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Association of Left Ventricular Systolic Function and Vasopressor Support with Survival Following Pediatric Out of Hospital Cardiac Arrest

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Abstract

Objective—To characterize the association of hospital discharge survival with left ventricular (LV) systolic function evaluated by transthoracic echocardiography (TTE) and vasoactive infusion support following return of spontaneous circulation (ROSC) after pediatric out-of-hospital cardiac arrest (OHCA).

Design—Retrospective case series

Setting—Single center tertiary care pediatric cardiac arrest and critical care referral center

Patients—Consecutive OHCA patients <18 years surviving to Pediatric Intensive Care Unit (ICU) admission who had a TTE obtained by the clinical team within 24 hours of admission from January 2006 to May 2012.

Interventions—None

Measurements and Main Results—Fifty-eight patients had a post-ROSC TTE performed within 24 hours of admission. The median time from ROSC to echo was 6.5 [IQR 4.7, 15.0] hours. LV systolic function was decreased in 24/58 (41%) patients. The mortality rate was 67% (39/58). Thirty-six patients (62%) received vasoactive infusions at the time of TTE and increased vasopressor inotropic score (VIS) was associated with increased mortality on univariate analysis ($p < 0.001$). After controlling for defibrillation, VIS and interaction between VIS and LV systolic function, decreased LV systolic function was associated with increased mortality (OR 13.7 [95% CI: 1.54, 122]).

Conclusions—In patients receiving TTE within the first 24 hours following ROSC after pediatric OHCA, decreased LV systolic function and vasopressor use were common. Decreased LV systolic function was associated with increased mortality.

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Keywords

cardiac arrest; resuscitation; echocardiogram; vasopressor; hemodynamic

Introduction

Up to 15,000 children in the United States suffer an out-of-hospital cardiac arrest (OHCA) every year.^{1–3} Between 10 and 30% of pediatric OHCA patients treated in the field have sustained return of spontaneous circulation (ROSC) and survive to be admitted to a pediatric intensive care unit (ICU). Of these, <40% survive to hospital discharge.^{3–5}

Over the last two decades a post-cardiac arrest syndrome characterized by myocardial dysfunction, brain injury, and a typical ischemia-reperfusion response has been described.^{6–8} Global myocardial dysfunction peaks within 8 hours of the ischemic insult, and is frequently associated with early hemodynamic instability.^{8–9} Though this myocardial dysfunction may be transient and reversible, decreased left ventricular (LV) systolic function following cardiac arrest has been associated with increased mortality in adults.^{7,10}

We sought to characterize the prevalence of LV systolic dysfunction and vasopressor support after successful return of spontaneous circulation following pediatric OHCA. We hypothesized that decreased LV systolic function on transthoracic echocardiography (TTE) within the first 24 hours following successful resuscitation would be associated with increased mortality.

Materials and Methods

We performed a retrospective study of patients 1 month to 18 years of age presenting to the Pediatric ICU at The Children's Hospital of Philadelphia from January 2006 through May 2012. Patients were included for analysis if they had an OHCA, received any chest compressions, had sustained ROSC > 20 minutes and TTE performed by the clinical team within 24 hours of PICU admission. The study was approved with a waiver of informed consent granted by the Children's Hospital of Philadelphia Institutional Review Board.

Patients were prospectively identified from September 2009 to May 2012 and entered into a cardiac arrest database. Patients from January 2006 through September 2009 were identified from existing hospital cardiac arrest databases. Search terms were "code," "arrest," "cardiac arrest," "CPR," "prior CPR," "hanging," "drowning," "near drowning," "SIDS," and "ALTE." All identified patients underwent chart review to verify they had received chest compressions and qualified as an OHCA.

