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Electrocardiographic Characteristics of Potential Organ Donors and Associations with Cardiac Allograft Utilization

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Abstract

Background—Current regulations require that all cardiac allograft offers for transplantation must include an interpreted 12-lead electrocardiogram (ECG). However, little is known about the expected ECG findings in potential organ donors, or the clinical significance of any identified abnormalities in terms of cardiac allograft function and suitability for transplantation.

Methods and Results—A single experienced reviewer interpreted the first ECG obtained after brainstem herniation in 980 potential organ donors managed by the California Transplant Donor Network from 2002-2007. ECG abnormalities were summarized, and associations between specific ECG findings and cardiac allograft utilization for transplantation were studied. ECG abnormalities were present in 51% of all cases reviewed. The most common abnormalities included voltage criteria for left ventricular hypertrophy (LVH), prolongation of the corrected QT interval (QTc), and repolarization changes (ST/T wave abnormalities). Fifty seven percent of potential cardiac allografts in this cohort were accepted for transplantation. LVH on ECG was a strong predictor of allograft non-utilization. No significant associations were seen between QTc prolongation, repolarization changes and allograft utilization for transplantation, after adjusting for donor clinical variables and echocardiographic findings.

Conclusions—We have performed the first comprehensive study of ECG findings in potential donors for cardiac transplantation. Many of the common ECG abnormalities seen in organ donors may result from the heightened state of sympathetic activation that occurs after brainstem herniation, and are not associated with allograft utilization for transplantation.

Keywords

electrocardiography; echocardiography; organ donor; long QT; transplantation

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Disclosures
None.

Organ Procurement Transplant Network (OPTN) regulations require that all cardiac allograft offers must include, among other data, an interpreted 12-lead electrocardiogram (ECG, OPTN policy 3.7.12.1). However, little is known about the expected ECG findings in potential organ donors, or the clinical significance of any identified abnormalities in terms of cardiac allograft function and suitability for cardiac transplantation. ST segment elevation or depression, T wave inversion, a prolonged QT interval, abnormal U waves, and voltage criteria for left ventricular hypertrophy (LVH) have been observed in patients with subarachnoid hemorrhage and traumatic brain injury—two common causes of brain death in potential organ donors.[1-6] Small studies comparing donor and recipient ECGs suggest reversal of pathological findings after transplantation, such as shortening of the QT interval[7] and reduction in voltage in the precordial leads,[8] suggesting that at least some ECG changes noted after brain death may be transient, and of little prognostic significance for allograft function and post-transplant outcomes.

Large working groups have attempted to standardize cardiac allograft acceptance criteria in terms of donor echocardiogram findings, and to clarify indications for pulmonary artery catheter use and hormonal therapy,[9] but the role of the ECG in donor evaluation has not yet been formally evaluated. The purpose of this study was to (1) describe ECG findings in a large, contemporary cohort of brain dead organ donors, (2) explore the relationship between donor ECG and echocardiogram findings, and (3) explore the relationship between ECG findings and cardiac allograft utilization.

Methods

Approval for this study was obtained from the California Transplant Donor Network (CTDN) Institutional Review Board. The medical records of all brain dead organ donors managed by CTDN between January 1, 2002 and December 31, 2007 were retrospectively reviewed for the first 12-lead ECG obtained after brainstem herniation. Donors less than 14 years and over 65 years of age were excluded, as their hearts were unlikely to be used for adult heart transplantation. Standard demographic data (e.g. sex, age, height, weight, cause of death), clinical data (laboratory values, inotrope use, echocardiogram findings), and data on cardiac allograft utilization were obtained from chart review.

Donor management

During the six-year time period studied, all brain dead organ donors at CTDN were managed according to a standardized protocol that included: Methylprednisolone administered at the onset of donor management and until organ procurement (15mg/kg every 12 hours); dopamine as the first-line inotropic agent (maximum 20 mcg/kg/min); phenylephrine as the second-line vasoactive agent (maximum 300 mcg/min); intravenous fluid and/or loop diuretic administration to obtain a goal central venous pressure of 5-8 mmHg and a urine output of > 30ml/hr; electrolyte repletion to achieve normalization of potassium, phosphorous, magnesium and calcium levels; empiric antimicrobial therapy with vancomycin and levofloxacin; and inhaled, nebulized albuterol (2.5 mg every four hours). Vasoactive and inotropic medications were titrated according to pulmonary artery catheter readings to achieve a target systemic vascular resistance of 800-1200 dynes-seconds/cm^5 and cardiac index >2 l/min/m^2 . Esmolol infusions were initiated for tachycardia that was deemed unrelated to beta-agonist infusion and were discontinued upon initiation of organ procurement. Thyroid hormone (levothyroxine) was administered when requested by the accepting transplant centers.

