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Authors
Olmos, Andrea
Feiner, John
Hirose, Ryutaro
et al.

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Impact of a quality improvement project on deceased organ donor management

Andrea Olmos, MD, **John Feiner, MD**, **Ryutaro Hirose, MD**, **Sharon Swain, RN, MSN**, **Annabel Blasi, MD**, **John P. Roberts, MD**, and **Claus U. Niemann, MD**

University of California, San Francisco (AO, JF, RH, JPR, CUN), California Transplant Donor Network, Oakland, California (SS), Hospital Clinic, Barcelona, Spain (AB)

Abstract

Context—Donors showed poor glucose control in the period between declaration of brain death and organ recovery. The level of hyperglycemia in the donors was associated with a decline in terminal renal function.

Objective—To determine whether implementation of a quality improvement project improved glucose control and preserved renal function in deceased organ donors.

Methods—Data collected retrospectively included demographics, medical history, mechanism of death, laboratory values, and data from the United Network for Organ Sharing.

Results—After implementation of the quality improvement project, deceased donors had significantly lower mean glucose concentrations (mean [SD], 162 [44] vs 212 [42] mg/dL; $P < .001$) and prerecovery glucose concentration (143 [66] vs 241 [69] mg/dL; $P < .001$). When the donor cohorts from before and after the quality improvement project were analyzed together, mean glucose concentration remained a significant predictor of terminal creatinine level ($P < .001$). Multivariate analysis of delayed graft function in kidney recipients matched to donors indicated that higher terminal creatinine level was associated with delayed graft function in recipients ($P < .001$).

Conclusion—The quality improvement project improved donor glucose homeostasis, and the data confirm that poor glucose homeostasis is associated with worsening terminal renal function.

Nationwide there is an acute shortage of organs available for transplant, especially for patients requiring kidney transplants.[1,2] In an effort to bridge the gap between the high demand for kidney transplants and the low supply of donors, the US Department of Health and Human Services has tried to expand the deceased donor pool by using more suboptimal donors, including extended-criteria donors and donation after cardiac death (DCD) donors.[3,4] However, the initial function of organs transplanted from these donors is often compromised.[5,6] Furthermore, kidneys transplanted from suboptimal donors have been associated with higher rates of delayed graft function (DGF), defined as the need for dialysis

Corresponding author: Claus U. Niemann, MD, University of California at San Francisco, Department of Anesthesia and Perioperative Care & Department of Surgery, Division of Transplantation, 521 Parnassus Avenue, C 450, San Francisco, CA 94143-0648 (claus.niemann@ucsf.edu).

within 7 days following transplant. As a result, the concept of aggressive donor management has gained favor.[5,7–15]

Certain donor characteristics that may affect donor organ quality are not modifiable, but others such as hemodynamic stability and metabolic homeostasis can be optimized before organ recovery. Terminal renal function in deceased donors (defined as renal function, reflected in either creatinine level or glomerular filtration rate, just before organ recovery) is one of the most important predictors of renal graft function in recipients following kidney transplant. Aggressive medical management during the period between declaration of brain death and organ recovery may preserve terminal renal function.[16–18]

Evidence from both animal and clinical studies suggests that hyperglycemia may be a source of additional injury to renal grafts, possibly through its effects on several molecular pathways.[19] Although the mortality benefit of tight glucose control in the intensive care unit is debated, studies in the critical care setting have demonstrated that tight glucose control is renoprotective in critically ill patients.[20–32] Despite this evidence, hyperglycemia in deceased organ donors has been largely ignored until recently. Blasi-Ibanez et al[33] demonstrated that poor glucose control in donors between declaration of brain death and organ recovery was not in line with accepted critical care standards. Furthermore, higher mean glucose concentration and greater variability in glucose concentrations were associated with worse terminal renal function in the donors.[33] In response to these results, the local organ procurement organization (OPO), California Transplant Donor Network (CTDN), implemented a quality improvement project (QIP) and modified its glycemic protocol for deceased donors to reflect standard critical care practices as detailed in the supplemental material.

The primary goal of the current study was to determine whether the QIP implementation across 80 hospitals in region 5 by CTDN resulted in improved glucose homeostasis in deceased organ donors. Given that the study by Blasi-Ibanez et al showed donor glucose control to be highly predictive of terminal renal function, one of the most important predictors of delayed graft function in kidney recipients, the secondary goal of the study is to increase the power of the original study by Blasi-Ibanez et al and assess the relationship between donor glucose control and early renal graft function in matched kidney recipients as determined by rates of delayed graft function.

