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Impact of donor cardiac arrest on heart transplantation

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Abstract

Background—Cardiac transplantation is an effective therapy for patients with end-stage heart failure, but it is still hindered by the lack of donor organs. A history of donor cardiac arrest raises trepidation regarding the possibility of poor post-transplant outcomes. The impact of donor cardiac arrest following successful cardiopulmonary resuscitation on heart transplant outcomes is unknown. Therefore, we sought to evaluate the impact of donor cardiac arrest on orthotopic heart transplantation using the United Network for Organ Sharing database.

Methods—We performed a secondary longitudinal analysis of all cardiac transplants performed between April 1994 and December 2011 through the United Network for Organ Sharing registry. Multiorgan transplants, repeat transplants, and pediatric recipients were excluded. Survival analyses were performed using Kaplan-Meier methods as well as multivariate adjusted logistic regression and Cox proportional hazard models.

Results—A total of 19,980 patients were analyzed. In 856 cases, the donors had histories of cardiac arrest, and in the remaining 19,124 cases, there was no history of donor cardiac arrest. The unadjusted 1-, 5-, and 10-year actuarial survival rates between the arrest and the nonarrest groups were not significantly different. Multivariate logistic regression demonstrated no difference in survival in the donor arrest group at 30 days, 1 year, or 3 years. Furthermore, the adjusted Cox proportional hazard model for cumulative survival also showed no survival difference between the 2 groups.

Conclusion—If standard recipient and donor transplantation criteria are met, a history of donor cardiac arrest should not prohibit the potential consideration of an organ for transplantation.

Cardiac transplantation is the most effective treatment for patients with end-stage heart failure.^{1,2} Unfortunately, only 1 in 4 donors who have given consent are utilized for heart transplantation, and the availability of suitable donor organs limits its effectiveness.³ Strategies to increase the donor pool may, therefore, have a substantial impact on the treatment of patients with severe end-stage heart failure.

A substantial proportion of organ donors have histories of cardiac arrest prior to their eventual diagnosis of brainstem death. Many of these patients are resuscitated successfully and regain normal cardiac function, as evidenced by hemodynamic and echocardiographic

data. In such circumstances, these donors are commonly approached with caution by transplant surgeons. Although many of these hearts may meet standard functional criteria for transplantation, they have been exposed to a period of warm ischemia, which may result in myocardial injury. The critical question is whether these hearts can withstand the subsequent ischemic injury associated with procurement, storage, and transportation. Previous studies have suggested that such donor hearts can be used for transplantation with successful short- and long-term clinical outcomes; however, the number of donors studied has been small.⁴ We sought to evaluate the impact of donor cardiac arrest on cardiac transplantation on a larger scale.

Methods

The Institutional Review Board at Duke University Medical Center approved this study. Individual patient consent was deemed unnecessary.

Data source

The United Network for Organ Sharing (UNOS) Standard Transplant Analysis and Research files were used for this analysis; it contains data regarding every organ donation and transplant event occurring in the United States since October 1, 1987. Data are compiled from individual centers and are entered by trained data-entry personnel, with quality-assurance controls in place including electronic data validation systems and on-site audits of participating institutions.⁵ The dataset used for the current study comprises a prospectively collected open cohort of heart transplantations performed between October 1987 and December 2011, with follow-up through March 2012.

Study design

We performed a retrospective cohort analysis of all adult heart transplantations in the United States as recorded in the UNOS database. The study period included transplants performed from November 1999 (when cardiac-arrest information was first recorded) through December 2011. Multiorgan transplants, pediatric recipients, and patients undergoing repeat transplantation were excluded. Patients were categorized on the basis of whether the donors had histories cardiac arrest. In the UNOS database, this is defined as cardiac arrest after the neurologic event that led to a declaration of brain death. Patients with unknown or missing donor arrest information were excluded.

