricular fibrillation.

Introduction

Cardiopulmonary resuscitation (CPR) is a widely known procedure used to save lives when patients undergo cardiac arrest. However, despite being extensively taught and used, CPR remains ineffective. A meta-analysis has shown that 23.8% of out-of-hospital CPR patients survive to admission, and a mere 7.6% of these patients ultimately survive to be discharged from the hospital [1]. Even when CPR takes place in hospital setting, the overall survival rates are not encouraging. It is reported that <50% of patients survive CPR [2-4], while < 20% of patients survive to discharge [2, 4]. These results suggest that when traditional CPR is not effective, alternate means of resuscitation are necessary.

As ECMO rises in popularity and use, there has been increasing interest in its viability and success when used during CPR (E-CPR). E-CPR provides a method to stabilize hemodynamics and provide end-organ perfusion when traditional CPR is inadequate and the cause of cardiac arrest is reversible. While many studies have assessed the efficacy of E-CPR in pediatric populations [5-7], fewer have investigated it in an adult population. The studies of adult populations have assessed the success of E-CPR in adult populations by mortality, and many have not taken end-organ function into account.

In a study that assessed the survival benefits of E-CPR compared to conventional CPR after a witnessed arrest, E-CPR provided a significantly higher return of spontaneous circulation, and a ~ 20% increase in survival rate at discharge [8]. Further studies on E-CPR have shown an increased rate of survival at 1-year [8], and 2-years [9] after discharge when compared to conventional CPR. Many of these studies on adult E-CPR have taken place under optimal conditions in institutions that have designated teams of E-CPR specialists, ready to cannulate patients as a part of the Code Team [8-10].

Through this investigation, there will be greater understanding of the benefits to be gained from E-CPR, such as successful hospital discharge with limited neurological damage, or organ procurement in non-survivors. If shown to be effective, this study will assert that E-CPR should be considered during in-hospital cardiac arrests.

Methods

From June 2010 through July 2014 a total of 107 adult ECMO procedures were done at our institution. Of those 107 procedures, 23 patients had E-CPR after failing to respond to traditional CPR. All E-CPR candidates were in-patients with a witnessed arrest, and the ECMO team was notified less than 20 minutes following the initial arrest. Our institution's E-CPR protocol was applied to all patients as follows; the attending physician in charge of a Code Blue determined whether ECMO was feasible within 20 minutes of unsuccessful resuscitation based on the exclusion criteria: patient's age > 70 years old; the presence of a patient's 'Do Not Resuscitate' orders; whether the patient has an uncorrectable baseline disease such as terminal cancer, advanced coronary artery disease, or a previous neurological deficit; if the patient has uncontrolled sepsis or bleeding. The code team notified the attending physician on call in the surgical cardiovascular ICU, who immediately evaluated the patient's risks and benefits. If all parties were in agreement that ECMO was necessary, the patient was cannulated at the bedside. Perfusionists were called in to set up the ECMO circuit. CPR was continued until ECMO was initiated; all E-CPR patients were started on veno-arterial ECMO. The cannulation procedure was followed as outlined by Lamb and colleagues in order to minimize the risk of excessive bleeding and limb ischemia [11]. Patients who underwent CPR, but no longer required CPR during ECMO cannulation were excluded from this study. In house attending intensivists from the surgical cardiovascular ICU (cardiothoracic surgeons) were responsible for ECMO

placement in all patients. Their coverage spanned all regular daytime working hours, and 4/7 nights with a nocturnist intensivist. Perfusionists were responsible for ECMO circuit setup, and were available in-house during all regular daytime hours. Perfusionsists were also on call during off-hours.

In our institution, 100% of the patients treated with E-CPR were placed on a standard hypothermia protocol at 33 ^{o}C for 24 hours to enhance neurological protection. Target temperature management was met in all patients via an Artic Sun cooling machine. Clinical neurological assessment was continued with both cooling and rewarming phases. If a patient had a focal neurological deficit, uncontrolled seizures, or cerebral oximetry desaturation, the patient was sent for a CT scan immediately—regardless of whether the patient was on or off ECMO. Once rewarmed, any persistent coma or neurological deficit necessitated a CT scan. Following a positive CT scan for suspected anoxic brain injury, neurology was consulted to evaluate neurological outcomes, and if necessary, a cerebral perfusion scan was performed on ECMO to diagnose potential brain death. If the patient was deemed to be non-recoverable, terminal ECMO decannulation was performed after consultations with the family, palliative care team, and the organ procurement agency.