Methods

Approval for this study from the Committee on Human Research at the University of California San Francisco was not required because deceased donors are not considered human subjects under federal law. CTDN developed a new insulin protocol, which was transitioned into the donor management protocols for its donation service area during a 2-month period and fully implemented by January 2009. Deceased organ donors were identified through the “CTDN” database during the period of January 2009 through August 2010. Pediatric donors (age < 18 years), DCD donors, and donors who had been enrolled in an ongoing prospective randomized study were excluded. Figure 1 summarizes the numbers in the cohorts; 241 deceased organ donors were identified after full QIP implementation and

enrolled in the current study ("post-QIP donor cohort"). The 2005–2006 cohort of 458 donors, reported originally in the study by Blasi-Ibanez et al before implementation of the QIP, was used as a historic control and was designated the "pre-QIP donor cohort." No other changes in deceased donor management occurred during the time period spanned by these 2 donor cohorts. All clinical data were prospectively collected as part of the clinical care of deceased organ donor and electronically stored (iTransplant, Transplant Connect). Pertinent data elements for this study were collected as previously described.[33]

A total of 543 deceased donors from both cohorts were matched with their respective kidney recipients for a total of 1036 recipients (kidneys from the remaining donors were discarded for a variety of reasons, including technical reasons and poor function). Matched kidney recipients were followed through the United Network for Organ Sharing (UNOS) database to assess for immediate graft function as defined by the presence or absence of DGF. Pediatric and combined kidney-pancreas transplant recipients were excluded from the analysis. Pertinent recipient data, including age, race, sex, and body mass index (BMI, calculated as weight in kilograms divided by height in meters squared) were recorded. When available, operative variables including cold and warm ischemia times also were recorded. Donor blood glucose and donor terminal creatinine levels and calculated glomerular filtration rate (GFR, calculated using the Modification of Diet in Renal Disease Study equation) were used as continuous variables and were the primary donor end points. DGF was the primary recipient end point.

Statistical Analysis

To examine the effect of the glucose management protocol modifications, we compared the pre-QIP donor cohort and the post-QIP donor cohort by using the Mann-Whitney U test for continuous variables, and a χ^2 or Fisher exact test for categorical variables. For univariate analyses, the relationship of continuous variables to terminal creatinine level or terminal GFR was analyzed by means of the Spearman rank correlation. For categorical variables, the Mann-Whitney U test or Kruskal-Wallis test was used. The relationship of continuous variables to DGF was analyzed by logistic regression. Categorical variables were analyzed by χ^2 test or Fisher exact test for 2-by-2 contingency tables.

For multivariate analyses of donor renal function, all predictors were considered for entry into the models. Stepwise regression with backward elimination was used to identify the most significant predictors. Once these variables had been entered into the model, all variables that could be reasonably considered in the model were entered and tested for statistical significance regardless of whether they were significant in the univariate analysis. The distributions of both terminal creatinine level and terminal GFR were skewed, making a simple linear model ineffective. Logarithmic transformation of both creatinine level and GFR resulted in a substantially improved distribution, which led to a sufficiently robust multivariate model. Analyses were run with and without "cohort" forced into the model. Results were not appreciably different without "cohort" in the model; however, because of diffrences between groups, results are shown with "cohort" in the models. Multivariate models for DGF were developed similarly and analyzed by multiple logistic regression.

Results of logistic regression for DGF were also confirmed by using a mixed-effects model with the kidney donor as a random effect.

Data are displayed as mean (SD) or as number (percentage) unless otherwise specified. Data were analyzed by using JMP 10.0 (SAS Institute) and Stata 12.1 (StataCorp). *P* less than .05 was considered statistically significant.

Results

Deceased Organ Donor Characteristics

Pre-QIP and post-QIP kidney donor and recipient cohorts are summarized in Figure 1. Donor characteristics are summarized in Table 1. The 2 donor cohorts differed significantly in ethnicity, with 10% of the pre-QIP donor cohort versus 5% of the post-QIP donor cohort being African American ($P = .02$). More donors in the pre-QIP cohort had experienced a cerebral vascular accident (52% vs 42%, $P = .01$), whereas more donors in the post-QIP cohort experienced anoxia (24% vs 13%, $P < .001$). The time (hours) between declaration of brain death and cross-clamping of the aorta was significantly longer in the post-QIP cohort than in the pre-QIP cohort (mean [SD], 45.2 [17.8] vs 49.2 [18.8], $P = .006$); however, this difference most likely does not reflect an adverse effect of the QIP and may be related to organ allocation issues.

Comparison of Glucose Homeostasis Before and After Implementation of the QIP

During the period between declaration of brain death and organ recovery, the post-QIP donor cohort had better glucose control than the pre-QIP donor cohort did (Figure 2). Compared with the pre-QIP donor cohort, the post-QIP donor cohort had significantly lower mean blood glucose concentration, glucose concentration variability as determined by standard deviation, maximum glucose concentrations, and prerecovery glucose concentrations (Table 2).

Association of Donor Characteristics With Terminal Renal Function

Terminal renal function is summarized in Table 2. In the univariate analysis of the post-QIP cohort (Table 3), older donor age was significantly related to both higher terminal creatinine level and lower terminal GFR. Higher BMI was correlated with higher terminal creatinine level ($P = .005$) and lower terminal GFR ($P = .002$). Terminal creatinine level did not differ between donors according to cause of brain death. However, GFR was higher for head trauma donors and lower for cerebral vascular accident donors ($P = .002$). The time between declaration of brain death and cross-clamping of the aorta was not related to terminal renal function. Donors with a history of hypertension had significantly higher terminal creatinine levels (mean [SD], 1.84 [1.35] vs 1.30 [0.94] mg/dL; $P < .001$; to convert to millimoles per liter, multiply by 88.4) and lower terminal GFR (56.4 [33.9] vs 81.3 [40.4] mL/min per 1.73 m^2; $P < .001$). Diabetes was not significantly correlated with terminal creatinine level or GFR.