ECG interpretation

All donor 12-lead ECGs were read and interpreted by a single experienced reviewer (B.D.). This reviewer was blinded to the donor's clinical data except for age, sex, and potassium level at the time of ECG procurement. Standard ECG criteria were used to diagnose cardiac rhythm, atrial and ventricular ectopy, right and left bundle branch block, anterior and posterior fascicular block, and right and left atrial and ventricular hypertrophy. Q waves of prior myocardial infarction and ST-T wave abnormalities indicative of acute myocardial injury were defined by the Joint European Society of Cardiology and American College of Cardiology universal criteria for myocardial infarction.[10] These criteria included: (a) ST-segment elevation at the J-point with cutoff points of ≥0.2 mV in men or ≥0.15 mV in women in leads V_2-V_3 and/or ≥0.1 mV in other leads; (b) horizontal or down-sloping ST-segment depression of ≥0.1 mV; and (c) T wave inversion of ≥0.1 mV. If any of these ECG criteria were present in 2 contiguous leads, a diagnosis of acute myocardial injury/infarction was made. Contiguity in the limb leads was defined by the Cabrera sequence of aVL, I, inverted aVR, II, aVF, and III.

Statistical Analysis

Donor ECG characteristics were summarized as means (± standard deviation) or percentages. Comparisons of ECG findings between transplanted and non-transplanted hearts were performed using Student's t-test for continuous variables and the chi-squared test for categorical variables. Multivariable logistic regression analyses were used to explore associations between donor ECG findings and cardiac allograft acceptance for transplantation, adjusting for donor age, sex, cause of death, race, height, blood type, and diagnosis of hypertension, diabetes, or coronary artery disease.

For each donor ECG, ST segments, T waves, and Q waves were defined as abnormal if an abnormality was seen in one or more of the 12 leads. We then tested for associations between these ECG abnormalities and allograft utilization in a series of three models (1) a simple univariate model, (2) a multivariable logistic regression model adjusting for donor demographic variables that may impact graft utilization decisions (age, sex, race, and cause of death), and (3) a multivariable logistic regression model that added echocardiographic abnormalities (LV dysfunction, regional wall motion abnormalities, and LVH).

Statistical analyses were performed using Stata version 9 (StataCorp LP, College Station, TX).

Results

A total of 1,569 donors were managed by CTDN between January 1, 2002 and December 31, 2007, and 1,085 had stored ECGs available for analysis. Fourteen donors were excluded as their ECGs were of poor quality or had missing leads (e.g. 2-3 lead rhythm strips only). After excluding 68 donors<14 years and 21 donors>65 years of age, 980 ECGs were included in the final study cohort. There were 391 donors in the overall CTDN cohort who were 14-65 years of age and did not have stored ECGs available for interpretation. These donors were older, and had a higher incidence of hypertension, diabetes, and coronary artery disease compared to donors with ECGs (Supplementary Table). They were less likely to receive hormonal therapy during the donor management period, and their hearts were less likely to be utilized for transplantation.

Donor cohort

The characteristics of the donor cohort are summarized in Table 1. Mean donor age was 38 ± 14 years, and 63% were male. The most common causes of death were cerebrovascular

(including subarachnoid hemorrhage and ischemic stroke, 47%), followed by head trauma (43%), and anoxia (9%). Twenty-six percent of donors had a history of hypertension, and 28% had a history of cocaine or methamphetamine use. One-third of donors had an elevated serum troponin level, defined in this study as a peak level ≥ 1.0 mcg/L, given the variety of assays (sandwich and immunoenzymatic) from multiple manufacturers used at different donor hospitals.

Ninety-three percent of donors in this cohort had at least one echocardiogram, and 16.5% had one or more additional echocardiograms, based on the discretion of the treating clinician. Associations between the ECG and first donor echocardiogram were studied. The median time elapsed between the ECG and echocardiogram was 98 minutes (IQR 29, 498). T he mean left ventricular ejection fraction (LVEF) was 62% ± 12%. Slightly more than half of donors had left ventricular hypertrophy (defined as LV septal or posterior wall thickness > 1.1 cm) and 20% had LV regional wall motion abnormalities.

When comparing clinical characteristics of donors whose hearts were or were not accepted for transplantation, we found that donors who died of cerebrovascular causes, who had a history of hypertension, diabetes, or coronary artery disease, or who had an elevated serum troponin level were less likely to be cardiac organ donors.