Pre-hospital and inpatient medical records were reviewed by a single trained investigator. Data extracted included: pre-arrest patient demographics and characteristics, cardiac arrest event details, vasoactive infusion dosing, hemodynamic variables and laboratory values at the time of TTE, and survival to hospital discharge outcomes. Vital signs and vasopressor data were obtained within 1 hour of TTE; troponin and B-type natriuretic peptide (BNP) were recorded if obtained within 12 hours of TTE; lactate, central venous oxygen saturation

(ScvO₂), ionized calcium, and pH were recorded if obtained within 4 hours of TTE and were exclusively arterial unless only free flowing venous sample was available.

Vasopressor and inotrope dosing were standardized by calculating a vasopressor inotropic score (VIS) at the time of TTE as previously described.^{11–13}
$$\text{VIS} = (\text{dopamine dose } [\mu\text{g/kg/min}] \times 1) + (\text{dobutamine dose } [\mu\text{g/kg/min}] \times 1) + (\text{epinephrine dose } [\mu\text{g/kg/min}] \times 100) + (\text{norepinephrine dose } [\mu\text{g/kg/min}] \times 100) + (\text{phenylephrine dose } [\mu\text{g/kg/min}] \times 100) + (\text{milrinone dose } [\mu\text{g/kg/min}] \times 10) + (\text{vasopressin dose } [\text{U/kg/min}] \times 10,000).$$

TTE images were reviewed by a board certified pediatric cardiologist blinded to patient history, VIS and outcome. Patients were eligible for analysis if there were adequate images for qualitative assessment of LV systolic function, because qualitative assessment using TTE is a reliable measure of systolic function when performed by expert echocardiographers.^{14–17} Additional TTE myocardial performance data (LV shortening fraction by M-Mode, qualitative right ventricular [RV] systolic function, LV diastolic function by E/A ratio of the mitral inflow, RV diastolic function by E/A ratio of the tricuspid inflow, septal wall motion abnormality as well as structural abnormality) were documented for each TTE when available. Myocardial performance abnormality is defined by an abnormality in one or more of these measures. Qualitative LV systolic function was categorized as “decreased” (mild, moderate or severe dysfunction) or “not decreased” (hyperdynamic or normal).

The primary outcome was in-hospital mortality. Summary statistics are reported as median and interquartile ranges [IQR: 25th–75th percentiles] for continuous variables and proportions as percentages for categorical variables. Fisher’s exact tests or Wilcoxon rank-sum tests were used to determine differences between groups. Non-collinear covariates were introduced into the multiple regression model if a univariate analysis with mortality revealed a p value <0.1. LV systolic function was *a priori* chosen to be included into the model because of its clinical and intuitive relevance.¹⁸ After creating our main effects model, we included the interaction between LV systolic function and VIS given their physiologic interdependence and clinical relevance. C-statistic was performed to determine the predictive accuracy of our model. A significance value of < 0.05 was used for all analyses. All statistical analyses were conducted using SAS software (version 9.2; SAS Institute, Cary, NC).

Results

Patients

Of 169 patients surviving OHCA to Pediatric ICU admission, 59 (35%) had TTEs performed within 24 hours of ICU admission. One patient did not have interpretable TTE images and was excluded resulting in 58 patients eligible for analysis. Forty-five percent (26/58) of patients had a pre-existing condition including chronic lung disease, asthma, congenital heart disease, developmental delay, cancer, prematurity, epilepsy and neuromuscular disease. Except for developmental delay (14), congenital heart disease (8) and chronic lung disease (7), all other pre-existing conditions were found in 5 patients with some patients having more than one pre-existing condition. Of the patients with

congenital heart disease, 5 had a prior TTE demonstrating normal LV function and 3 did not have a prior TTE for evaluation. Of the 8 patients with a first documented cardiac arrest rhythm of ventricular fibrillation or pulseless ventricular tachycardia, cause of arrest included arrhythmia (6), drowning (1) and unknown (1). Twenty-five patients (43%) were clinically managed with therapeutic hypothermia targeting a core temperature between 32 and 34°C in the first 24 hours after ROSC.