Combined Cohorts

In the univariate analysis of the pre-QIP donor cohort by Blasi-Ibanez et al,[33] higher mean glucose concentration between declaration of brain death and organ recovery, maximum glucose concentration, prerecovery glucose concentration, and glucose concentration variability were all significantly associated with worsening terminal renal function.

In the univariate analysis of the post-QIP donor cohort, higher mean glucose concentrations between declaration of brain death and organ recovery were significantly associated with higher terminal creatinine level (P= .03) but were not significantly associated with terminal GFR (P = .06). However, unlike in the pre-QIP donor cohort, maximum glucose concentration, prerecovery glucose concentration, and glucose concentration variability were not associated with either terminal creatinine level or GFR (Table 3).

In order to achieve greater power and increase the range of donor glucose concentrations, datasets were combined and the univariate analysis for donor glucose and terminal renal function was repeated. Higher mean glucose concentration between declaration of brain death and organ recovery, maximum glucose concentration, and glucose concentration variability were again all significant determinants of higher terminal creatinine level and lower terminal GFR (Table 4).

Results of multivariate analysis for renal function as measured by terminal creatinine level and terminal GFR are shown in Table 5. Male sex, nonwhite ethnicity, and higher BMI were all significant determinants of worse terminal renal function. Donor history of hypertension was associated with higher terminal creatinine level and lower terminal GFR and remained highly statistically significant in the multivariate model. Mean glucose concentration correlated strongly with both higher terminal creatinine level and lower terminal GFR in the multivariate analysis. Differences between the 2 cohorts, including race, cause of death, and time from declaration of brain death to organ donation, which could be potential confounders, were addressed by including these variables in the multivariable models. When included in the multivariate analysis, these factors were not predictive of terminal renal function.

Immediate Allograft Function in Matched Kidney Recipients

Kidney Recipient Characteristics—A total of 1036 recipients receiving a renal graft from both cohorts were followed up. The mean age was 51.4 (SD, 15.1) years; 62% of the recipients were male; 38% of recipients were white, 23% Hispanic, 20% Asian, and 15% African American; mean BMI was 26.5 (SD, 5.1).

Association of Donor and Recipient Variables With DGF—For analysis of outcomes, pediatric recipients and patients receiving a simultaneous pancreas transplant were excluded, leaving 896 recipients who contributed data for analysis. Univariate analysis demonstrated that donor age, sex, ethnicity, BMI, history of diabetes, and history of hypertension were not associated with recipient DGF (Table 6). Longer cold ischemia time was significantly associated with the occurrence of DGF in the recipients (P = .004). Donor terminal renal function was highly correlated with DGF in the matched recipients. Donor

glucose measurements were not significantly associated with recipient DGF. Of the recipient variables for which data were available, male sex and higher BMI were significantly associated with DGF.

Results of multivariate analysis for recipient DGF can be found in Table 7. Separate models were developed for terminal renal function using creatinine level or GFR. Older donor age was a significant predictor of recipient DGF in the model based on creatinine level. Longer cold ischemia time remained a significant predictor of recipient DGF in the multivariate analysis. Donor terminal creatinine and terminal GFR were highly associated with DGF. Recipient male sex and higher recipient BMI were significant predictors of recipient DGF.

Discussion

The QIP implemented by “CTDN” was successful in improving glucose homeostasis in deceased organ donors. The data from this study confirmed that poor glucose control in deceased donors is an independent predictor of worsening terminal renal function. Furthermore, like previous studies, the current study showed that poor terminal renal function in the same large donor cohort is associated with DGF in corresponding kidney recipients.

From the time that a patient is declared brain dead to the time that the organs are recovered, the OPO takes over care of the donor. Therefore, donor management is standardized across hospitals in a given donation service area managed by a single OPO. A growing concern for donor management has led to the development of donor management goals (DMGs, critical care end points aimed at restoring and stabilizing the physiological functions of deceased donors).[34] Like CTDN’s QIP to improve donor glucose control, these DMGs can be easily implemented given that all hospitals in a given donation service area are covered by one OPO. This arrangement makes it much easier and more feasible to educate OPO staff, implement a DMG and ensure adherence, given that it is a more centralized process. However, most evidence for DMGs is retrospective and results are conflicting regarding which individual DMGs significantly affect organ yield.[34–36] Furthermore, we lack data on how the individual DMGs affect the quality of specific donor organs and the function of grafts in corresponding recipients.