Patient demographics, E-CPR survival, survival to discharge, and organ and neurological recovery were retrospectively analyzed through an Institutional Review Board approved database (Thomas Jefferson University approval #10D.155). The acute physiology and chronic health evaluation II (APACHE II) [12], model for end stage liver disease (MELD) [13], simplified acute physiology score II (SAPS II) [14], and sequential organ failure assessment (SOFA) [15] scores were calculated based on the pre-ECMO, peri-ECMO and post-ECMO data.

Statistical analyses were performed using Chi-square or Fisher's exact tests for categorical variables and Student's t-tests for continuous variables, as appropriate, to identify the risk factors for ECMO death. Similar analyses were performed to identify the risk factors for hospital death among the ECMO survivors. Our sample size was too small for a multivariate analysis. The results were expressed as number with percentage, or mean ± standard deviation. P-values < .05 were considered to be significant.

Results

Patients: The 23 patients who received E-CPR at our institution consisted of 15 males and 8 females with a mean age of 46 ± 12 years. The primary diagnoses leading to E-CPR in the patients were acute myocardial infraction (AMI) (n=9), non-ischemic malignant ventricular tachy-arrhythmia (n=5), myocarditis (n=2), acute pulmonary embolus (n=2), hypothermia (n=2), acute rejection (n=1), drug induced cardiac arrest (n=1), and post-cardiotomy failure (n=1). The initial cardiac rhythms in E-CPR patients were ventricular tachycardia or fibrillation (n=8) or pulseless electrical activity (n=15). All patients with a ventricular tachyarrhythmia received electrical defibrillation or cardioversion as appropriate; however, all 8 patients deteriorated into pulseless electrical activity or cardiac arrest prior to the establishment of ECMO. E-CPR was performed in the intensive care unit (ICU) (n=9), catheterization lab (n=7), emergency department (n=5), and operating room (n=2). Of the patients who had an AMI, 2 patients had a percutaneous coronary intervention (PCI) prior to ECMO. The average time of CPR prior to ECMO was 54 ± 30 min. With the exception of 2 patients, all underwent femoral cannulation as described previously. The remaining patients had an open sternum and were cannulated centrally between the aorta and the right atrium.

ECMO survival: Following E-CPR, the average time on ECMO was 6.2 ± 5.5 days. 14 patients (61%) survived ECMO and 9 patients (39%) died while on ECMO (Figure 1). The causes of death in patients who died on ECMO were anoxic brain injury (n=4), stroke (n=4), and bowel necrosis (n=1). Among the patients who suffered an anoxic brain injury, 2 donated multiple organs for transplant. Among the 14 ECMO survivors, 13 had unchanged or improved status in at least one organ (liver function improved or unchanged in 13, kidney function improved or unchanged in 13, and lactate improved in 12, unchanged in 1 as shown in Table 1). Acute renal failure occurred during ECMO in 5 patients. All 5 of these patients were managed by CVVHD during ECMO treatment. Among the patients who survived ECMO, two were post-cardiotomy failure status post coronary artery bypass graft. Of the variables tested pertaining to patient demographics, clinical risk factors, and pre-ECMO laboratory data, only pre-ECMO creatinine levels (p=0.022), and pre-ECMO pH (p=0.039) correlated with ECMO survival (Table 2). No verified ICU or disease-specific risk scores pre-ECMO had a correlation with E-CPR survival (Table 2). On ECMO, cardiac (myocardial standstill, [p=0.034]), and any neurological complications (stroke and anoxic brain injury) (p=0.001) were factors correlated with death during ECMO (Table 3). Of the risk scores calculated with data from 24 hours after ECMO initiation, the MELD, SOFA, and APACHE II scores were correlated with ECMO survival after E-CPR (Table 4). Isolated data from 24 hours after ECMO initiation associated with ECMO survival included: lactate levels (survivors: 3.7 ± 2.5; non survivors: 8.8 ± 5.3; p=0.015), bicarbonate levels (survivors: 27.1 ± 2.7; non survivors: 22.6 ± 1.3; p= 0.0001), and creatinine levels (survivors: 1.3 ± 0.5; non survivors: 1.9 ± 0.5; p= 0.007).

