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Continuous Renal Replacement Therapy for Children ≤10 kg: A Report from the Prospective Pediatric Continuous Renal Replacement Therapy Registry

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Abstract

OBJECTIVE—To report circuit characteristics and survival analysis in children weighing ≤10 kg enrolled in the Prospective Pediatric Continuous Renal Replacement Therapy (ppCRRT) Registry.

STUDY DESIGN—We conducted prospective cohort analysis of the ppCRRT Registry to: (1) evaluate survival differences in children ≤10 kg compared with other children; (2) determine demographic and clinical differences between surviving and non-surviving children #10 kg; and

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(3) describe continuous renal replacement therapy (CRRT) circuit characteristics differences in children #5 kg versus 5–10 kg.

RESULTS—The ppCRRT enrolled 84 children ≤10 kg between January 2001 and August 2005 from 13 US tertiary centers. Children ≤10 kg had lower survival rates than children >10 kg (36/84 [43%] versus 166/260 [64%]; $P < .001$). In children >10 kg, survivors were more likely to have fewer days in intensive care unit prior to CRRT, lower Pediatric Risk of Mortality 2 scores at intensive care unit admission and lower mean airway pressure (Paw), higher urine output, and lower percent fluid overload (FO) at CRRT initiation. Adjusted regression analysis revealed that Pediatric Risk of Mortality 2 scores, FO, and decreased urine output were associated with mortality. Compared with circuits from children 5–10 kg at CRRT initiation, circuits from children #5 kg more commonly used blood priming for initiation, heparin anticoagulation, and higher blood flows/effluent flows for body weight.

CONCLUSION—Mortality is more common in children who are ≤10 kg at the time of CRRT initiation. Like other CRRT populations, urine output and FO at CRRT initiation are independently associated with mortality. CRRT prescription differs in small children.

INTRODUCTION

Continuous renal replacement therapy (CRRT) has become the preferred dialysis option to support children with acute kidney injury (AKI) admitted to an intensive care unit (ICU).1 There continues to be limited pediatric data on the patterns of CRRT use, its safety, and its effect on ultimate patient survival in the ICU.[1–5] To study these factors in a systematic fashion, the Prospective Pediatric Continuous Renal Replacement Therapy (ppCRRT) Registry was established in 2001. The ppCRRT has reported data on hundreds of children who received CRRT in pediatric ICUs throughout the US, describing survival rates superior to those reported from single center experience or smaller studies.[6]

The provision of CRRT to younger and smaller children brings added risks because of higher circuit extracorporeal volumes relative to patient blood volume, higher relative blood flows, and alternative indications/diseases. No CRRT device has been specifically designed and tested for use in children, and devices designed for adult care are adapted for pediatric use. Few reports detail the technical aspects, outcomes, and potential variables associated with mortality in small children. In 2003, Symons et al performed a retrospective review of children <10 kg who received CRRT between 1993 and 2001 at 5 tertiary pediatric centers.[2] Their report suggested that children <10 kg had lower overall survival, especially those weighing less than 3 kg, and that the prescription, indications, and variables associated with survival were different than for larger children. However, because of the Retrospective data collection design, this study was limited in its ability to describe specific CRRT characteristics and ascertain variables associated with mortality. The ppCRRT prospectively collected data on 344 children who received CRRT from 13 centers between January 2001 and August 2005. We analyzed data from children ≤10 kg (84/344 [24.4%] of the entire ppCRRT) to improve our understanding of CRRT in small children who weigh ≤10 kg at CRRT initiation, and to test the following hypotheses: (1) demographic and clinical characteristics of children who weigh #10 kg differ between survivors and non-survivors, even after controlling for potential confounders; (2) the circuit characteristics of those who

weigh#5 kg at CRRT initiation differ from children whose weight was >5 kg; and (3) mortality is higher in children ≤10 kg compared with other children in the ppCRRT Registry.