In a retrospective study of 458 deceased organ donors, Blasi-Ibanez et al[33] demonstrated poor glucose control in donors between declaration of brain death and organ recovery. Furthermore, the level of hyperglycemia in donors correlated with a decline in terminal renal function. In response to these data, CTDN implemented a QIP, modifying its glucose homeostasis protocol to reflect standard critical care practices. In this follow-up study, donors from the post-QIP cohort were managed according to the new glucose protocol.

Between declaration of brain death and organ recovery, donors from the post-QIP cohort had significantly lower glucose concentrations, showing that “CTDN’s” QIP improved glucose homeostasis in deceased donors. Data from the pre-QIP donor cohort showed that higher glucose concentrations (mean, maximum, prerecovery, variability) were highly associated with worse terminal renal function. In the post-QIP donor cohort, only mean glucose

concentration was significantly correlated with terminal renal function. There are several possible explanations for why we did not observe the same robust relationships in the data from the post-QIP donor cohort alone. This cohort of donors may have been underpowered to detect a correlation between donor glucose control and renal function (type 2 error). Also, glucose control in the post-QIP cohort may have been too tight, without enough spread to demonstrate a strong relationship between hyperglycemia and terminal renal function. Finally, there may be a threshold value for glucose below which glucose concentration does not have a strong impact on renal function. This threshold may be the plasma glucose concentration at which glucose appears in the urine (approximately 200 mg/dL; to convert to millimoles per liter, multiply by 0.0555).[37] When data from the 2 cohorts were combined, mean and maximum glucose concentrations as well as glucose concentration variability were again highly associated with terminal renal function, supporting our hypothesis that glucose control in the post-QIP cohort was too narrow to show the effect of hyperglycemia on terminal renal function.

We then assessed renal graft function in the corresponding recipients by examining rates of delayed graft function. An accumulating body of evidence indicates that DGF in kidney recipients increases rates of acute rejection, reduces graft and patient survival, and increases health care costs.[8–15,18,38,39] DGF is a universally captured end point and also consistently documented in the UNOS database.

We assessed the occurrence of DGF in kidney recipients matched to donors in this study. We did not demonstrate a highly significant direct correlation between donor glucose control and DGF in recipients. In the first portion of the study, we demonstrated a strong correlation between donor hyperglycemia and donor terminal renal function by using combined data from both cohorts. Given the existence of risk prediction models identifying terminal creatinine level as one of the most significant risk factors for DGF, we would expect to find associations between hyperglycemia in deceased organ donors and DGF in matched recipients.[16,17]

Several factors may contribute to the observed results. The study may be underpowered to detect an association between donor glucose control and recipient DGF. The threshold for initiating dialysis on postoperative kidney transplant recipients may have also changed with evolving clinical practice and most likely varies between providers and institutions. Another important factor is the presence of additional recipient variables that affect the occurrence of DGF.[14,16–18,38,39] These recipient variables may obscure correlations between donor variables, such as glucose homeostasis, and DGF.

Assessing the effects that a donor management intervention has on the recipients of the corresponding organs is difficult for multiple reasons. For example, managing data from multiple relational databases is challenging. Also, when the predictor variable is being measured in one population (the donors) and the outcome of interest is being measured in a separate heterogeneous population (the recipients) with its own set of interacting variables, it becomes very difficult to control for all interactions and to identify independent correlations.

Despite these barriers to donor-recipient research, we made an indirect correlation between glucose control in deceased donors and DGF in recipients. We first demonstrated that mean glucose concentration during the period between declaration of brain death and organ recovery is an independent predictor of terminal renal function. We then went on to show that terminal renal function in the same set of donors is an independent predictor of DGF in the matched kidney recipients. Evidence is accumulating that terminal creatinine level in deceased donors is a strong predictor of DGF in kidney recipients. Therefore, a donor management strategy that can improve terminal renal function (organ quality) may ultimately improve outcomes in the recipients of those kidneys.

The study has several strengths and limitations. One of the strengths is that by combining donors from both the pre-QIP cohort and the post-QIP cohort, we have a large donor cohort from a single OPO that uses a common protocol. Another strength is that the 2 donor cohorts were demographically fairly similar, which is important when assessing the effect of an intervention in a retrospective manner.

One significant limitation of the study is that the data were not collected in a randomized or prospective manner, leading to many confounding relationships. Confounding interactions are particularly problematic when assessing for a relationship between glucose control in donors and DGF in recipients. However, our multivariate models suggest that the effect of donor glucose concentration on terminal renal function and the effect of terminal renal function on recipient DGF are not related to other more significant variables.

These results suggest that aggressive management of hyperglycemia in deceased donors is a donor management strategy with the potential to improve the quality of donor kidneys and the outcomes of transplanted renal allografts. In order to address the limitations of a retrospective study design and more rigorously examine the effects of donor glucose control and recipient outcomes, the next step would be to conduct a prospective randomized study evaluating the effects of intensive insulin therapy on renal function in deceased donors. Such a study will hopefully validate the benefits of aggressive glucose management in deceased donors.