Characteristics of donor electrocardiograms

Electrocardiographic findings after brain death are summarized in Table 2. Mean heart rate was 102 ± 20 bpm, and 97% were in sinus rhythm. One or more ECG abnormalities were present in 51% of the ECGs studied. Atrial and ventricular ectopy were rare, as were atrioventricular block, conduction delays (including right and left bundle branch block), and fascicular block.

A notable finding was prolongation of the corrected QT interval: the mean QTc was 449 ± 48 msec, while 21% of donors had QTc>480 msec and 15% had QTc>500 msec. QT prolongation was significantly associated with cause of death: 28% of donors who died of cerebrovascular causes had a QTc>480 msec, compared to 23% of donors who died from anoxia, 20% of those who died from CNS tumors, and 14% of those who died from head trauma ($p<0.001$). This finding was more common in female donors (OR 2.7, 95% CI 2.0-3.7, $p<0.001$) compared to males. Among donors dying of cerebrovascular causes, 38% of females had a QTc>480 msec, and 28% had QTc>500 (versus 19% and 13% for males, respectively, $p<0.001$). Prolongation of the QTc interval was associated with lower serum potassium levels. The mean serum potassium level was 4.0 ± 0.6 mmol/L in donors with QTc<480 msec and 3.7 ± 0.5 mmol/L in donors with QTc≥480 msec ($p<0.0001$).

Also of note was the high prevalence of voltage criteria for LVH, present in 8% of potential organ donors. This finding was significantly more common in donors who died of cerebrovascular causes (14.7%), compared to those who died of head trauma (2.6%) or anoxia (1.1%), $p<0.001$, even after adjusting for donor history of hypertension (OR 10.9, 95% CI 1.5-80.5, $p=0.02$).

Finally, repolarization changes were present in 22% of the donor ECGs examined. Two percent of donor ECGs met criteria for pathologic ST elevations, 10% of donor ECGs demonstrated significant ST depressions, and 12% had significant T wave inversions, while 18% had non-specific ST-T wave abnormalities. Q waves suggestive of prior myocardial infarction were found in 7% of donor ECGs. Overall, 51% of donor ECGs were classified as “abnormal” due to one or more of the above findings.

Correlations between donor ECG and echocardiographic findings

Left ventricular hypertrophy was present on 8% of donor ECGs and 54% of echocardiograms. Given this disparity, LVH on ECG was found to have high specificity (97%) for increased LV wall thickness, but low sensitivity (11%). The presence of LVH on ECG increased the odds of increased LV wall thickness by 3.5-fold (95% CI 1.9-6.6, $p<0.001$).

The finding of an elevated serum troponin level ≥1.0 mcg/L was not associated with repolarization abnormalities on ECG, as defined by the presence of significant ST elevations, ST depressions, or T wave inversions. Specifically, having an elevated troponin level increased the odds of repolarization abnormalities by only 1.3-fold (95% CI 0.9-1.7, $p=0.2$) and had a sensitivity of 38% and a specificity of 67% for the presence of significant ST-T wave abnormalities.

Finally, the presence of pathologic Q waves on ECG had a high specificity for reduced LV ejection fraction (defined as LVEF<50%, specificity=97%) and LV regional wall motion abnormalities (RWMA, specificity=96%), albeit sensitivity was low (12% for reduced LVEF, 15% for RWMA).

Donor ECG findings and cardiac allograft utilization for heart transplantation

Fifty seven percent (N=560) of donor allografts in this cohort were accepted for heart transplantation. The results of multivariable analyses examining associations between donor ECG predictors and cardiac allograft utilization are presented in Table 3. These models were adjusted for donor age, sex, cause of death, blood type, race, height, and diagnosis of hypertension, diabetes, and coronary artery disease. Our analyses demonstrate that prolongation of the PR and QRS intervals are associated with decreased allograft utilization. Specifically, for every 10 msec increase in the PR interval, the odds of allograft utilization decreases by 10% (OR 0.9, 95% CI 0.84-0.98, $p=0.01$). Similarly, for every 10 msec increase in the QRS interval, the odds of allograft utilization decreases by 18% (OR 0.82, 95% CI 0.72-0.93, $p=0.002$). Notably, prolongation of the QT interval was not associated with reduced allograft utilization, after adjusting for relevant covariates.

As a general category, repolarization abnormalities on ECG (defined as the presence of pathologic ST elevations, ST depressions, and/or T wave inversions) were not associated with reduced allograft utilization; however, changes in individual leads did reveal significant associations. Specifically, ST segment changes in leads I, V1, V2, and V3 and T wave inversions in leads I, II, and aVR were associated with reduced allograft utilization. Finally, the presence of pathologic Q waves in leads V1 and V2, suggestive of prior anteroseptal myocardial infarction, were also associated with reduced allograft utilization. When grouped by the presence of any ST segment, T wave, or Q wave abnormality on the 12-lead ECG, pathologic Q waves and T wave inversions were associated with allograft non-utilization in unadjusted models. However, after adjusting for donor demographic variables these results were attenuated towards the null, with only pathologic Q waves remaining associated with non-utilization. Finally, after adjusting for echocardiographic abnormalities (left ventricular ejection fraction<50%, regional wall motion abnormalities, and LVH), no significant associations between ECG abnormalities and allograft utilization remained (Table 4).