METHODS

Details of the ppCRRT Registry design have been reported previously.[3–9] In brief, the ppCRRT Registry uses a prospective observational format; all centers practice according to local standards of care and agree to collect the same data. Decisions to initiate, alter, or terminate CRRT are made by the clinicians based on their local standards of care and local clinical practice. The ppCRRT Registry did not direct any specific aspect of patient care or any specifics of the CRRT circuit. This analysis for infants ≤10 kg comprises data collected from 11 US pediatric centers who enrolled at least 1 patient ≤10 kg: Baylor College of Medicine/Texas Children's Hospital, Houston, Texas; Children's Hospital, Boston, Massachusetts; Children's Hospital & Regional Medical Center, Seattle, Washington; CS Mott Children's Hospital, Ann Arbor, Michigan; University of Alabama at Birmingham/ Children's of Alabama, Birmingham, Alabama; Children's Mercy Hospitals and Clinics, Kansas City, Missouri; Emory University/Children's Healthcare of Atlanta at Egleston, Atlanta, Georgia; All Children's Hospital, St. Petersburg, Florida; Helen DeVos Children's Hospital, Grand Rapids; Lucille Packard Children's Hospital, Stanford, California; and The Cleveland Clinic, Cleveland, Ohio. Data from 2 other centers (Columbus Children's Hospital, Columbus, Ohio; and Children's National Medical Center, Washington DC) are included in the entire ppCRRT Registry, but these centers did not enroll children ≤10 kg into the registry.

The ppCRRT Registry collected clinical data from the time of admission into the ICU until CRRT initiation (ICU data), clinical data at the time of CRRT initiation (CRRT data), and data from each CRRT circuit (CRRT circuit data). Data were collected between January 1, 2001 and August 31, 2005. Each participating center's Institutional Review Board approved the study prior to subject enrollment and data collection. Data were pooled to a central registry database. Specific ICU data included: interval from ICU admission to CRRT initiation, urine output in the 24 hours prior to CRRT initiation, fluid input from ICU admission to CRRT initiation, fluid output from ICU admission to CRRT initiation, pressor dependency prior to CRRT, serum creatinine at ICU admission, central venous pressure at CRRT initiation, total number of inotropic agents infusing at CRRT initiation, ability to wean inotropic medications during CRRT, weight at the time of ICU admission, mean airway pressure (Paw) at both CRRT initiation and termination, and duration of ICU stay. Data obtained at the time of CRRT initiation include patient age, sex, height, weight, primary disease leading to CRRT initiation, relevant comorbid illnesses, reason for CRRT initiation (development or prevention of fluid overload [FO], electrolyte imbalance, or both), Paw, and serum creatinine. Using the method described by Goldstein et al, percent FO (%FO) for each child was determined at the time of CRRT initiation, using the ICU admission weight in kg as the baseline weight of comparison.[10]

$$\%\mathrm{FO} - [\text{“Fluid in” (liters)} - \text{“fluid out” (liters)}] / [\text{ICU admission weight (kg)} \times 100\%]$$

To assess illness severity, Pediatric Risk of Mortality (PRISM) 2 scores were calculated at ICU admission and CRRT initiation. PRISM 2 scores are derived from 14 clinical variables from 5 different organ system domains and have been validated as an objective measure for stratifying critical pediatric illness.[11] CRRT circuit and filter data collected included CRRT machine brand, anticoagulation method, priming solution, blood pump flow rate, and circuit life. CRRT modality was categorized based on the use of purely convective therapy with replacement fluids (continuous hemofiltration), use of dialysate only (continuous hemodialysis), or circuits using both dialysate and replacement fluids (continuous hemodiafiltration). Each center's investigator initially characterized a patient's primary illness and comorbid conditions at the time of CRRT. These were then centrally grouped into distinct categories. The primary outcome measure was patient survival to ICU discharge.

Statistical Analyses

Descriptive statistics were performed to determine differences between survivor and non-survivors and differences in circuit characteristics between those with weight #5 kg compared with those with weight >5 kg. Shapiro–Wilk test and normal probability plot were used to test for normality of data. Normally distributed continuous variables were compared using Student t-test and reported as mean (±SD), and non-normally distributed variables were analyzed using Mann–Whitney test and reported as median (25% IQR, 75% IQR). Categorical variables were analyzed using chi-square analysis if 2 variables were present and Mantel–Haenszel c2 if 3 or more categorical variables were present. For all descriptive statistics, a P value of <.05 was considered statistically significant. The association between demographics and survival was initially analyzed using crude univariate analysis. Then, logistic regression modeling was performed to control for potentially confounding variables. Given our sample size, we were not able to include all variables for the multiple regression models. Based on clinical and statistical significance variables from the univariate analysis, we included inborn error of metabolism, multiple organ dysfunction, PRISM 2 score% FO (categorically divided by 10% intervals), Paw, and urine output at CRRT start into a multiple regression model. Variables with P value of >.2 were eliminated with a stepwise, backward selection approach. For parsimony, our final multivariable-adjusted model only included variables which remained significant at $P < 0.1$. SAS 9.2 (SAS Institute Inc, Cary, North Carolina) was used for statistical analysis.