Echocardiographic Data

Median time from admission to TTE was 4.5 hours [IQR: 2.9–10.6 hours]. Median time from ROSC to TTE (n=46) was 6.5 hours [IQR 4.7, 15.0 hours]. Forty-one percent of patients (24/58) had decreased LV systolic function. Quantitative shortening fractions were determined in 38 patients (66%). Among patients with qualitatively decreased LV systolic function and M-Mode measurements for quantitative shortening fraction assessment available, all had shortening fractions $\leq 27\%$ (18/18, Table 2). Other myocardial performance abnormalities included: abnormal RV systolic function 18% (10/56), abnormal septal wall movement 42% (19/44), mitral valve E/A reversal indicative of abnormal LV diastolic function 64% (23/36) and tricuspid valve E/A reversal indicative of abnormal RV diastolic function 65% (20/31). Overall, 79% (46/58) of patients had evidence of a myocardial performance abnormality on initial TTE. Ten patients had repeat TTE within 72 hours. Eight patients had no change in qualitative LV systolic function. One patient transitioned from hyperdynamic LV systolic function to mild dysfunction and died. One patient transitioned from mild LV systolic dysfunction to normal function and survived. Four patients (7%) had a new diagnosis of structural heart disease based upon post-ROSC TTE: LV non-compaction (2), anomalous right coronary artery arising from the left coronary sinus (1), and ventricular septal defect (1).

There were no significant differences in demographics, pre-event or arrest characteristics between groups with and without LV systolic dysfunction (Table 1). Low systolic, diastolic, mean and central venous blood pressures at the time of echocardiographic assessment were not associated with decreased LV systolic function (Table 2). Troponin level was the only other laboratory or hemodynamic variable significantly associated with decreased LV systolic function ($p=0.01$, Table 2). Thirty-six patients (62%) were treated with vasopressor support at the time of TTE. Of those on support, 27 patients (75%) were treated with dopamine, 25 (69%) with epinephrine, 4 (11%) with vasopressin, 2 (6%) with norepinephrine, 2 (6%) with dobutamine, 2 (6%) with phenylephrine and 1 (3%) with milrinone. VIS at the time of TTE was not associated with LV systolic function ($p=0.45$, Table 2).

Survival Outcome

In our cohort of post-ROSC patients evaluated by TTE within 24 hours of PICU admission, mortality was 67% (39/58). The overall mortality of patients admitted to the PICU after OHCA during that same time period was 48% (81/169). Within our cohort, no demographic or pre-arrest characteristics were associated with mortality. Arrest characteristics associated with mortality were: witnessed status, first documented cardiac arrest rhythm, number of epinephrine doses, defibrillation, primary cause of arrest and duration of chest compressions

(Table 3). No patient with a first documented rhythm of ventricular fibrillation or pulseless ventricular tachycardia died.

Seven patients (18%) died from a cardiac etiology, specifically re-arrest or withdrawal due to refractory circulatory failure, which were not associated with decreased LV systolic function (OR = 0.8 [0.15, 4.18], $p=0.79$). In patients with decreased LV systolic function who died, cause of death included brain death [9, (50%)], withdrawal due to neurologic failure [5, (28%)], withdrawal due to respiratory failure [1, (6%)], and withdrawal due to refractory circulatory failure [3, (17%)]. No patients with decreased LV systolic function died after re-arrest. Patients who had a lower temperature, higher heart rate, higher lactate level and higher mean airway pressure at the time of TTE were more likely to die. Patients managed with targeted temperature management $< 34^{\circ}\text{C}$ composed 42% (8/19) of survivors and 44% (17/39) of non-survivors. A higher VIS at the time of TTE was also associated with increased mortality ($p<0.001$, Table 4). No myocardial performance measures, including LV ($p=0.09$) or RV ($p=0.05$) diastolic dysfunction, were associated with mortality on univariate analysis (Table 4).