The primary predictor variable for our analysis was the occurrence of cardiac arrest in the donor. Additional covariates were included in risk-adjustment models that were determined a priori based on clinical characteristics previously established in the literature as impacting postoperative outcomes. These covariates were recipients' age; recipients' sex; sex mismatch; cause of heart failure; presence of diabetes; body mass index (kg/m^2); size mismatch (defined as body mass index difference $> 20\%$)⁶; serum creatinine at the time of transplant; pretransplant location (nonhospitalized, hospitalized, or intensive care unit); inotrope requirement at the time of transplant (yes/no); days on the waitlist; center volume (quantified as a continuous variable, defined as the number of heart or heart/lung transplants performed by the transplant center during the study period); year of transplant; total

ischemic time; donor age; donor blood urea nitrogen (mg/dL)/creatinine (mg/dL) ratio; and donor/recipient race mismatch.⁷

The primary outcome variable was perioperative and long-term overall survival. For patients in the donor-arrest group, a secondary analysis was performed to assess the association between duration of arrest time and post-transplant survival.

Statistical analysis

Baseline recipient and donor characteristics were described for the study population and stratified by the presence or absence of cardiac arrest in the donor, using medians and interquartile ranges or 25th/75th percentiles for continuous variables and proportions (frequency, percentage) for discrete variables. Comparisons of continuous and ordered categorical variables were made using Kruskal-Wallis tests, and unordered categorical variables were compared using the Pearson χ^2 test. Survival curves were constructed for each group using the Kaplan-Meier method, and comparisons were made using the log-rank test. Multivariable logistic regression was performed to assess the association between cardiac arrest in the donor and 30-day, 1-year, 3-year, and 5-year mortality, after adjusting for potential con-founders described above. Missing data for ischemic time ($n = 1,265/21,383$ [5.9%]) were imputed using multivariable regression based on the predictor variables of distance (in miles) from donor hospital to transplant center and the center's volume. Multivariable Cox proportional hazard modeling was used to assess the independent effect of donor cardiac arrest on cumulative risk for death, adjusting for potential confounders as described above. The proportionality assumption was verified by assessment of Schoenfeld residual plots.⁸

In donors who underwent cardiac arrest, a subanalysis of duration of arrest time was performed for the subset of cases in which this information was available ($n = 845/930$ [90.9%]). Survival curves were constructed by arrest time quartile using the Kaplan-Meier method and compared by the log-rank test. Cox proportional hazard modeling was performed to assess the independent effect of duration of arrest time (analyzed as a continuous variable) on cumulative risk for death, adjusting for potential confounders, as described above. In addition, the interaction between donor arrest time and total ischemic time was also assessed. The proportionality assumption was again verified by assessment of Schoenfeld residual plots.

A probability value of $P < .05$ was used to indicate statistical significance for all comparisons and analyses. Statistical analyses were performed using JMP v 10.0 (SAS Institute, Cary, NC) and R, v 2.15.1 (R Core Team, Vienna, Austria).

Results

A total of 21,383 heart transplant recipients were included in the analysis. Of these, 930 (4.3%) had received their allografts from donors who had undergone cardiac arrest (Donor Arrest), and 20,453 came from donors without histories of cardiac arrest (No Arrest). Patients excluded from the final study population are summarized in Figure 1. The median follow-up time was 3.7 years. The Donor Arrest group had a greater incidence of sex

mismatch (31% vs 27.6%, $P = .02$); race mismatch (46.5% vs 42.8%, $P = .03$); black race (20.8% vs 16.6%, $P = .01$); and intravenous inotropic support at the time of donor harvest (48.5% vs 43.3%, $P = .002$) compared to the No Arrest group. Donor Arrest recipients also had fewer incidences of ischemic heart disease (39.9% vs 44.4%, $P < .001$) and shorter times on the waitlist (median 72 vs 84 days, $P = .02$). There was no significant difference between groups with regard to age, sex, and comorbidity profile, including diabetes, hypertension, chronic obstructive pulmonary disease, cerebrovascular disease, or baseline renal function (Table I).