RESULTS

In our cohort of patients weighing ≤10 kg, weight range was 1.3–10 kg (median 4.4 kg). Patient age range was 1 day-2.9 years old (median 69 days). Boys outnumbered girls by 3:2. The most common location for vascular access was the femoral site (57%); the most common size was 7F (57%); and the most common CRRT modality was continuous hemodialysis (60%). On average, these children were admitted to the ICU 2 days prior to CRRT initiation. More than two-thirds of these small children undergoing CRRT had multiple organ dysfunction as part of their acute illness. Survival among cohort in the ppCRRT weighing ≤10 kg was 36/84 (43%) (Table I). There were no differences between survivors and non-survivors with respect to age, weight, sex, catheter size, location, or CRRT mode. Survivors were more likely to have lower number of days in ICU prior to

CRRT, lower PRISM 2 scores at ICU admission and lower PRISM 2 scores, lower P_{aw}, lower % FO, and higher urine output at CRRT initiation. Children who were able to achieve dry weight during their CRRT course were more likely to survive than children remaining fluid overloaded (78% vs 35%, P = .002). Sepsis was the most common primary diagnosis (30%) followed by cardiac disease (19%), inborn errors of metabolism (15%), hepatic, pulmonary, oncology, renal, and other. Those with primary renal diagnosis had 80% survival, and those with hepatic diagnosis had the worst prognosis as none of the 9 infants survived. Etiology of renal disease which culminated in primary renal diagnosis include: autosomal recessive polycystic kidney disease, cortical necrosis, chronic kidney disease of undetermined etiology, congenital nephrotic syndrome, and bilateral renal agenesis. The number of subjects per diagnosis and survival by primary diagnosis are shown in Table II. The number of subjects and survival by center are shown in Table III.

The survival of children with weight #5 kg was similar to children weighing 5–10 kg (21/48 [44%] vs 15/36 [42%]; P = not significant). However, children ≤10 kg had lower survival than children in the ppCRRT Registry who were >10 kg (36/84 [43%] vs 166/261 [64%]; P < .001) (Figure 1).

Table IV shows univariate analysis for survival. Patients who initiated CRRT with >20% FO at the time of CRRT initiation had worse survival compared with those with <10% FO (OR = 4.1 [95% CI = 1.5–11.1]; P < .01). Patients with lower levels of FO (<10%) at CRRT initiation appeared to have better survival than those with 10%-20% FO (50%) but the difference was not statistically significant. OR for mortality were higher in those children with higher PRISM 2 scores, higher %FO, higher Paw at CRRT start, and more days in ICU prior to CRRT. Multiple regression analysis for mortality showed that PRISM 2 score at CRRT, urine output at CRRT and %FO prior to CRRT were independently associated with mortality (Table V). Specifically, after controlling for PRISM 2 score and urine output, those with >20% FO at the time of CRRT initiation had 4.9 times higher odds of death than those who initiated CRRT with <10% FO (95% CI = 1.3–17.7; P < .01). In addition, we performed a multiple regression analysis to determine if the interaction between PRISM 2 at CRRT initiation and FO was significant. Indeed, the interaction between these 2 variables was significant (df = 1; estimate 0.005 SE = 0.002, c 2 = 6.8, P value <.001 with an area under the curve of 0.76; Figure 2). Because infants with inborn error of metabolism receive CRRT for non-renal reasons, they have different characteristics. Differences between infants in the ppCRRT who were placed on CRRT due to inborn errors versus others are shown in Table VI Infants with inborn errors had lower weight, age, ICU length of stay, and days in the ICU scores before CRRT. They had lower Paw, higher glomerular filtration rate, higher PRISM 2 scores, higher urine output, and less FO at time of CRRT initiation. Although not statistically significant, those with inborn errors trended to have better survival than infants on CRRT for other indications (8/13 [61%] vs 28/71 [38%]; P = .22) A total of 170 circuits were described in the 48 infants #5 kg. A total of 251 circuits were described in the 36 subjects >5 kg. Circuits of infants#5 kg more commonly used heparin for anticoagulation, were initiated with a blood prime, had higher blood flows/kg, and had higher daily effluent volume/1.73 m2 (total daily clearance) (Table VII). No differences in circuit life between infants #5 kg and infants 5–10 kg were apparent.