Hospital Survival: Seven of the 14 patients (50%) who survived ECMO were successfully discharged from the hospital, yielding a hospital survival rate of 30% (7/23) (Figure 1). The

causes of death in the seven patients who died following successful ECMO decannulation were anoxic brain injury post cardiac arrest despite cooling and ECMO (n=2), stroke (n=1), brain death from unknown reason (n=2), sepsis (n=1), and AMI from the non-revascularized coronary artery (n=1). All causes of death appeared to be related to the initial insult leading to cardiac arrest. Of the variables tested pertaining to patient demographics, clinical risk factors, pre-ECMO laboratory data and/or disease-specific risk scores, only pre-ECMO bilirubin levels were correlated with hospital survival ($p=0.040$) (Table 5). No pre-ECMO ICU or disease-specific risk scores were found to correlate with hospital survival after E-CPR. Risk scores taken 24 hours after the initiation of ECMO were not found to have a significant correlation with hospital survival after E-CPR. The only isolated data from 24 hours after ECMO initiation associated with hospital survival was 24-hour PaO2 (survivors: 197 ± 115; non survivors: 248 ± 110; $p=0.037$) which had an association with hospital survival. 1 patient who developed acute renal failure survived ECMO but died before discharge. The overall length of the hospital stay after E-CPR was 43 ± 28 days. All patients who were discharged from the hospital did not demonstrate any gross neurological deficits during a follow up visit 4-6 weeks following discharge from a rehabilitation facility. No patients discharged from the hospital were bed-ridden. There were no limitations of daily activity at 6-8 week follow-ups from the date of discharge.

Discussion

CPR is a technique used around the world to allow patients a second chance at life. When traditional CPR fails in a hospital setting, there are alternative procedures for resuscitation. With the popularity of ECMO growing, and the number of ECMO trained individuals increasing, E-CPR can provide a method by which a patient can be resuscitated when traditional CPR is ineffective.

The present study assesses the survival rate, neurological recovery and end organ function of E-CPR patients in our institution, while attempting to identify risk factors for patients undergoing E-CPR. When compared to previously published CPR survival rates (<20%), our E-CPR survival rate demonstrated reasonable recovery for arrested patients [2, 4].

Technical issues of cannulation during E-CPR remain an issue that has been discussed in the literature. A previous study showed an ischemia complication rate of 30% [27]. However, using our E-CPR technique, including standard peripheral cannulation and the use of distal profusion catheters in all patients, the risk of lower leg ischemia complications has been lowered dramatically.

Our study supports a number of findings from an E-CPR study done in pediatric patients, which demonstrated that patient demographic factors such as age, weight and sex do not effect survival [6], and another paper indicating a correlation with duration of ECMO [16]. Our study also partially agrees with major conclusions from previous studies of adult E-CPR patients, which indicate that a state of acidosis is correlated with poor E-CPR outcomes [20]. However, our results contradicted the findings of a number of other studies, including those which claimed that low inotrope levels, low pre-ECMO lactate levels, shorter CPR duration, and low pre-ECMO SOFA scores were associated with favorable outcomes in E-CPR patients [17-19]. We found no association between any pre-ECMO risk scores (MELD, SAPS II, SOFA, APACHE II) and ECMO survival. Different from previous reports, the current study investigated end-organ function to determine if ECMO allowed a patient's vital organs to remain in functional condition. Many prior studies have not completed a similar examination.

Interestingly, some of the strongest correlations found in the present study relate to values taken 24 hours after the initiation of ECMO. The MELD, SOFA and APACHE II scores taken at

this time point significantly predicted ECMO survival, with a higher score yielding a worse prognosis. Knowing this, along with completing a neurological evaluation, provides clinicians with a benchmark to form a prognosis of whether the patient will survive ECMO following E-CPR. Rather than having to wait a number of days, by calculating MELD, SOFA and APACHE II scores 24 hours after E-CPR, physicians and families can gain better insight into the ultimate chances the patient has at survival.