In a multivariable analysis controlling for defibrillation, there is a significant interaction between VIS and LV systolic function. Specifically, when VIS equals 0, the odds of death are decreased for those with normal LV Systolic versus those with decreased LV systolic function (adjusted OR 13.7 [1.54 – 121.9]; $p = 0.019$; Table 5). Further, as VIS increases, the odds of death is significantly increased for those with normal LV systolic function such that the difference in odds of death between the two LV function groups no longer differs (Table 5). The c-statistic for our final model was 93.6%.

Discussion

In this single center retrospective study, 41% (24/58) of children evaluated with TTE following OHCA had decreased LV systolic function. Importantly, decreased LV systolic function was associated with increased mortality when VIS was 0. Clinical parameters such as heart rate, blood pressure and central venous pressure assessed near the time of echocardiogram were not useful in differentiating patients with and without decreased LV systolic function.

The demographic and arrest characteristics associated with survival in our cohort is similar to previously published pediatric OHCA populations.^{1,19} We chose LV systolic function as our primary exposure as it is often evaluated after cardiac arrest for treatable causes of hypotension and has been associated with increased mortality in pediatric and adult populations following cardiac arrest.^{7,10,20–21} Decreased LV systolic function was common in our cohort and, after controlling for defibrillation, VIS and interaction between VIS and LV systolic function, was associated with increased mortality. When comparing patients with good and poor LV systolic function, as VIS increased from 5 to 15, the odds of death between these two groups was not different.

A heterogeneous pattern of myocardial performance abnormalities were found on TTE. Though our hypothesis focused on post-arrest LV systolic function, diastolic dysfunction

was even more common. No pre-arrest or arrest characteristics evaluated were associated with abnormal LV systolic function post-arrest. Chang et al. demonstrated an association between number of epinephrine doses and decreased LV systolic function in their cohort of adult OHCA patients.¹⁰ Laurent et al. also demonstrated an association between number of epinephrine doses and hemodynamic insufficiency defined by vasopressor requirement.⁷ It is unclear why, within our cohort, we did not find this association. It is possible that children and adults are distinctly different as 47% and 33% of patients in the Laurent and Chang studies, respectively, had coronary artery occlusion as likely cause of arrest. Further, Chang found that history of myocardial infarction was an independent predictor of abnormal LV systolic function on multivariate analysis. Epinephrine, as theorized in their discussion, may serve to increase myocardial oxygen demand in a setting of decreased supply and distribution. Such precipitating coronary pathology infrequently occurs in children. It is also possible that post cardiac arrest syndrome is distinctly different in its hemodynamic manifestation within the pediatric population, similar to septic shock. Use of echocardiography may also not be the most precise means of evaluating hemodynamic status, though remains a safe and readily available resource within larger centers.

Usual clinical and laboratory parameters to assess LV systolic function such as heart rate, blood pressures and BNP documented near the time of echocardiogram were not helpful in discriminating our patients with decreased LV systolic function. Despite our continued reliance upon clinical examination and serum biomarkers to construct individual hemodynamic profiles, surrogate markers thought to represent core physiologic variables are frequently not reliable or validated in the literature.^{22–24} The data from our study suggest the need for improved individual post-cardiac arrest hemodynamic characterization, and perhaps the development of a personalized, goal-directed approach to management as recommended by The International Liaison Committee on Resuscitation in their 2008 Consensus Statement.⁶

Patients who died had lower body temperature at the time of echocardiogram than those who survived, although there were similar rates of therapeutic hypothermia between groups. There is evidence that impaired thermoregulation post-arrest may be associated with injury severity and mortality.^{25–27} Further, brain injury is a known component of post cardiac arrest syndrome. Within brain injury literature, extremes of temperature dysregulation appear to be associated with poor outcomes.^{28–29}

In the mid-1990s, post-cardiac arrest myocardial dysfunction was first described in animal models of cardiac arrest, including both systolic and diastolic LV dysfunction.^{8,30–31} Without inotropic support, the myocardial dysfunction became progressively worse over the first several hours post-cardiac arrest and resolved within 24 hours. Further laboratory studies showed that the post-cardiac arrest LV dysfunction could be ameliorated by continuous inotropic infusions.^{31–32}