DISCUSSION

In this prospective registry analysis of children ≤10 kg at CRRT initiation, we show that small children who require renal replacement therapy for AKI or inborn errors of metabolism have higher mortality than bigger children in the same registry. Although the CRRT prescription is different and technical considerations make CRRT more challenging, it is a procedure that can be used to support very small children. This analysis provides information about practice patterns, technical considerations, indications, variables associated with mortality, and outcomes in these children. This analysis parallels findings of the entire ppCRRT Registry that indicate that sepsis and multiorgan failure are very common reasons for CRRT even in small children. Moreover, survivors were more likely to start CRRT sooner after ICU admission, have lower Paw pressures at CRRT initiation, lower PRISM 2 scores, maintained more urine output before CRRT initiation, and less FO at CRRT initiation. Subjects who were unable to achieve dry weight after CRRT initiation had higher mortality. FO has been shown to be associated with mortality in critically ill children and adults.[3,12–18] We provide data suggesting that these effects are applicable to small children. After controlling for PRISM 2, FO, P_{aw}, urine output at CRRT, and the inability for CRRT to return patient to dry weight, all point to pulmonary edema as an etiology for mortality. We note the interaction between PRISM 2 score and FO at CRRT initiation was highly predictive of mortality. This data suggests that clinical management strategies to prevent FO and early initiation of CRRT for ultrafiltration may improve outcomes. However, more data is needed before evidence-based recommendations can be made. In the meantime, we contend that fluid provision should be viewed as a drug with potential side effects and potentially negative consequences. Cumulative volume imbalance (including intravenous flushes) should be tracked in all children at risk for FO, and the benefits of excessive fluid provision need to be weighed against potential detrimental effects on ultimate clinical outcome. Similarly, although early CRRT initiation could allow for improved nutritional provision, better metabolic balance, and less FO, these benefits need to be weighed against the potential risks of this therapy on an individual basis. Our data suggests that the clinician’s approach and prescription of CRRT for younger and smaller children differs from older and larger children, underscoring that there are size specific clinical considerations that impact therapy. The lack of pediatric specific equipment is a barrier to providing the best application of this therapy to small children with AKI. Because there are currently no CRRT devices approved by the US Food and Drug Administration for individuals weighing ≤11 kg, use of any of these devices is considered off-label in such children. A smaller filter is available in Europe,[19,20] and will be undergoing Food and Drug Administration Investigational Device Exemption trials in the US. Newer machines such as the Cardio Renal Pediatric Dialysis Emergency Machine[21] have been developed that use smaller extracorporeal volumes and lower blood flows. Evaluations of these machines are currently being performed in Europe. The 43% survival rates for infants ≤10 kg at CRRT initiation in this ppCRRT Registry is similar to those found by Symons et al when they reported a 38% survival rate from 5 tertiary centers published in 2003.[2] In addition, we found similar associations regarding survival and the primary medical diagnosis, with a trend toward better outcomes in infants with metabolic conditions, and no clear survival advantage among the various CRRT modalities. Unlike prior publications, our report identifies specific clinical

variables associated with survival, compares mortality between this cohort of small children and larger children, and analyzes technical and prescription differences between those <5 kg versus those >5 kg at the time of CRRT initiation. Unlike prior retrospective or single-center reports, strength of this analysis is derived from its prospective data collection from a large patient population across multiple hospitals and providers. Nonetheless, we acknowledge several important limitations. We acknowledge that many variables, such as complications of access placement, cause of death, and other interventions were not collected and included in this analysis. Therefore, because not all known or unknown variables are available, causal inferences cannot be made between exposure variables (such as FO) and outcomes (such as mortality). Before strong causal inferences can be made, more observational data and larger randomized clinical trials are greatly needed. We also acknowledge that registry data is subject to center and patient selection bias. Furthermore, because the registry did not suggest or give concrete guidelines on who is a CRRT candidate or when to initiate CRRT, the selection of patients may not encompass all sick infants ≤10 kg at these institutions during this time. Finally, we acknowledge that the data is derived from a cohort treated between 2001 and 2005 may not reflect current practice patterns, although the general clinical approach to the institution of CRRT in small children has not changed. This data will serve as comparison for new pediatric registry studies, such as the prospective pediatric AKI research group, which will begin to collect data in 2013. Although there is a continued trend towards CRRT in North America, peritoneal dialysis is commonplace in many places, which do not have access to CRRT technologies. Peritoneal dialysis provides some potential benefits, (no need for central catheter, lower cost, and lower complexity) over hemodialytic CRRT. Unfortunately, it is difficult to compare data from this registry to children who receive peritoneal dialysis.