Finally, voltage criteria for LVH on the 12-lead ECG was a strong predictor of allograft non-utilization. Specifically, the presence of LVH reduced the odds of graft acceptance for transplantation by 77% (univariate OR 0.23, 95% CI 0.13-0.38, $p<0.001$). This association remained highly significant after adjusting for donor demographic variables and echocardiographic abnormalities (multivariate OR 0.36, 95% CI 0.18-0.72).

Discussion

We have presented the first large-scale study describing ECG characteristics after brain death in potential organ donors. Using a well-characterized cohort of almost 1,000 potential donors, we have described typical ECG findings, correlations between abnormalities seen on ECG and echocardiography, and associations between ECG findings and cardiac allograft utilization for transplantation. A notable finding of this study was the relatively high proportion of donor ECGs that met voltage criteria for LVH. This abnormality was found in 8% of all donor ECGs, and 10% of the ECGs of donors aged 30-39 years. In contrast, only 2.7% of healthy men aged 30-39 years enrolled in the Manitoba Heart Study, a prospective cohort study of cardiovascular disease in Canadian air force pilots, demonstrated ECG voltage criteria for LVH.[11] Similarly, at most 1.3% of men aged 35-39 years in the original Framingham Heart Study had “definite” LVH and another 3.4% had “possible” LVH on ECG.[12] We hypothesize that many brain dead organ donors who meet voltage criteria for LVH may actually have transient myocardial edema resulting from the dramatic physiologic changes that occur after brain death. The physiologic changes after brain death have been well described, and likely represent a multi-factorial process resulting from activation of the sympathetic nervous system, diffuse loss of vasomotor tone, endothelial dysfunction, and hormone depletion.[13, 14] Classic baboon studies have demonstrated that the initial Cushing reaction that accompanies brainstem herniation results in direct myocardial injury. Within minutes after brain death, an “autonomic storm” occurs[15] in which serum epinephrine levels increase by 1,100%, norepinephrine by 300%, and dopamine by 200%.[16, 17] These processes result in interstitial myocardial edema[18] that may mimic myocardial hypertrophy.[19] Fortunately, these myocardial changes are often transient,[20] suggesting that the presence of LVH on donor ECGs does not necessarily represent pathologic left ventricular remodeling and should not, in and of itself, exclude a graft from acceptance for transplantation.

Another common feature of donor ECGs was that of a prolonged QTc interval. Etiologies for QTc prolongation in this setting include sympathetic stimulation and autonomic dysregulation, especially since the autonomic nervous system is an important modulator of ventricular repolarization.[21] Another contributing factor may be hypokalemia, which is often observed after brain death, and may be due to catecholamine-induced stimulation of a β-adrenergic receptor linked to membrane Na+/K+-ATPase[22, 23] or acquired diabetes insipidus.[24, 25] In this study, we did find significantly lower serum potassium levels in donors with QTc prolongation. QTc prolongation in this cohort was not associated with reduced allograft utilization or adverse recipient outcomes after transplantation. Of interest is a study performed on 112 heart transplant donor:recipient pairs demonstrating shortening of the QTc interval after transplantation.[7] An exception worth mentioning may be cases of genetic long QT syndromes. These cases may be identified by markedly prolonged QTc intervals pre-transplant in donors with an unexplained mechanism of death. In such a scenario, the transplant recipient may be at heightened risk of cardiac arrhythmias.

A final common feature of donor ECGs is that of repolarization abnormalities, which include significant ST elevations, ST depressions, and T wave inversions. These findings were present in approximately 20% of donor ECGs, which is higher than one may expect given the relatively young age and lack of cardiovascular disease in the general organ donor population. Once again, these ECG changes may reflect the exaggerated state of sympathetic activity that occurs after brainstem herniation that may result in direct myocardial injury. Endomyocardial biopsy specimens in this setting have shown contraction band necrosis, or histologic evidence of microinfarction secondary to catecholamine-mediated calcium overload.[16] In some cases, these physiologic changes may result in frank left ventricular dysfunction and elevated serum troponin levels.[20, 26, 27] Prior studies, however, have demonstrated reversibility of left ventricular dysfunction during tailored donor