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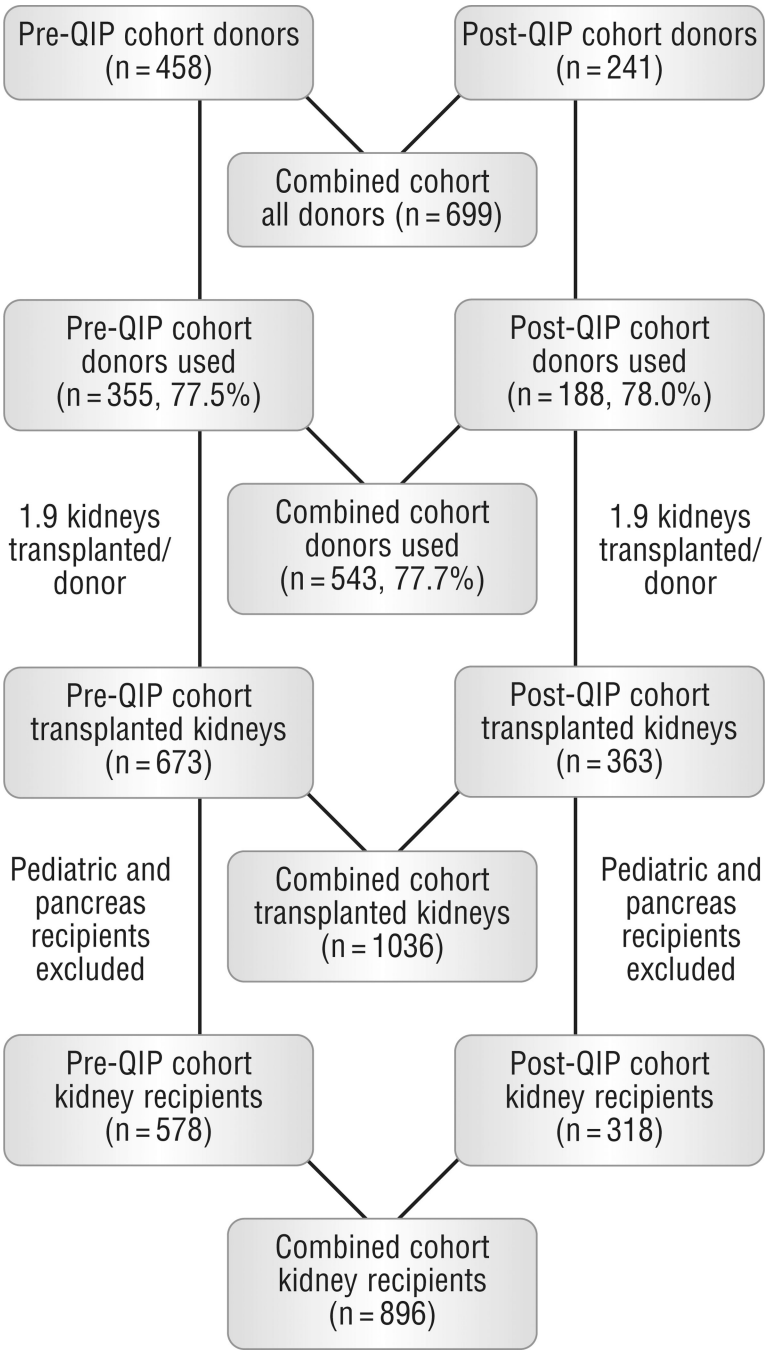

Figure 1.
Flow diagram shows the donor and recipient numbers before and after the quality improvement project (QIP).