Although most would agree that CRRT is superior for treatment of disease, which require high effluent rates (such as inborn error of metabolism), incorporation of acute peritoneal dialysis into multicenter registries such as the ppCRRT Registry would be invaluable. Although survival in children ≤10 kg is worse than in larger children, CRRT is feasible in even the smallest infants and a good proportion of these critically ill children do indeed survive. Numerous systematic issues need to be addressed to improve outcomes in this vulnerable population. Currently, there are minimal evidence-based guidelines for dialysis prescription in very small children and most clinicians use approaches based on experience in adults or larger children. The safety, efficacy, and clinical consequences of different approaches for priming the CRRT machine for therapy initiation need to be explored. New devices designed for infants and small children receiving CRRT need to be developed and tested. Improvement in the early diagnosis of AKI or metabolic conditions such as hyperammonemia, may allow for earlier or more effective CRRT provision. Any long-term clinical or survival benefits of CRRT provision versus use of peritoneal dialysis or more conventional hemodialysis techniques in this population has also yet to be determined. To answer these questions and advance the care provided to these children so that outcomes can be optimized, collaborative groups such as the ppCRRT will need continued support.

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ABBREVIATIONS

AKI	Acute kidney injury
CRRT	Continuous renal replacement therapy
%FO	Percent FO
FO	Fluid overload
ICU	Intensive care unit
P_{aw}	Mean airway pressure
ppCRRT	Prospective Pediatric Continuous Renal Replacement Therapy
	PRISMPediatric Risk of Mortality

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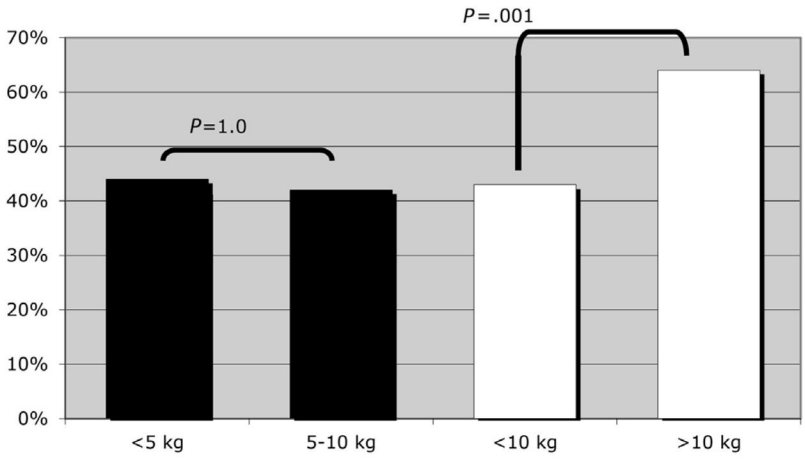