When compared to donors without histories of cardiac arrest, donors who had undergone cardiac arrest were more likely to have had diabetes (3.7% vs 2.4%, $P = .02$); higher terminal creatinine (median, interquartile range 1.1 [0.8, 1.6] vs 1.0 [0.8, 1.3], $P < .0001$); and higher incidence of cocaine use (16.7% vs 13.2%, $P = .002$) (Table II).

Using Kaplan-Meier methods, we found no significant difference in unadjusted survival between the No Arrest group and the Donor Arrest group (median 10.6 vs 10.2 years, respectively; $P = .9924$) (Fig 2). Based on multivariable logistic regression analyses, the Donor Arrest group demonstrated no significant difference in mortality at 30 days: adjusted odds ratio (AOR): 0.91, 95% confidence interval (CI): 0.65-1.28, $P = .58$; 1 year (AOR: 0.96, CI: 0.76-1.20, $P = .69$); 3 years (AOR: 1.03, CI: 0.84-1.26, $P = .77$); or 5 years (AOR: 0.97, CI: 0.79-1.21, $P = .82$) (Table III). Additionally, on multivariable adjusted Cox proportional hazard modeling, the Donor Arrest group demonstrated no significant difference in cumulative risk for death (adjusted hazard ratio, 1.02, CI: 0.89-1.16, $P = .82$). This relationship was found to be equivalent when the patients for whom ischemic time was imputed ($n = 1,265/21,383$ [5.9%]) were excluded from the analysis (data not shown).

Of the 930 recipients in the Donor Arrest group, information about the donor arrest time was available for 845 (90.9%) cases. The median interquartile range duration of arrest time was 15 (8, 25) min. The Donor Arrest cohort was divided into 4 groups based on duration of arrest time: quartile 1 (0 to 8 min); quartile 2 (9 to 15 min); quartile 3 (16 to 25 min); and quartile 4 (> 25 min). Unadjusted survival times were evaluated via the Kaplan-Meier method. Quartile 1 demonstrated significantly improved survival compared to quartile 2 ($P = .005$), quartile 4 ($P = .029$) (Fig 3), and the No Arrest group ($P = .036$) (Fig 4). Furthermore, for this subset of patients, multivariable Cox proportional hazard modeling demonstrated that increased duration of arrest time, as a continuous variable, was associated with an increased risk for death (adjusted hazard ratio per 5-min increase: 1.06, CI: 1.01-1.11, $P = .02$). Of note, the interaction between duration of arrest time and total ischemic time was not significant ($P = .63$).

Discussion

The purpose of this study was to determine the impact of donor cardiac arrest on both short- and long-term outcomes after orthotopic heart transplantation. There were no differences in either 30-day mortality or overall survival between recipients of hearts with or without histories of donor cardiac arrest. Additionally, we demonstrated that increasing duration of arrest time was significantly associated with decreasing survival.

Current outcomes after heart transplantation are excellent and continue to improve.⁹ The most critical issue facing cardiac transplantation is a donor shortage. Therefore, any efforts that can expand the donor pool will undoubtedly have a profound impact on the cardiac transplantation community. Donors with satisfactory hemodynamic and echocardiographic data and histories of cardiac arrest pose a special dilemma for the transplanting surgeon. It is difficult to ascertain whether the myocyte injury incurred during circulatory arrest is reversible or irreversible. This will continue to be a challenge. Data from this retrospective review support the notion that if standard donor and recipient transplantation criteria are met, a history of donor cardiac arrest should not preclude an organ for consideration for clinical transplantation. Widespread recognition of the safety of using organs from this donor population may allow for expansion of the donor pool.

The results of our study also imply that duration of arrest time is an important factor that should be considered during the evaluation process of donors with histories of cardiac arrest. As expected, increasing duration of arrest time was associated with decreased survival. We also demonstrated that recipients who received hearts from donors with short durations of arrest time, less than 8 min, had significantly greater unadjusted survival than recipients in the No Arrest group. This is a conceptually important finding.