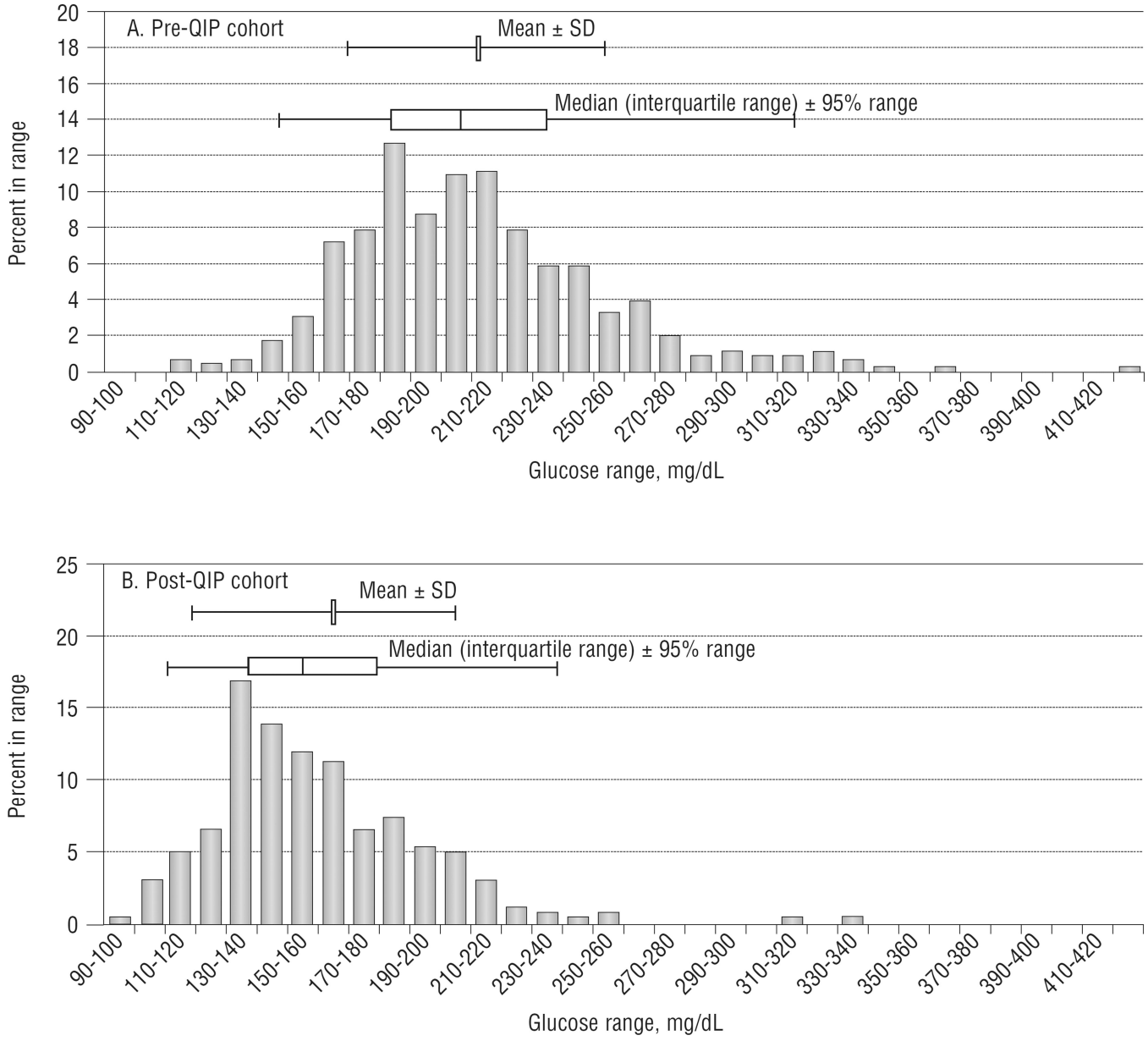

Figure 2.

A histogram of the distribution of glucose values for the pre–quality improvement project (QIP) cohort is shown in panel A and for the post-QIP cohort in panel B. The mean ± SD is indicated at the top of the figure by line and error bars. The box plot shows the median and interquartile range, and the error bars show the 95th percentile range. The groups differed significantly, $P < .001$.

Table 1

Organ donor data: all donors combined from before and after quality improvement project (QIP)[a]

Characteristic	Pre-QIP cohort (n = 458)	Post-QIP cohort (n = 241)	All (n = 699)	*P*
Age, mean (SD), years	43.2 (14.9)	44.3 (14.8)	43.6 (14.9)	.34
Sex				.63
Female	198 (43.2)	99 (41.1)	297 (42.5)	
Male	260 (56.8)	142 (58.9)	402 (57.5)	
Body mass index (BMI),[b] mean (SD)	26.9 (5.9)	27.5 (7.2)	27.1 (6.4)	.31
Obese (BMI ≥30)	129 (28.2)	66 (27.4)	195 (27.9)	.86
Ethnicity				.01
African American	48 (10.5)	13 (5.4)	61 (8.7)	.02
Asian	47 (10.3)	22 (9.1)	69 (9.9)	.69
White	260 (56.8)	133 (55.2)	393 (56.2)	.69
Hispanic	103 (22.5)	70 (29.0)	173 (24.7)	.06
Other	0 (0.0)	3 (1.2)	3 (0.4)	.04
Cause of brain death				<.001
Head trauma	162 (35.4)	79 (32.8)	241 (34.5)	.50
Cerebrovascular accident	238 (52.0)	101 (41.9)	339 (48.5)	.01
Anoxia	58 (12.7)	58 (24.1)	116 (16.6)	<.001
Comorbid conditions				
Hypertension	165 (36.0)	86 (35.7)	251 (35.9)	>.99
Diabetes	52 (11.4)	37 (15.4)	89 (12.7)	.15
Extended-criteria donor	117 (25.5)	62 (25.7)	179 (25.6)	>.99
Simplified Donor Risk Index, mean (SD)	1.08 (0.40)	1.10 (0.41)	1.09 (0.40)	.43
Hours from first declaration of brain death to cross-clamping,[c] mean (SD)	45.2 (17.8)	49.2 (18.8)	46.6 (18.2)	.006
% with at least 1 kidney transplanted	355 (77.5)	188 (78.0)	543 (77.7)	.92
Any liver transplanted	385 (84.1)	192 (79.7)	577 (82.5)	.17
Any pancreas transplanted	99 (21.6)	40 (16.6)	139 (19.9)	.13

[a] Data in second, third, and fourth columns are number (percentage) unless otherwise indicated in the first column.

[b] Calculated as weight in kilograms divided by height in meters squared.