FIGURE 1.
survival data by weight

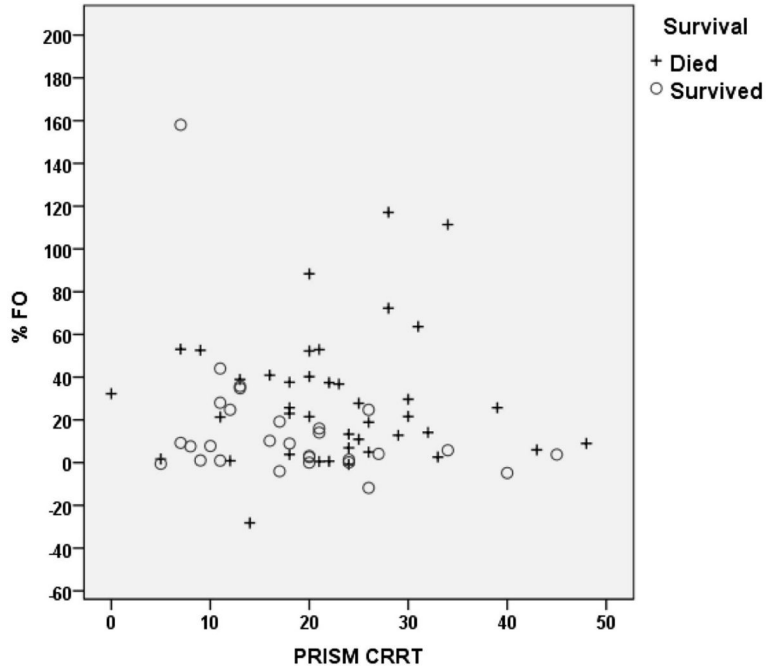

FIGURE 2.
Association between PRISM 2 scores and FO in children ≤10kg on CRRT

TABLE I

Demographics: Survivors versus non-survivors

	Survivors (N = 36)	Non-survivors (N = 48)	*P* value
Demographic variable			
Male sex	21/36 (58%)	30/48 (63%)	.82
Weight (kg) *	4.4 (2.8–7.8)	4.4 (3.2–7.1)	.71
Age (d) *	69 (9.5–289)	71 (10.2–286.2)	.96
ICU clinical data			
ICU admit length of stay (d) *	17 (7–34)	14 (3.5–25.5)	.12
Days in ICU prior to CRRT *	1 (0–3)	4 (1–13)	.01
Pressor dependency	23 (64%)	38 (79%)	.14
PRISM score at ICU admit *	16 (9–22) (n = 29)	21 (13–26.5) (n = 41)	.03
Inotrope no. at CRRT initiation *	2 (0–2)	1 (1–2)	.81
CRRT clinical			
data Paw @	11 (8–16) (n = 25)	16 (12–24) (n = 41)	.007
CRRT start *	19.5 (8.9–35.0)	20 (6.8–37.8)	.64
GFR mL/min/1.73 M2 @ CRRT start *			
PRISM II score @ CRRT start *	17 (11–24) (N = 30)	22 (18–28.5) (N = 41)	.03
FO @ CRRT start *	8% (0.36%–20%)	22.3% (4%–44%)	.03
Urine output (mL/kg/h) @ CRRT start *	1.1 (0.2–35)	0.6 (0.0–1.6)	.02
Paw @ CRRT conclusion *	10 (8–14) (N = 24)	17.5 (14–28) (N = 36)	<.001
Days of CRRT *	3.5 (2–10)	6 (1–14)	.71
CRRT catheter site			.79
Femoral vein	22 (63%)	26 (54%)	
Internal jugular vein	9 (26%)	13 (27%)	
Subclavian vein	3 (9%)	7 (15%)	
Other	1 (3%)	2 (4%)	
CRRT catheter size			.7
5F	3 (9%)	2 (4%)	
7F	21 (62%)	24 (54%)	
8F	7 (20%)	13 (29%)	
9F	3 (9%)	5 (11%)	
12.5F	0	1 (2%)	
CRRT modality			.59
CVVHD	19 (53%)	31 (65%)	
CVVH	7 (19%)	8 (16%)	
CVVHDF	10 (28%)	9 (19%)	

CVVH, continuous hemofiltration; *CVVHD*, continuous hemodialysis; *CVVHDF*, continuous hemodiafiltration; *GFR*, glomerular filtration rate.

*Median (IQR).

†Mean ± SD.

TABLE II

Differences in survival depending on primary diagnosis

Primary diagnosis	Number/total (% of cohort)	Survive	Non-survivors	Survival versus non-survival —*P* value
Sepsis	25/84 (30%)	9/25 (36%)	16/25 (64%)	.37
Cardiac disease	16/84 (19%)	6/16 (38%)	10/16 (62%)	.59
Inborn error of metabolism	13/84 (15%)	8/13 (62%)	5/13 (38%)	.15
Hepatic	9/84 (11%)	0/9 (0%)	9/9 (100%)	<.01
Oncology *	6/84 (7%)	3/6 (50%)	3/6 (50%)	.73
Primary pulmonary	5/84 (6%)	3/5 (60%)	2/5 (40%)	.44
Renal †	5/84 (6%)	4/5 (80%)	1/5 (20%)	.09
Other ‡	5/84 (6%)	3/5 (75%)	2/5 (40%)	.19

* 3 neuroblastoma, 2 acute lymphocytic leukemia, 1 hemophagocytic syndrome.