management,[20, 28] and a lack of association between elevated donor troponin levels and recipient post-transplant outcomes.[27] These studies, among others, suggest that ST-T wave changes should not preclude acceptance of a cardiac allograft for transplantation. It may, in fact, be prudent to repeat the ECG after a period of hemodynamic stability. Caution should be taken, however, when Q waves consistent with prior myocardial infarction are seen on ECG, as this finding has high specificity for the presence of left ventricular dysfunction and regional wall motion abnormalities on echocardiography. While we were unable to retrospectively determine the cause of allograft non-utilization in this study, we did demonstrate that ECG abnormalities were no longer predictive of utilization after adjusting for echocardiographic abnormalities; this finding suggests that the echocardiogram, when available, plays a larger role in allograft acceptance decisions.

Several limitations of this study deserve mention. We analyzed the first donor ECG obtained after brainstem herniation. The time interval between brainstem herniation and ECG acquisition ranged from minutes to several hours; this may represent an important confounder, as the donor physiologic state after herniation may change significantly over time. We also know that ECG changes are often dynamic, and may be influenced by concomitant medications and treatments. For example, administration of QT-prolonging drugs (such as quinolone antibiotics and antiarrhythmics) may have accounted for some cases of QT prolongation seen in this cohort. Another limitation is the lack of data on the reason for allograft non-utilization. Cardiac allograft acceptance for transplantation is a complex decision in which multiple donor and recipient factors are weighed by the accepting physician or surgeon. We are unable to retrospectively determine the extent to which the donor ECG influenced individual decisions. Finally, donor echocardiograms were interpreted at local hospitals and were not centrally reviewed; we therefore cannot verify the accuracy of echocardiogram interpretation and measurements.

Conclusions

Abnormal ECG findings are common after brain death, and are present in over half of potential organ donors. We combined the strengths of a well-characterized cohort of 980 organ donors with central ECG interpretation to describe common ECG abnormalities after brain death and to explore the associations between these findings and allograft utilization. The predominant ECG abnormalities identified may result from the massive sympathetic activation that occurs after brainstem herniation and in most cases are not associated with allograft utilization for transplantation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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CLINICAL PERSPECTIVE

Current regulations require organ procurement organizations to obtain a 12-lead electrocardiogram (ECG) on all potential cardiac organ donors. However, little is known about expected ECG findings after brain death, which represents a unique physiologic state of massive sympathetic activation, nor about the association between ECG changes and graft acceptance by the recipient team. We reviewed the first ECG obtained after brainstem herniation in a cohort of 980 potential organ donors managed by the California Transplant Donor Network from 2002-2007 in order to describe common ECG findings in organ donors and to explore the relationship between ECG abnormalities, echocardiographic findings, and graft acceptance for heart transplantation. We determined that abnormal ECG findings were present in over half of potential organ donors. Voltage criteria for left ventricular hypertrophy (LVH), prolongation of the corrected QT interval (QTc), and repolarization changes (ST/T wave abnormalities) were common. LVH on ECG had a low sensitivity (11%) but high specificity (97%) for increased LV wall thickness on echocardiogram and predicted non-utilization of the donor heart for transplantation (OR 0.23, $p<0.001$). QT interval prolongation and repolarization changes were not associated with graft utilization. In summary, ECG abnormalities are common in the organ donor population. In many cases these abnormalities may reflect physiologic changes that occur after brain death