Neurological complications remain a major cause of patient death. Although one patient who developed a stroke during ECMO survived ECMO, the majority of patients who suffered from anoxic brain injuries or strokes during ECMO developed clinical brain death. Despite the nature of ECMO, and the hypothermia protocols initiated, we found that 8 patients (35%) had clinical brain death while on ECMO, and an additional four patients (17%) had major neurological events after ECMO removal, thus limiting their survival. The timing and causative factors for the neurological events were unclear, and neurological protection remains important in improving the survival after E-CPR. That being said, the neurological recovery among hospital survivors was encouraging. All of the hospital survivors in our study were successfully discharged without any gross neurological deficits. ECMO treatment has been associated with the risk of neurological complications, with E-CPR raising that risk even further [21-22]. Prior studies have compared the efficacy of E-CPR to conventional CPR in preventing long-term neurological damage, and have shown that E-CPR is significantly better at neurological protection than conventional CPR [9]. Recently, a single center observational study assessing the use of aggressive E-CPR combined with hypothermia for cardiac arrest patients demonstrated similar results of full neurological recovery for the E-CPR survivors [28].

Organ preservation is another benefit of ECMO treatment. In all but one of the E-CPR survivors, organ function was either improved or unchanged (Table 1). This is a phenomenon that has previously been associated with beneficial outcomes from ECMO treatment [23]. Furthermore, in two patients who did not survive ECMO and died of anoxic brain injury, solid organs were harvested for transplantation. The procurement and sustained function of organs from ECMO non-survivors is a distinct benefit to be gained from ECMO, as reported previously by our group and others [24-26]. Results from our current study demonstrated the efficacy of E-CPR in also allowing organs to be harvested for transplantation in patients who have suffered an anoxic brain injury. Organ procurement is clearly a topic that has many potential ethical implications, though, and this study does not thoroughly investigate the boundaries that physicians should adhere to when attempting to procure an organ for transplant.

This study was limited due to its retrospective nature, and small sample size. Many patients who received CPR at our institution during the study period were not candidates for ECMO due to their age, various comorbidities, and access to ECMO circuits. In the present setting at our institution, the ECMO team is not a regular part of the Code Team. This leads to limitations as to when E-CPR can be performed due to variable availability of attending physicians, perfusionists and ECMO circuits. This is a setting that differs from that described in a number of previous reports on E-CPR [8-10]. Ideally, our E-CPR sample could have been compared to an internal control group, but our hospital CPR records are incompletely documented, and the CPR patient population differed from ours. While there were nearly 450 Code Blues at our institution during this time period, only 23 patients were given E-CPR. This may have led to bias as to who was treated with E-CPR rather than conventional CPR.

Conclusion

E-CPR provides a viable alternative to traditional CPR for patients refractory to conventional resuscitation measures. E-CPR should be strongly considered when the materials and personnel are available, and patients are unresponsive to conventional CPR. The proper use of E-CPR may greatly improve hospital outcomes for patients who suffer an in-hospital cardiac arrest.

Table 1: Organ function before and after ECMO (ECPR Survivors)

	Pre-ECMO	Post-ECMO
Creatinine (mg/dl)	1.1 ± 0.4	1.1 ± 0.3
AST (IU/L)	147 ± 177	133 ± 178
Lactate (mmol/L)	7.0 ± 5.4	2.1 ± 1.5
Murray score	2 ± 1	1 ± 1

Table 2: Patient demographics, clinical risk factors, laboratory data, and risk scores before E-CPR, comparing ECMO survivors to ECMO non-survivors.