Soon after the descriptions of post-cardiac arrest myocardial dysfunction in animal investigations, Laurent et al. demonstrated that myocardial dysfunction was common following out-of-hospital cardiac arrests in adults.⁷ Importantly, they showed that a low cardiac index at 24 hours post-resuscitation was associated with worse multi-organ

dysfunction and mortality. Further, Chang et al. noted that LV ejection fraction < 40% six hours after adult OHCA resuscitation predicted mortality.¹⁰ Consistent with animal studies, the myocardial dysfunction typically resolved within 24–72 hours in adult survivors following OHCA.⁷

In addition to the animal and adult data, Checchia and colleagues evaluated troponins and echocardiograms in 24 children with OHCA who survived to hospital admission.²⁰ Among their patients who had echocardiographic evaluations within 24 hours of hospital admission, higher troponin levels were associated with worse LV systolic function, and decreased LV systolic function was associated with increased mortality.²⁰ They did not report diastolic data. Our echocardiographic findings in a substantially larger population (58 children) were generally consistent with their data: LV systolic dysfunction was common and associated with both increased troponin levels and increased mortality. Our data also demonstrated that post-arrest diastolic dysfunction was common when data were available for evaluation.

Our study had several limitations. As with all retrospective studies, there is also potential for bias. First, of the 169 pediatric patients surviving OHCA and presenting to the PICU, 111 did not receive TTE evaluations within 24 hours of presentation. Forty-two (38%) of these patients died prior to discharge compared with a 67% mortality in our cohort. It is likely clinicians performed echocardiograms on patients with greater severity of illness which may have contributed to differences in outcome. Despite this, our cohort of patients with TTE have similar demographics and survival outcome compared to previously described pediatric OHCA populations.^{1,2,19} Second, the timing of echocardiogram after ROSC was variable, so the trajectory of myocardial dysfunction could not be delineated and some patients may have developed myocardial dysfunction at other times post-ROSC. However, the median time to echocardiogram was 6.5 hours, which is within the window of peak myocardial dysfunction as described in adult and animal models. Third, we selected qualitative measure of LV systolic function as our primary exposure in part because not all images allowed for quantitative metric assessment. Importantly, 67% of our patients had quantitative assessments, and the quantitative assessments were strongly associated with the qualitative grouping. However, given the retrospective nature of this descriptive study, it is difficult to interpret whether left ventricular dysfunction is a significant contributor to the cause of death, or simply a surrogate marker for severity of illness. Finally, though we noted a large proportion of diastolic dysfunction in our post-arrest population, we were not able to obtain measures of diastolic dysfunction in all of our evaluated patients. Prospective standardized TTE measures of systolic and diastolic function at specific time points after ROSC is a logical next step toward determining potential goal-directed therapy targets.

Conclusions

In patients receiving TTE within the first 24 hours following admission after pediatric OHCA, 41% of patients with TTE data had decreased LV systolic function. Clinical parameters such as heart rate, blood pressure and central venous pressure were not useful in differentiating patients with and without decreased LV systolic function. Importantly, LV systolic dysfunction was associated with increased mortality.

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Table 1

Patient Demographics, Pre-Event and Event Data by Left Ventricular Systolic Function Category