Table 1

Donor Characteristics

	All donors	Transplanted hearts	Non-Transplanted hearts	p-value*
	n=980	n=560	n=420	
Demographics				
Age (years)	38 ± 14	32 ± 13	45 ± 12	<0.0001
Sex (Male)	617 (63%)	394 (70%)	223 (53%)	<0.001
Cause of death				<0.0001
Anoxia	92 (9%)	48 (9%)	44 (11%)	
Cerebrovascular	455 (47%)	177 (32%)	278 (66%)	
Head trauma	424 (43%)	329 (59%)	95 (23%)	
Central nervous system tumor	5 (0.5%)	3 (0.5%)	2 (0.5%)	
Other	3 (0.3%)	2 (0.4%)	1 (0.2%)	
Race				0.01
Caucasian	536 (55%)	295 (53%)	241 (57%)	
Hispanic	249 (25%)	165 (30%)	84 (20%)	
African-American	109 (11%)	61 (11%)	48 (11%)	
Asian	59 (6%)	26 (5%)	33 (8%)	
Other	27 (3%)	13 (2%)	14 (3%)	
Height (cm)	171 ± 11	173 ± 10	169 ± 11	<0.0001
Weight (kg)	79 ± 20	80 ± 18	79 ± 22	0.7
Clinical history				
Cardiopulmonary Resuscitation	203 (21%)	111 (20%)	92 (22%)	0.4
Defibrillation	66 (7%)	33 (6%)	33 (8%)	0.2
Smoking	526 (55%)	290 (53%)	236 (57%)	0.2
Cocaine/ Amphetamines	255 (28%)	148 (28%)	107 (28%)	0.8
Hypertension	252 (26%)	82 (15%)	170 (41%)	<0.001
Diabetes	63 (7%)	19 (4%)	44 (11%)	<0.001
Coronary artery disease	26 (3%)	5 (1%)	21 (5%)	<0.001
Laboratory values				
Troponin (peak) ≥ 1.0 mcg/L	312 (34%)	163 (31%)	149 (38%)	0.03
Vasoactive Medications				
Dopamine (mcg/kg/min)	811 (83%)	458 (82%)	343 (84%)	0.4
Peak dopamine	6.3 ± 4.9	6.2 ± 4.8	6.5 ± 5.0	0.4
Final dopamine	2.1 ± 2.2	2.0 ± 1.8	2.1 ± 2.6	0.4
Neosynephrine (mcg/min)	801 (82%)	465 (83%)	336 (80%)	0.2
Peak neosynephrine	107 ± 93	105 ± 90	111 ± 97	0.3
Final neosynephrine	27 ± 41	24 ± 35	30 ± 47	0.04
Epinephrine	38 (4%)	22 (4%)	16 (4%)	0.9
Norepinephrine	51 (8%)	35 (10%)	16 (7%)	0.2
Esmolol	190 (21%)	109 (21%)	81 (21%)	0.9
Hormonal therapy				

	All donors	Transplanted hearts	Non-Transplanted hearts	p-value*
	n=980	**n=560**	**n=420**	
Corticosteroids	977 (99%)	559 (100%)	418 (99%)	0.1
Methylprednisolone (per 24 hrs)	2.2 ± 1.0	2.1 ± 0.9	2.3 ± 1.0	0.01
Thyroxine	189 (21%)	116 (22%)	73 (19%)	0.2
Echocardiogram				
Echocardiogram performed	899 (93%)	549 (99%)	350 (85%)	<0.001
Left ventricular ejection fraction (%)	62 ± 12	64 ± 9	58 ± 15	<0.0001
Regional wall motion abnormalities	175 (20%)	72 (13%)	103 (30%)	<0.001
Left ventricular hypertrophy†	453 (54%)	254 (49%)	199 (63%)	<0.001

*transplanted vs non-transplanted hearts

†septal or posterior wall thickness > 1.1 cm

Table 2

Characteristics of Donor Electrocardiograms

	All donors	Transplanted hearts	Non-Transplanted hearts	p-value*
	n=980	n=560	n=420	
Rate	102 ± 20	103 ± 18	100 ± 21	0.01
Sinus rhythm	952 (97%)	545 (97%)	408 (97%)	0.9
Chamber enlargement				
Atrial enlargement				0.1
None	846 (88%)	495 (90%)	351 (86%)	
Left	73 (8%)	34 (6%)	39 (10%)	
Right	29 (3%)	17 (3%)	12 (3%)	
Both	9 (1%)	3 (1%)	6 (2%)	
Left ventricular hypertrophy	79 (8%)	20 (4%)	59 (14%)	<0.001
Fascicular block				0.09
None	955 (98%)	549 (99%)	406 (97%)	
Left anterior fascicular block	14 (1%)	4 (1%)	10 (2%)	
Left posterior fascicular block	3 (0.3%)	2 (0.4%)	1 (0.2%)	
Atrioventricular Block				0.01
None	964 (99%)	555 (99%)	409 (98%)	
1 st degree	10 (1%)	1 (0.2%)	9 (2%)	
2nd degree	1 (0.1%)	1 (0.2%)	0	
3rd degree	0	0	0	
Conduction delay				
None	939 (96%)	542 (97%)	397 (95%)	
Intraventricular conduction delay	18 (2%)	6 (1%)	12 (3%)	
Right bundle branch block	18 (2%)	9 (2%)	9 (2%)	
Left bundle branch block	1 (0.1%)	0	1 (0.2%)	
Intervals				
PR	137 ±21	134± 20	141 ± 22	<0.0001
QRS	84 ± 13	84 ± 11	86 ± 14	0.01
QTc	449 ± 48	445 ± 47	454 ± 48	0.01
Ectopy				
None	953 (98%)	548 (98%)	405 (97%)	
Premature atrial contractions	9 (1%)	5 (1%)	4 (1%)	
Premature ventricular contractions	13 (1%)	5 (1%)	8 (2%)	
Ischemia/Injury	214 (22%)	113 (20%)	101 (24%)	
ST elevation	15 (2%)	8 (1%)	7 (2%)	0.8
ST depression	101 (10%)	51 (9%)	50 (12%)	0.2
T wave inversion	115 (12%)	59 (11%)	56 (13%)	0.2
Non-specific ST-T wave abnormalities	175 (18%)	103 (18%)	72 (17%)	0.6
Prior myocardial infarction				<0.001
None	905 (93%)	533 (96%)	372 (89%)	