	ECMO Survivors N=14	ECMO Non-Survivors N=9	p
Pre ECMO demographics			
Age (yr.)	46 ± 10	45 ± 16	0.881
Male gender	9 (64%)	6 (67%)	0.907
Body weight (kg)	84 ± 22	88 ± 19	0.637
Body surface area (m^2)	1.96 ± 0.27	2.02 ± 0.25	0.592
Body mass index (kg/m^2)	28 ± 6.2	29 ± 4.5	0.712
Clinical risk factors			
Smoking history	5 (36%)	3 (33%)	0.907
Coronary artery disease	5 (36%)	4 (44%)	0.675
Diabetes	2 (14%)	4 (44%)	0.108
Laboratory data			
White blood cell count (B/L)	13.8 ± 8.3	12.8 ± 5.9	0.742
Hemoglobin (g/dl)	11.5 ± 3.4	12.2 ± 3.1	0.605
Platelet count (B/L)	206 ± 125.4	180.4 ± 99.3	0.129
PaO_2 (mm Hg)	164 ± 144	147 ± 153	0.802
$PaCO_2$ (mm Hg)	45 ± 13	47 ± 17	0.772
HCO_3 (mmol/L)	19 ± 5.8	15 ± 7.1	0.217
Creatinine (g/dl)	1.1 ± 0.4	1.7 ± 0.7	0.022
Bilirubin (mg/dl)	0.9 ± 0.8	0.7 ± 0.8	0.589
AST (IU/L)	147 ± 177	595 ± 889	0.213
ALT (IU/L)	95 ± 108	392 ± 588	0.182
Lactate (mmol/L)	7.0 ± 5.4	13.7 ± 9.3	0.072
pH	7.24 ± 0.17	7.05 ± 0.21	0.039
CPR time before ECMO (min)	52 ± 28	57 ± 35	0.693
Initial rhythm: VAC/VFib	2 (14%)	6 (67%)	0.010
Pre-ECMO Scores			
Pre-ECMO MELD	10.0 ± 3.6	17.6 ± 11.6	0.094
Pre-ECMO SAPS II	61.6 ± 8.1	56.3 ± 20.6	0.639
Pre-ECMO SOFA	14.3 ± 1.1	13.5 ± 2.2	0.388
Pre-ECMO APACHE II	33.1 ± 8.1	35.4 ± 9.2	0.590

Table 3: Complications during ECMO.

	ECMO Survivors N=14	ECMO Non-Survivors N=9	p
Time on ECMO support (days)	7.8 ± 6.3	3.6 ± 2.1	0.033
Any neurological complications	1 (7%)	8 (89%)	<0.001
Anoxic brain injury	0	4 (44%)	0.006
Stroke	1 (7%)	4 (44%)	0.034
Myocardial stunning	1 (7%)	4 (44%)	0.034
Hemothorax	2 (14%)	1 (11%)	0.825
Massive hemoptysis	2 (14%)	0	0.235
Pneumonia	2 (14%)	0	0.235
Cannula Site Bleeding	5 (36%)	2 (22%)	0.493
Liver failure	1 (7%)	2 (22%)	0.295
Ischemic bowel	0	1 (11%)	0.202
Leg ischemia	0	0	NA

Table 4: Risk scores 24 hours after E-CPR.

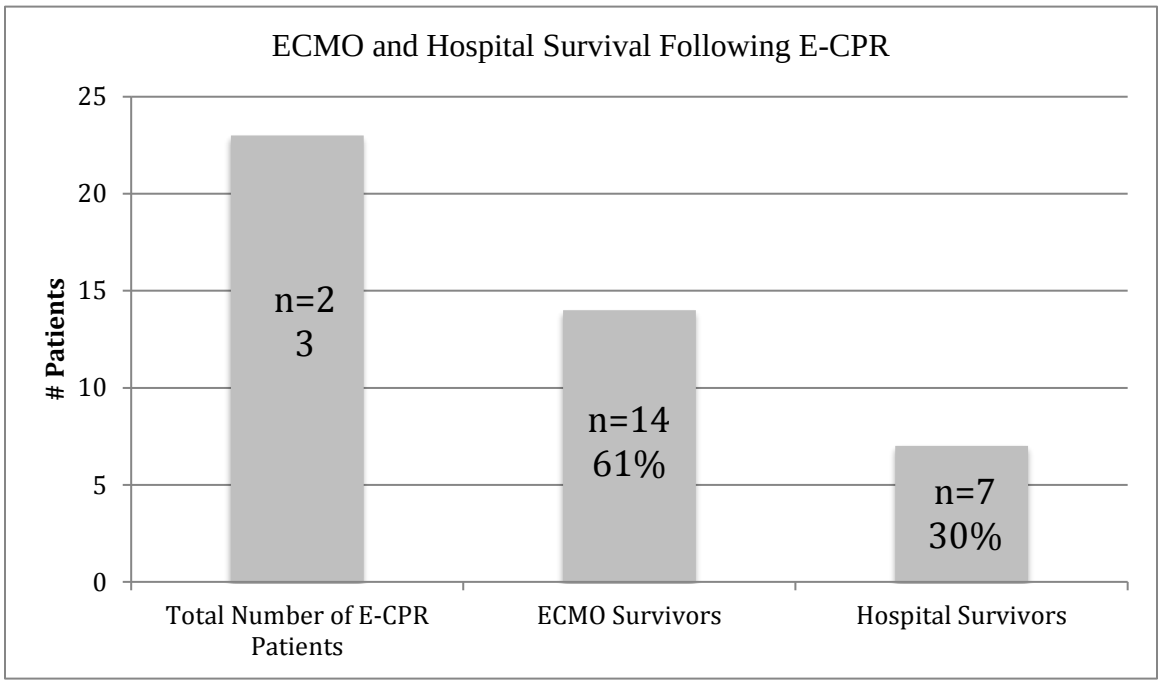
	ECMO Survivors N=14	ECMO Non-Survivors N=9	p
MELD 24hr of ECMO	15.4 ± 4.1	22.2 ± 6.3	0.011
SAPS II 24hr of ECMO	48.1 ± 7.8	53.0 ± 4.2	0.285
SOFA 24hr of ECMO	11.4 ± 1.9	14.5 ± 2.7	0.031
APACHE II 24hr of ECMO	22.8 ± 4.1	29.8 ± 6.7	0.013