[c] Data were available for only 239 of the 241 donors in the post-QIP cohort, thus data are for a total of 697 donors instead of 699.

Table 2

Donor laboratory values before and after the quality improvement project (QIP)

Characteristic	Pre-QIP cohort (n = 458)	Post-QIP cohort (n = 241)	*P*
Glucose level, mean (SD), mg/dL			.55
Admission	205 (81)	221 (139)	< .001
Prerecovery	241 (69)	143 (66)	< .001
Mean	212 (42)	162 (44)	< .001
Maximum	321 (92)	240 (79)	< .001
Standard deviation	65 (30)	53 (32) (n = 239)	< .001
Terminal glucose level, No. (%) of donors			< .001
≥ 200 mg/dL	332 (72.5)	27 (11.2)	
≥ 250 mg/dL	179 (39.1)	7 (2.9)	
Creatinine level, mean (SD), mg/dL			
Admission	1.08 (0.62) (n = 456)	1.29 (1.29)	.20
Terminal	1.27 (0.96)	1.49 (1.13)	.04
Change	0.19 (0.66) (n = 456)	0.21 (1.29)	.41
% Change	21.4 (58.8) (n = 456)	32.5 (76.1)	.40
Glomerular filtration rate, mL/min per 1.73 m^2			
Admission	86.7 (44.7) (n = 456)	82.4 (38.7)	.15
Terminal	79.8 (56.4)	72.4 (40.0)	.02
Change	−7.1 (53.2) (n = 456)	−10.0 (39.7)	.51
% Change	−2.0 (57.8) (n = 456)	3.5 (113.6)	.40

Table 3

Univariate analysis of donor terminal renal function (creatinine level and calculated glomerular filtration rate [GFR]) in the post–quality improvement project cohort (n = 241)

	Outcomes, *P*	
Predictor	**Terminal[a] creatinine level**	**Terminal[a] GFR**
Age	.04	<.001
Sex	<.001	.80
Body mass index[b] (BMI)	.005	002
Obese (BMI ≥ 30)	.013	.002
Ethnicity	.42	.30
African American	.16	.64
Asian	.47	.12
White	.24	.78
Hispanic	.73	.59
Cause of brain death	.43	.002
Head trauma	.14	<.001
Cerebrovascular accident	.43	.005
Anoxia	.38	.38
Comorbid conditions		
Hypertension	<.001	<.001
Diabetes	.23	.07
Hours from brain death to cross-clamp	.96	.18
Admission creatinine level	<.001	<.001
Admission GFR	<.001	<.001
Glucose		
Admission	.47	.51
Prerecovery	.28	.63
Mean	.03	.063
Standard deviation	>.99	.81
Maximum	.66	.75

[a] "Terminal" refers to last value before aortic cross-clamp.

[b] Calculated as weight in kilograms divided by height in meters squared.

Table 4

Univariate analysis of donor terminal renal function (creatinine level and calculated glomerular filtration rate [GFR]) in the combined cohorts from before and after the quality improvement project (n = 699)

		Outcomes, *P*	
Predictor	**Mean (SD)**	**Terminal creatinine level**	**Terminal GFR**
Glucose level, mg/dL			
Admission	210 (105)	.10	.26
Prerecovery	207 (82)	.38	.53
Mean	194 (49)	.007	<.001
Standard deviation	61 (31) (n = 697)	.001	<.001
Maximum	293 (96)	.03	.001

Table 5

Multivariate analysis of donor terminal renal function (creatinine level and calculated glomerular filtration rate [GFR]) in the combined cohorts from before and after the quality improvement project (n = 699)

	Outcomes, *P*	
Predictors	**Terminal[a] creatinine level**	**Terminal[a] GFR**
Age		.002
Sex	<.001	.01
Body mass index[b]	.002	.002
Ethnicity	.01	.03
Hypertension	<.001	.002
Admission creatinine level or GFR	<.001	<.001
Mean glucose level	<.001	<.001

[a]"Terminal", last value before cross-clamping. Admission creatinine level was used for analysis of terminal creatinine level, and admission GFR was used for analysis of terminal GFR.

[b]Calculated as weight in kilograms divided by height in meters squared.

Table 6

Univariate predictors of delayed graft function (DGF) in the combined cohorts[a]