	<i>n (%)</i>	<i>Decreased Left Ventricular Systolic Function n=24 n (%)</i>	<i>Not Decreased Left Ventricular Systolic Function n=34 n (%)</i>	<i>p-value</i>
Gender				0.58
Male	41 (71)	18(75)	23 (68)	
Pre-Existing Condition				
Any	26 (45)	10 (42)	16 (47)	0.68
Witnessed Arrest	22 (38)	11 (46)	11 (32)	0.41
Bystander CPR	35 (60)	14 (58)	21 (62)	0.99
First Documented Cardiac Arrest Rhythm				0.93
Asystole/PEA	31 (53)	14 (58)	17 (50)	
VF/VT	19 (33)	3 (13)	5 (15)	
Unknown	8 (14)	7 (29)	12 (35)	
Number of Epinephrine Doses				0.71
0	11 (19)	5 (21)	6 (18)	
1	7 (12)	1 (4)	6 (18)	
2	4 (7)	2 (8)	2 (6)	
3	14 (24)	5 (21)	9 (26)	
4	10 (17)	6 (25)	4 (12)	
5	2 (3)	1 (4)	1 (3)	
6	9 (16)	4 (17)	5 (15)	
Unknown	1 (2)	0 (0)	1 (3)	
Defibrillation	16 (28)	9 (37)	7 (21)	0.23
Primary Cause of Arrest				0.73
ALTE/SIDS	14 (24)	6 (25)	8 (24)	
Drowning	8 (14)	1 (4)	7 (21)	
Respiratory Failure	2 (3)	1 (4)	1 (3)	
Airway Obstruction	6 (10)	3 (13)	3 (9)	
Arrhythmia	7 (12)	4 (17)	3 (9)	
Hypotension/Shock	3 (5)	2 (8)	1 (3)	
Trauma	9 (16)	4 (17)	5 (15)	
Ingestion/Toxin	2 (3)	1 (4)	1 (3)	
Unknown	7 (12)	2 (8)	5 (15)	
	<i>Median (IQR)</i>	<i>Decreased Left Ventricular Systolic Function n=24 Median (IQR)</i>	<i>Not Decreased Left Ventricular Systolic Function n=34 Median (IQR)</i>	<i>p-value</i>
Age (years)	4.1 (0.57–11.0)	6.0 (0.52–14.4)	3.7 (0.57–8.9)	0.49
Duration CPR (minutes, n=54)	25.0 (13.0–42.0)	25.0 (15.0–45.0)	25.5 (13.0–41.0)	0.75

ALTE/SIDS, acute life threatening event/sudden infant death syndrome; CPR, cardiopulmonary resuscitation; PEA, pulseless electrical activity

Table 2

Laboratory and Hemodynamic Variable Association at Time of Transthoracic Echocardiogram by Left Ventricular Systolic Function Category

		Decreased Left Ventricular Systolic Function n=24	Not Decreased Left Ventricular Systolic Function n=34	
	n	Median (IQR)	Median (IQR)	p-value
Temperature (°C)	56	34.5 (32.6 – 36.5)	34.8 (33.3 – 36.6)	0.69
Heart Rate (bpm)	57	123 (92 – 142)	126.5 (97.0 – 153.0)	0.48
Systolic Blood Pressure (mmHg)	57	110 (91 – 126)	107.0 (93.0 – 124.0)	0.44
Diastolic Blood Pressure (mmHg)	57	64(47 – 75)	56.5 (46.0 – 71.0)	0.24
Mean Arterial Pressure (mmHg)	52	83 (69.0 – 91.0)	77.0 (60.0 – 93.0)	0.49
Central Venous Pressure (mmHg)	25	10.0 (6.0 – 13.0)	10.0 (8.0 – 15.0)	0.28
Lactate (mmol/L)	54	6.9 (3.3 – 11.9)	5.2 (1.5 – 7.9)	0.13
pH	57	7.3 (7.2 – 7.4)	7.3 (7.2 – 7.4)	0.91
Ionized Calcium (mmol/L)	54	1.1 (1.0 – 1.2)	1.2 (1.1 – 1.2)	0.22
B-type Natriuretic Peptide (pg/mL)	13	169.9 (79.4 – 1633.6)	93.9 (76.2 – 195.1)	0.48
Troponin (ng/mL)	36	6.6 (1.0–22.8)	0.37 (0.14 – 1.9)	0.01
Mean Airway Pressure (cmH ₂ O)	57	17.0 (15.0 – 23.0)	14.5 (11.0 – 20.0)	0.10
Vasopressor Inotropic Score	58	11.5 (0 – 28)	5 (0 – 20)	0.45
LV Shortening Fraction (%)	38	26.0 (20.0 – 28.0)	35.0 (33.0 – 39.0)	<0.001
SvO ₂ (%)	22	56.5 (49.6 – 79.9)	65.8 (61.6 – 77.5)	0.37