The most likely explanation is ischemic preconditioning. Initially described by Murry and colleagues, in 1986, as a phenomenon in which brief periods of ischemia followed by reperfusion prior to a sustained ischemic event lead to (1) a delay in ATP depletion; (2) preservation of intracellular structure; (3) decrease in oxygen consumption; and (4) delay and/or reduction in cellular necrosis despite an increase in total ischemic time.¹⁰ Furthermore, the strength of cardioprotection depends heavily on the duration of time from the end of preconditioning ischemia to the onset of index ischemia. A window of 24 to 72 hours is necessary for genomic modulation and new protein expression.¹¹ Unfortunately, time between the return of spontaneous circulation in the donor after cardiac arrest and the application of the aortic cross-clamp was not available for this analysis, but anecdotally, many donors probably fall into this 24- to 72-h window. Therefore, a donor history of short cardiac arrest, less than 8 min, may offer cardioprotection to the ensuing ischemia associated with the procurement, cold preservation, and transportation of clinical heart transplantation.

The results of this investigation should be interpreted in the context of the limitations of this study. First, this was an observational study and therefore, allocation to each respective group was not randomized. Modeling techniques do not account entirely for the lack of randomization among cohorts, and the impact of unmeasured confounders is not known. Imbalances in the baseline characteristics among groups were present and may have affected our results. Second, clinicians considering a donor with a history of cardiac arrest may have utilized additional diagnostic information, such as cardiac biomarkers or may have incorporated recipient variables into their decision to accept or decline the heart. Furthermore, the quantity and quality of hearts that were not utilized for transplant cannot be determined on the basis of this review. Additionally, the precise time of donor cardiac arrest is difficult to ascertain from the UNOS database, which defines it as a cardiac arrest event occurring after the neurologic insult that led to a declaration of brain death. This definition excludes donors with cardiac arrest immediately prior to or synchronous with their

neurologic injuries. Last, outcome measures other than survival, such as quality of life, were not analyzed.

This review demonstrates that hearts from donors with histories of cardiac arrest can be successfully transplanted with excellent outcomes in selected cases. The ultimate decision to accept or decline a heart should be based on the clinical judgment of an experienced clinician after thoughtful consideration of both donor and recipient selection criteria.

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Fig 1.
Study inclusion algorithm.

Fig 2.
Unadjusted Kaplan-Meier survival curves by donor cardiac arrest status.

Fig 3.
 Unadjusted Kaplan-Meier survival curves by resuscitation time quartile.

Fig 4. Unadjusted Kaplan-Meier survival curves comparing no donor arrest and first quartile of resuscitation time.