Variable	DGF (n = 213)	No DGF (n = 683)	Odds ratio[b] (95% CI)	*P*
Donors				
Age, mean (SD), years	44.5 (13.8)	42.8 (13.5)	1.01 (1.00–1.02)	.11
Sex, No. (%) of donors				.47
Female	82 (38.5)	283 (41.4)		
Male	131 (61.5)	400 (58.6)	1.13 (0.82–1.55)	
BMI,[c] mean (SD)	27.9 (6.9)	27.2 (6.2)	1.02 (0.99–1.04)	.15
Obese (BMI ≥ 30), No. (%) of donors	69 (32.4)	196 (28.7)	1.19 (0.85–1.66)	.30
Ethnicity, No. (%) of donors				.18
African American	8 (3.8)	54 (7.9)	0.49 (0.23–1.07)	.04
Asian	22 (10.3)	60 (8.8)	1.22 (0.72–2.08)	.50
White	121 (56.8)	404 (59.2)	Reference, 1.00	.58
Hispanic	61 (28.6)	161 (23.6)	1.27 (0.88–1.81)	.15
Other	1 (0.5)	4 (0.6)	0.83 (0.09–7.55)	>.99
Cause of brain death, No. (%) of donors				.76
Head trauma	69 (32.4)	239 (35.0)		.51
Cerebrovascular accident	104 (48.8)	317 (46.4)	1.11 (0.71–1.73)	.58
Anoxia	40 (18.8)	125 (18.3)	1.14 (0.80–1.61)	.92
Comorbid conditions, No. (%) of donors				
Hypertension	70 (32.9)	209 (30.6)	1.11 (0.80–1.54)	.55
Diabetes mellitus	24 (11.3)	66 (9.7)	1.19 (0.72–1.94)	.51
Hours from brain death to cross-clamping, mean (SD)	51.0 (20.3)	48.9 ± 19.1	1.01 (1.00–1.01)	.18
Hours of cold ischemia, mean (SD)	16.1 (9.3) (n = 207)	14.1 (8.0) (n = 659)	1.03 (1.01–1.04)	.004
Minutes of warm ischemia, mean (SD)	34.3 (14.6) (n = 149)	33.5 (12.5) (n = 488)	1.00 (0.99–1.02)	.52
Extended criteria, No. (%) of donors	49 (23.0)	127 (18.6)	0.00 (0.00–0.00)	.17
Creatinine level, mean (SD), mg/dL				
Baseline	1.1 (0.5)	1.0 (0.4) (n = 682)	1.49 (1.04–2.14)	.03
Terminal[d]	1.3 (0.6)	1.1 (0.5)	1.84 (1.39–2.44)	<.001
GFR terminal,[d] mean (SD), mL/min per 1.73 m^2	71.7 (28.7)	85.5 (59.6)	0.99 (0.98–0.99)	<.001
Glucose level, mean(SD), mg/dL				
Terminal[d]	202 (86)	206 (80)	1.00 (1.00–1.00)	.52
Mean	195 (48)	196 (45)	1.00 (1.00–1.00)	.73

Variable	DGF (n = 213)	No DGF (n = 683)	Odds ratio[b] (95% CI)	*P*
Standard deviation	68 (41)	64 (32)	1.00 (1.00–1.01)	.14
Maximum	323 (118)	314 (117)	1.00 (1.00–1.00)	.34
Terminal[d] glucose level, No. (%) of donors				
≥200 mg/dL	101 (47.4)	347 (50.8)	0.87 (0.64–1.19)	.43
≥250 mg/dL	49 (23.0)	173 (25.3)	0.88 (0.61–1.27)	.53
Recipients				
Age, mean (SD), years	55.8 (11.8)	53.9 (13.0)	1.01 (1.00–1.02)	.06
Sex, No. (%) of recipients				.001
Female	60 (28.2)	276 (40.4)	Reference, 1.00	
Male	153 (71.8)	407 (59.6)	1.73 (1.23–2.42)	
BMI,[c] mean (SD)	27.7 (5.3) (n = 196)	26.7 (5.0) (n = 561)	1.04 (1.01–1.07)	.02
Ethnicity, No. (%) of recipients				.08
African American	47 (22.1)	97 (14.2)	1.70 (1.09–2.63)	
Asian	44 (20.7)	161 (23.6)	0.96 (0.63–1.46)	
White	72 (33.8)	252 (36.9)	Reference, 1.00	
Hispanic	42 (19.7)	154 (22.5)	0.95 (0.62–1.47)	
Other	8 (3.8)	19 (2.8)	1.47 (0.62–3.51)	
Days on waiting list, mean (SD)	1118 (879)	1058 (895)	1.00 (1.00–1.00)	.39
Allocation PRA 80%, No. (%) of recipients	7 (3.3)	19 (2.8)	1.19 (0.49–2.87)	.65

Abbreviations: BMI, body mass index; GFR, calculated glomerular filtration rate; PRA, plasma reactive antibody.

[a] Pediatric recipients and pancreas transplants were excluded.

[b] Odds ratios for continuous variable are for each unit increase.

[c] Calculated as weight in kilograms divided by height in meters squared.

[d] The word “terminal” indicates the last value before cross-clamping of aorta.

Table 7

Multivariate analysis of predictors of delayed graft function[a]

	P	
Predictor	**Creatinine model**	**GFR model**
Donor's age	.03	.41
Donor's terminal renal function		
Creatinine level	<.001	NA
GFR	NA	<.001
Cold ischemia time	.004	.006
Recipient's sex	.005	.003
Recipient's body mass index[b]	.01	.009

Abbreviations: GFR, glomerular filtration rate; NA, not applicable.

[a] Pediatric recipients and pancreas transplants were excluded. Models were done separately with creatinine or GFR as predictors. The word "terminal" indicates last laboratory value before donor cross-clamp.

[b] Calculated as weight in kilograms divided by height in meters squared.