bpm, beats per minute; °C, degrees Celsius; mmHg, millimeters of mercury; mmol/L, millimoles per Liter; pg/ml: picograms per milliliter; ng/ml, nanograms per milliliter; cmH₂O, centimeters of water; LV, left ventricle; IQR, interquartile range; SvO₂, central venous oxygen saturation

Table 3

Patient Demographics, Pre-Event and Event Data by Discharge Survival Outcomes

	<i>n</i> (%)	<i>Non-survival n=39</i> (%)	<i>Survival n=19</i> (%)	<i>p-value</i>
Male Gender	41 (71)	25 (64)	16 (84)	0.14
Pre-existing Condition	26 (45)	17 (44)	9 (47)	0.79
Witnessed Arrest	22 (38)	11 (28)	11 (58)	0.04
Bystander CPR	35 (60)	24 (62)	11 (58)	0.99
First Documented Cardiac Arrest Rhythm				<0.001
Asystole/PEA	31 (53)	26 (67)	5 (26)	
VF/VT	19 (33)	0 (0)	8 (42)	
Unknown	8 (14)	13 (33)	6 (32)	
Number of Epinephrine Doses				<0.001
0	11 (19)	1 (3)	10 (53)	
1	7 (12)	5 (13)	2 (11)	
2	4 (7)	3 (8)	1 (5)	
3	14 (24)	10 (26)	4 (21)	
4	10 (17)	9 (23)	1 (5)	
5	2 (3)	1 (3)	1 (5)	
6	9 (16)	9 (23)	0 (0)	
Unknown	1 (2)	1 (3)	0 (0)	
Defibrillation	16 (28)	5 (13)	11 (58)	<0.001
Primary Cause of Arrest				0.01
ALTE/SIDS	14 (24)	11 (28)	3 (16)	
Drowning	8 (14)	3 (8)	5 (26)	
Respiratory Failure	2 (3)	2 (5)	0 (0)	
Airway Obstruction	6 (10)	4 (10)	2 (11)	
Arrhythmia	7 (12)	1 (3)	6 (32)	
Hypotension/Shock	3 (5)	3 (8)	0 (0)	
Trauma	9 (16)	9 (23)	0 (0)	
Ingestion/Toxin	2 (3)	2 (5)	0 (0)	
Unknown	7 (12)	4 (10)	3 (16)	
	<i>Median (IQR)</i>	<i>Non-survival n=39 Median (IQR)</i>	<i>Survival n=19 Median (IQR)</i>	<i>p-value</i>
Age (years)	4.1 (0.57–11)	3.2 (0.4–9.7)	6.8 (0.57–14.6)	0.17
Duration CPR (minutes, n= 54)	25.5 (13.0–42.0)	36.0 (21.0–50.0)	10.0 (2.0–20.0)	<0.001

ALTE/SIDS, acute life threatening event/sudden infant death syndrome; CPR, cardiopulmonary resuscitation; PEA, pulseless electrical activity

Table 4

Laboratory and Hemodynamic Variable Association at Time of Transthoracic Echocardiogram by Discharge Survival Outcomes (*p*-values presented are from univariate regression model)