Table 5: Patient demographics of ECMO survivors, comparing hospital survivors to hospital non-survivors.

	Hospital Survivors N=7	Hospital Non-Survivors N=7	p
Pre ECMO demographics			
Age (yr.)	50 ± 8	43 ± 10	0.148
Male gender	4 (57%)	5 (71%)	0.577
Body weight (kg)	87 ± 28	80 ± 16	0.604
Body surface area (m^2)	1.97 ± 0.34	1.95 ± 0.20	0.895
Body mass index (kg/m^2)	30 ± 7.3	26 ± 4.9	0.339
Clinical risk factors			
Smoking history	2 (29%)	3 (43%)	0.577
Coronary artery disease	3 (43%)	2 (29%)	0.577
Diabetes	2 (29%)	0	0.127
Laboratory data			
White blood cell count (B/L)	13.9 ± 10.5	13.6 ± 6.1	0.942
Hemoglobin (g/dl)	11.4 ± 3.9	11.5 ± 3.2	0.988
Platelet count (B/L)	218 ± 154.0	194 ± 99.9	0.735
PaO_2 (mm Hg)	129 ± 154	198 ± 136	0.426
$PaCO_2$ (mm Hg)	42 ± 14	47 ± 14	0.580
HCO_3 (mmol/L)	18 ± 5.9	19 ± 6.1	0.776
Creatinine (g/dl)	1.1 ± 0.5	1.1 ± 0.2	0.999
Bilirubin (mg/dl)	0.2 ± 0.2	1.3 ± 0.9	0.040
AST (IU/L)	45 ± 24	207 ± 206	0.132
ALT (IU/L)	37 ± 22	130 ± 127	0.161
Lactate (mmol/L)	5.3 ± 4.6	8.3 ± 6.0	0.382
pH	7.25 ± 0.21	7.23 ± 0.14	0.842
CPR time before ECMO (min)	56 ± 34	44 ± 16	0.524
Initial rhythm: VTach/VFib	1 (14%)	1 (14%)	1.000
Pre ECMO scores			
Pre-ECMO MELD	10.6 ± 3.9	9.8 ± 3.5	0.789
Pre-ECMO SAPS II	53.0 ± 0	63.8 ± 7.5	0.063
Pre-ECMO SOFA	14.0 ± 1.0	14.4 ± 1.1	0.666
Pre-ECMO APACHE II	35.7 ± 9.9	30.0 ± 4.7	0.237

Scores 24hr after ECMO			
MELD 24hr of ECMO	12.5 ± 4.3	17.0 ± 3.1	0.095
SAPS II 24hr of ECMO	46.3 ± 6.7	50.7 ± 9.9	0.533
SOFA 24hr of ECMO	11 ± 0	11.5 ± 2.3	0.607
APACHE II 24hr of ECMO	22.0 ± 4.69	23.4 ± 4.0	0.591

Figure 1. ECMO survival after E-CPR and hospital survival.



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