	All donors	Transplanted hearts	Non-Transplanted hearts	p-value*
	n=980	n=560	n=420	
Inferior	21 (2%)	7 (1%)	14 (3%)	
Anterior	44 (5%)	14 (3%)	30 (7%)	
ST segment deviation†				
I	0.05 ± 0.3	0.02 ± 0.2	0.08 ± 0.4	0.001
II	0.2 ± 0.6	0.2 ± 0.6	0.2 ± 0.6	0.8
III	0.1 ± 0.4	0.1 ± 0.4	0.1 ± 0.5	0.5
aVF	0.1 ± 0.4	0.2 ± 0.4	0.1 ± 0.5	0.6
aVL	0.03 ± 0.3	0.02 ± 0.1	0.05 ± 0.4	0.06
aVR	0.09 ± 0.3	0.08 ± 0.3	0.09 ± 0.4	0.5
V1	0.09 ± 0.3	0.07 ± 0.3	0.1 ± 0.4	0.03
V2	0.2 ± 0.5	0.2 ± 0.5	0.2 ± 0.6	0.1
V3	0.3 ± 0.6	0.2 ± 0.5	0.3 ± 0.7	0.01
V4	0.3 ± 0.8	0.3 ± 0.6	0.3 ± 1.0	0.2
V5	0.2 ± 0.7	0.2 ± 0.5	0.3 ± 1.0	0.02
V6	0.2 ± 0.5	0.1 ± 0.4	0.2 ± 0.6	0.1
Abnormal T wave				
I	79 (8.1%)	26 (4.6%)	53 (12.6%)	<0.001
II	91 (9.3%)	40 (7.1%)	51 (12.1%)	0.01
III	77 (7.9%)	45 (8.0%)	32 (7.6%)	0.8
aVF	86 (8.8%)	44 (7.9%)	42 (10%)	0.2
aVL	54 (5.5%)	17 (3%)	37 (8.8%)	<0.001
aVR	45 (4.6%)	14 (2.5%)	31 (7.4%)	<0.001
V1	36 (3.7%)	15 (2.7%)	21 (5%)	0.06
V2	95 (9.7%) 105	44 (7.9%)	51 (12.1%)	0.03
V3	(10.7%) 135	48 (8.6%)	57 (13.6%)	0.01
V4	(13.8%) 133	61 (10.9%)	74 (17.6%)	0.002
V5	(13.6%)	58 (10.4%)	75 (17.9%)	0.001
V6	118 (12%)	47 (8.4%)	71 (16.9%)	<0.001
Q wave				
I	2 (0.2%)	1 (0.2%)	1 (0.2%)	0.8
II	5 (0.5%)	3 (0.5%)	2 (0.5%)	0.9
III	11 (1.1%)	5 (0.9%)	6 (1.4%)	0.4
aVF	11 (1.1%)	5 (0.9%)	6 (1.4%)	0.4
aVL	8 (0.8%)	4 (0.7%)	4 (1.0%)	0.7
aVR	1 (0.1%)	0	1 (0.2%)	0.3
V1	27 (2.8%)	3 (0.5%)	24 (5.7%)	<0.001
V2	25 (2.6%)	2 (0.4%)	23 (5.5%)	<0.001
V3	16 (1.6%)	3 (0.5%)	13 (3.1%)	0.002
V4	5 (0.5%)	2 (0.4%)	3 (0.7%)	0.4
V5	4 (0.4%)	2 (0.4%)	2 (0.5%)	0.8
V6	4 (0.4%)	2 (0.4%)	2 (0.5%)	0.8

	All donors	Transplanted hearts	Non-Transplanted hearts	p-value*
	n=980	n=560	n=420	
U waves				
I	0	0	0	
II	5 (0.5%)	5 (0.9%)	0	0.05
III	6 (0.6%)	5 (0.9%)	1 (0.2%)	0.2
aVF	6 (0.6%)	4 (0.7%)	2 (0.5%)	0.6
aVL	1 (0.1%)	0	1 (0.2%)	0.3
aVR	2 (0.2%)	2 (0.4%)	0 (0%)	0.2
V1	2 (0.2%)	2 (0.4%)	0	0.2
V2	18 (1.8%)	15 (2.7%)	3 (0.7%)	0.02
V3	22 (2.2%)	17 (3.0%)	5 (1.2%)	0.05
V4	19 (1.9%)	13 (2.3%)	6 (1.4%)	0.3
V5	13 (1.3%)	8 (1.4%)	5 (1.2%)	0.8
V6	7 (0.7%)	4 (0.7%)	3 (0.7%)	1.0