Table I
Baseline recipient characteristics

Characteristic	Total sample (n = 21,383)	Donor cardiac arrest status		P value
		No arrest (n = 20,453)	Donor arrest (n = 930)	
Age	55 (46, 61)	55 (46, 61)	55 (45, 62)	.5818
Age 60	6,819 (31.9%)	6,498 (31.8%)	321 (34.5%)	.0789
Female gender	5,142 (24.1%)	4,899 (24.0%)	243 (26.1%)	.1288
Gender mismatch	5,926 (27.7%)	5,638 (27.6%)	288 (31.0%)	.0234*
Race				.0124*
White	15,541 (72.7%)	14,889 (72.8%)	652 (70.1%)	
Black	3,593 (16.8%)	3,400 (16.6%)	193 (20.8%)	
Hispanic	1,528 (7.2%)	1,466 (7.2%)	62 (6.7%)	
Asian	507 (2.4%)	492 (2.4%)	15 (1.6%)	
Other/unknown	214 (1.0%)	206 (1.0%)	8 (0.9%)	
Race mismatch between donor and recipient	9,175 (42.9%)	8,743 (42.8%)	432 (46.5%)	.0256*
Etiology of heart failure				.0006*
Ischemic	9,445 (44.2%)	9,074 (44.4%)	371 (39.9%)	
Hypertrophic	417 (2.0%)	392 (1.9%)	25 (2.7%)	
Idiopathic	7,394 (34.6%)	7,077 (34.6%)	317 (34.1%)	
Congenital	553 (2.6%)	532 (2.6%)	21 (2.3%)	
Valvular	468 (2.2%)	450 (2.2%)	18 (1.9%)	
Other	3,104 (14.5%)	2,926 (14.3%)	178 (19.1%)	
Recipient comorbidities				
Diabetes	4,963 (23.5%)	4,745 (23.5%)	218 (23.5%)	.9773
Hypertension	4,856/12,155 (40.0%)	4,693/11,752 (39.9%)	163/403 (40.4%)	.8362
Chronic obstructive pulmonary disease	432/12,290 (3.5%)	414/11,883 (3.5%)	18/407 (4.4%)	.3120
Cerebrovascular disease	484/12,279 (3.9%)	471/11,873 (4.0%)	13/406 (3.2%)	.4360
Creatinine at transplant	1.2 (1.0, 1.5)	1.2 (1.0, 1.5)	1.2 (1.0, 1.5)	.6048
BMI (kg/m ²) at transplant	26.3 (23.2, 29.6)	26.3 (23.2, 29.6)	26.6 (23.5, 30.1)	.0154*
Pre-transplant status				.1304
Hospitalized	3,929 (18.4%)	3,737 (18.3%)	192 (20.7%)	
Intensive care unit	6,265 (29.4%)	5,990 (29.4%)	275 (29.7%)	
Life support at time of transplant				
Intravenous inotropes	9,307 (43.5%)	8,856 (43.3%)	451 (48.5%)	.0018*
Ventilator	564 (2.6%)	539 (2.6%)	25 (2.7%)	.9216
Intra-aortic balloon pump	1,120 (5.2%)	1,060 (5.2%)	60 (6.5%)	.0893
Ventricular assist device	5,604/17,049 (32.9%)	5,334/16,228 (32.9%)	270/821 (32.9%)	.9916
Hemodynamics at time of transplant				
Mean PA pressure (mm/Hg) (n = 18,452)	28 (21, 35)	28 (21, 35)	28 (21, 35)	.7327
PVR (Wood units) (n = 16,563)	2.1 (1.4, 3.0)	2.1 (1.3, 3.0)	2.1 (1.4, 2.9)	.5868
Cardiac index (L/min/m ²) (n = 18,047)	2.2 (1.8, 2.7)	2.2 (1.8, 2.7)	2.2 (1.8, 2.7)	.6515

Characteristic	Total sample (n = 21,383)	Donor cardiac arrest status		P value
		No arrest (n = 20,453)	Donor arrest (n = 930)	
Peak PRA class 1 (n = 2,726)	21 (7, 51)	21 (7, 51)	18 (6,43)	.3121
Peak PRA class 2 (n = 1,437)	20 (6, 55)	20 (6, 56)	17 (6,47)	.3485
Recipient blood type				.3171
A	10,092 (47.2%)	9,663 (47.2%)	429 (46.1%)	
B	2,998 (14.0%)	2,878 (14.1%)	120 (12.9%)	
O	8,293 (38.8%)	7,912 (38.7%)	381 (41.0%)	
Days on waitlist	83 (25, 236)	84 (25, 236)	72 (23, 229)	.0227*

*
P < .05.

Median (interquartile range) for non parametric continuous variables. N (%) for categorical variables. If data is missing for >5% of the study population, the denominator is give for categorical variables and “n” given for continuous variables. Wilcoxon signed-rank test for continuous variables. Pearson Chi-Square test for categorical variables.

BMI, Body mass index; PA, pulmonary artery; PVR, pulmonary vascular resistance; PRA, panel reactive antibody.