	<i>n</i>	Non-Survival <i>n</i> =39	Survival <i>n</i> =19	<i>OR</i> [95% <i>CI</i>]	<i>p</i> -value
		<i>Median (IQR)</i>	<i>Median (IQR)</i>		
Temperature (°C)	56	34.2 (32.6 – 35.4)	36.5 (33.4 – 36.9)	0.68 [0.49, 0.94]	0.018
Heart Rate (bpm)	57	136.5 (103.0 – 165.0)	109.0 (82.0 – 132.0)	1.02 [1.00, 1.04]	0.03
Systolic Blood Pressure (mmHg)	57	108.5 (81.0 – 124.0)	110.0 (102.0 – 129.0)	0.98 [0.96, 1.00]	0.073
Diastolic Blood Pressure (mmHg)	57	60.5 (46.0 – 74.0)	57.0 (48.0 – 66.0)	1.00 [0.98, 1.04]	0.59
Mean Arterial Pressure (mmHg)	52	79.0 (60.0 – 91.0)	77.0 (69.0 – 89.0)	0.99 [0.97, 1.03]	0.84
Central Venous Pressure (mmHg)	25	10.5 (7.0 – 15.0)	9.0 (8.0 – 10.0)	1.12 [0.89, 1.39]	0.32
Lactate (mmol/L)	54	7.0 (4.1 – 11.9)	3.0 (1.5 – 3.4)	1.42 [1.11, 1.81]	0.005
pH	56	7.3 (7.2 – 7.4)	7.3 (7.2 – 7.4)	1.96 [0.37, 10.4]	0.43
Ionized Calcium (mmol/L)	54	1.1 (1.0 – 1.2)	1.2 (1.1 – 1.2)	0.03 [0.00, 7.2]	0.22
B-type Natriuretic Peptide (pg/mL)	13	93.9 (37.1 – 234.2)	140.1 (84.4 – 206.0)	1.00 [0.99, 1.00]	0.42
Troponin (ng/mL)	36	2.5 (0.2 – 13.3)	0.6 (0.2 – 1.9)	1.17 [0.92, 1.47]	0.19
SvO2 (%)	22	59.5 (46.9 – 76.0)	71.4 (63.8 – 78.7)	0.96 [0.89, 1.04]	0.32
Mean Airway Pressure (cmH ₂ O)	57	16.5 (14.0 – 24.0)	12.0 (9.0 – 19.0)	1.08 [0.99, 1.18]	0.058
Vasopressor Inotropic Score	58	13.0 (5.0 – 50)	0.0 (0.0 – 0.0)	1.12 [1.02, 1.23]	0.02
	<i>n</i> (%)	Non-Survival <i>n</i> =39 (%)	Survival <i>n</i> =19 (%)		<i>p</i> -value
Left Ventricular Systolic Function	58				
Not Decreased	34 (59)	21 (54)	13 (68)		
Decreased	24 (41)	18 (46)	6 (32)	1.86 [0.59, 5.89]	0.29
Left Ventricular Diastolic Dysfunction *	35	22	13		
Present	22 (63)	16 (73)	6 (46)	3.1 [0.74, 13.11]	0.12
Absent	13 (37)	6 (27)	7 (54)		
Right Ventricular Diastolic Dysfunction *	31	21	10		
Present	20 (65)	16 (76)	4 (40)	4.8 [0.95, 24.1]	0.057
Absent	11 (35)	5 (24)	6 (60)		

bpm, beats per minute; °C, degrees Celsius; mmHg, millimeters of mercury; mmol/L, millimoles per Liter; pg/ml, picograms per milliliter; ng/ml, nanograms per milliliter; cmH₂O, centimeters of water; IQR, Interquartile range

* Echo data assessing diastolic dysfunction not complete

Table 5

Odds of death if LV shortening fraction qualitatively decreased after controlling for defibrillation, Vasopressor Inotropic Score and the interaction between LVSF and VIS. LVSF: LV shortening fraction; VIS: Vasopressor Inotropic Score

	OR 95% CI	<i>p</i>
Decreased Qualitative LV Shortening Fraction	13.71 [1.54, 121.95]	0.019
Defibrillated	0.06 [0.008, 0.44]	0.006
Vasopressor Inotropic Score	1.7 [1.09, 2.65]	0.019
LVSF + VIS Interaction	0.61 [0.39, 0.95]	0.031