*transplanted vs non-transplanted hearts

†absolute value of ST elevation or depression

Table 3

Electrocardiographic Predictors of Cardiac Allograft Utilization

	Odds Ratio	95% Confidence Interval	p-value*
Rate (per 10 bpm increase)	1.00	0.9-1.1	0.9
Chamber enlargement			
Atrial enlargement			
Left	1.1	0.6-1.9	0.8
Right	0.8	0.3-1.9	0.6
Left ventricular hypertrophy	0.3	0.2-0.5	<0.001
Intervals			
PR (per 10 msec increase)	0.9	0.8-0.98	0.01
QRS (per 10 msec increase)	0.8	0.7-0.9	0.002
QTc (per 10 msec increase)	1.0	0.9-1.0	0.9
Myocardial ischemia			
Ischemia/Injury	1.3	0.9-1.9	0.2
Prior myocardial infarction	0.6	0.3-1.2	0.2
ST segment deviation†			
I	0.5	0.2-0.9	0.03
II	1.1	0.8-1.4	0.7
III	1.2	0.8-1.6	0.4
aVL	0.5	0.2-1.2	0.1
aVR	1.1	0.7-1.6	0.8
aVF	1.3	0.9-1.8	0.2
V1	0.6	0.4-0.9	0.04
V2	0.7	0.5-0.9	0.03
V3	0.7	0.5-0.9	0.01
V4	0.9	0.7-1.2	0.6
V5	0.9	0.7-1.2	0.5
V6	1.1	0.8-1.5	0.7
Abnormal T wave			
I	0.5	0.3-0.9	0.01
II	0.6	0.3-0.9	0.03
III	0.9	0.5-1.5	0.6
aVL	0.6	0.3-1.3	0.2
aVR	0.4	0.2-0.8	0.01
aVF	0.7	0.4-1.3	0.3
V1	0.6	0.3-1.4	0.3
V2	0.8	0.5-1.3	0.4
V3	0.8	0.5-1.3	0.4
V4	0.7	0.5-1.1	0.2
V5	0.7	0.5-1.2	0.2
V6	0.7	0.4-1.1	0.1

Q wave	Odds Ratio	95% Confidence Interval	p-value*
I	‡		
II	0.8	0.1-8.0	0.9
III	0.8	0.2-3.5	0.8
aVR	§		
aVL	0.3	0.1-1.5	0.1
aVF	0.8	0.2-3.5	0.8
V1	0.2	0.04-0.6	0.01
V2	0.1	0.03-0.6	0.01
V3	0.3	0.07-1.2	0.09
V4	1.7	0.2-15.3	0.6
V5	0.8	0.04-18	0.9
V6	0.8	0.03-18	0.9

*Adjusted for: donor age, sex, cause of death, race, height, hypertension, diabetes coronary artery disease, and blood type

†Absolute value of ST segment elevation or depression

‡Only 2 ECGs had Q waves in lead I

§Only 1 ECG had a Q wave in lead aVR

Table 4

Abnormal ST segments, T waves, and Q waves as Predictors of Cardiac Allograft Utilization: Unadjusted and Adjusted Models

Model	Any ST/T/Q Abnormality		ST segment abnormalities		Pathologic Q waves		Abnormal T wave inversions	
	OR (95% CI)	p-value	OR (95% CI)	p-value	OR (95%CI)	p-value	OR (95% CI)	p-value
Unadjusted	0.76 (0.59, 0.98)	0.037	0.89 (0.68, 1.16)	0.38	0.35 (0.18, 0.65)	9.42E-004	0.71 (0.53, 0.96)	0.026
Adjusted for donor demographics *	0.91 (0.68, 1.23)	0.55	0.95 (0.69,1.30)	0.74	0.43 (0.21, 0.90)	0.024	0.89 (0.63, 1.26)	0.52
Adjusted for donor demographics and echocardiographic abnormalities †	1.10 (0.78, 1.55)	0.59	1.05 (0.73, 1.51)	0.78	1.00 (0.42, 2.38)	1.00	0.95 (0.64, 1.40)	0.80

*Age, sex, race, cause of death

†Left ventricular ejection fraction < 50%, left ventricular regional wall motion abnormalities, left ventricular hypertrophy