Table II
Baseline donor/graft characteristics

Characteristic	Total sample (<i>n</i> = 21,383)	Donor cardiac arrest status		<i>P</i> value
		No arrest (<i>n</i> = 20,453)	Donor arrest (<i>n</i> = 930)	
Donor/graft characteristics				
Donor age	29 (21, 42)	29 (21, 42)	29 (21, 40)	.3335
Donor diabetes	526 (2.5%)	492 (2.4%)	34 (3.7%)	.0163*
Terminal creatinine	1.0 (0.8, 1.3)	1.0 (0.8, 1.3)	1.1 (0.8, 1.6)	<.0001*
Donor BMI (kg/m ²)	25.6 (22.8, 29.1)	25.6 (22.8, 29.1)	26.0 (22.8, 30.2)	.0007*
Donor/recipient size mismatch [†]	7,115 (34.5%)	6,787 (34.4%)	328 (36.5%)	.1812
Donor cigarette use	5,089 (24.0%)	4,896 (24.1%)	193 (20.9%)	.0262*
Donor cocaine use	2,793 (13.3%)	2,641 (13.2%)	152 (16.7%)	.0021*
Donor history of alcohol abuse	2,046/13,145 (15.6%)	1,956/12,467 (15.7%)	90/678 (13.3%)	.0912
HLA mismatch level				.9705
0	24/18,160 (0.1%)	23/17,364 (0.1%)	1/796 (0.1%)	
1	90/18,160 (0.5%)	87/17,364 (0.5%)	3/796 (0.4%)	
2	551/18,160 (3.0%)	527/17,364 (3.0%)	24/796 (3.0%)	
3+	17,495/18,160 (96.3%)	16,727/17,364 (96.3%)	768/796 (96.5%)	
CMV mismatch	4,445/19,668 (22.6%)	4,273/18,815 (22.7%)	172/854 (20.1%)	.0792
Clinical infection in donor	8,077 (39.6%)	7,645 (39.2%)	432/883 (48.9%)	<.0001*
Vasopressin within 24 hrs of procurement	7,860/13,304 (59.1%)	7,464/12,617 (59.2%)	396/687 (57.6%)	.4312
Inotropic medications at procurement	8,646/15,405 (56.1%)	8,199/14,632 (56.0%)	447/773 (57.8%)	.3278
Diuretics within 24 hrs of procurement	11,829 (57.8%)	11,260 (57.5%)	569 (63.2%)	.0008*
Left ventricular EF (<i>n</i> = 598; 4,870)	60 (55, 65)	60 (55, 65)	60 (55, 65)	.0532
Ischemic time (hours) (<i>n</i> = 20,118)	3.2 (2.5, 3.9)	3.2 (2.5, 3.9)	3.2 (2.5, 3.8)	.5526

* $P < .05$.

[†] Donor/recipient size mismatch is defined as a difference in body mass index of > 20%.

Median (interquartile range) for non parametric continuous variables. N (%) for categorical variables. If data is missing for >5% of the study population, the denominator is give for categorical variables and "n" given for continuous variables. Wilcoxon signed-rank test for continuous variables. Pearson Chi-Square test for categorical variables.

BMI, Body mass index; CMV, cytomegalovirus; EF, ejection fraction; HLA, human leukocyte antigen.

Table III
Multivariate analysis of survival endpoints

		95% confidence interval		
Donor cardiac arrest prediction of mortality	Adjusted odds (hazard) ratio	Lower	Upper	<i>P</i> value
Multivariable logistic regression				
30-day mortality	0.90	0.64	1.27	.5510
1-year mortality	0.95	0.76	1.19	.6462
3-year mortality	1.02	0.83	1.25	.8473
5-year mortality	0.97	0.78	1.20	.7815
Multivariable Cox proportional hazard				
Cumulative risk of death	1.01	0.88	1.16	.8878

Adjusted for recipient age, gender, gender mismatch, etiology of heart failure (ischemic versus non-ischemic disease), diabetes, body mass index, size mismatch, creatinine at the time of transplant, pre transplant location (non-hospitalized, hospitalized, or intensive care unit), inotrope requirement at the time of transplant, days on the waitlist, center volume, year of transplant, ischemic time, donor age, donor BUN/creatinine ratio, donor/recipient race mismatch.