† Autosomal recessive polycystic kidney disease, cortical necrosis, unknown cause of chronic kidney disease, renal agenesis, congenital nephrotic syndrome.

‡ 2 nephrotoxin, 1 congenital diaphrmatic hernia, 1 Omenn's syndrome, status post bone marrow transplant, 1 censored.

TABLE III

Survival by centers

Center	Number	Survival
1	26	13/26
2	13	0/13
3	11	6/11
4	9	5/9
5	7	2/7
6	5	2/5
7	4	3/4
8	4	2/4
9	2	2/2
10	2	0/2
11	1	1/1

TABLE IV

Univariate analysis

Variable	Crude OR	*P* value
PRISM II Score @ CRRT start	1.05 (0.99–1.1)	.08
PRISM II score at ICU admit	1.05 (1.0–1.1)	.06
Pressor dependency	1.1 (0.7–1.7)	.69
FO	1.0 (0.99–1.0)	.07
FO groups		
<10% versus 10%–20%	1.34 (0.34–5.5)	.56
<10% versus >20%	4.1 (1.5–11.1)	.01
P_{aw} @ CRRT start	1.04 (0.99–1.1)	.09
P_{aw} @ CRRT conclusion	1.23 (1.1–1.4)	<.001
Days in ICU prior to CRRT	1.04 (0.99–1.1)	.11
Urine output (mL/kg/h) @ CRRT start	0.74 (0.6–0.96)	.02
Multiorgan dysfunction syndrome	5.0 (1.2–21.4)	.03
Inborn errors of metabolism	0.42 (0.12–1.4)	.15
Days in ICU prior to CRRT	1.04 (0.98–1.1)	.15
Hepatic	∞	NA
ICU admit length of stay	1.0 (0.99–1.0)	.81

NA, not applicable

TABLE V

Adjusted logistic regression analysis *

Variable	aOR	*P* value
PRISM II score at CRRT	1.1 (1.0–1.2)	.02
FO groups		
<10% versus 10%–20%	0.9 (0.17–4.67)	.25
<10% versus >20%	4.8 (1.3–17.7)	.01
Urine output (mL/kg/h) @ CRRT start	0.72 (0.53–0.97)	.04

Variables used in the model include: PRISM 2 score, P_{aw}, and urine output at CRRT, %FO (categorically divided by 10% intervals), multiple organ dysfunction, and inborn error of metabolism.

* 66/84 observations used for analysis (40 death vs 26 survival).

TABLE VI

Differences between subjects with primary diagnosis of inborn error of metabolism versus other

	Inborn error (N=13)	Other (N=71)	P value
Demographic variable			
Male sex	9/13 (69%)	30/71 (42%)	.55
Weight (kg) *	3 (2.8–3.5)	5.1 (3.3–7.8)	.001
Age (d) *	5 (4–7)	101 (21.5–332)	.001
ICU clinical data			
ICU admit length of stay (d) *	6 (2–8)	20 (7–42)	.001
Days in ICU prior to CRRT *	0 (0–0)	3 (1–11)	.001
PRISM score at ICU admit *	23 (17–31) (n = 11)	17 (10–23) (n = 59)	.43
Multiorgan system failure Missing data n = 24	3/13 (23%)	46/71 (65%)	.001
Inotrope no. at CRRT initiation *	1 (0–2)	1 (1–2)	.47
Survivor	8/13 (61%)	28/71(38%)	.22
CRRT clinical data			
P_{aw} @ CRRT start *	11 (8–16) (n = 10)	16 (12–24) (n = 57)	.001
GFR mL/min/1.73 m^2 @ CRRT start *	47.3 (29.6–60.0)	14.8 (7.9–24.2)	.001
PRISM 2 Score @ CRRT start *	26 (24–33) (n = 11)	20 (12–25) (n = 59)	.009
FO @ CRRT start *	1.3% (−0.5%–6.9%)	22.2% (3.5%–38.2%)	.001
Urine output (mL/kg/h) @ CRRT start *	5.4 (3.5–8)	0.7 (0.04–1.4)	.002
P_{aw} @ CRRT end *	8.5 (7–12) (N = 10)	16 (11–23) (N = 50)	.003
Days of CRRT *	2 (1–2)	6 (1–14)	.001
CRRT catheter site			.15
Femoral vein	8 (62%)	40(56%)	
Internal jugular vein	3(23%)	19(27%)	
Subclavian vein	0	7(15%)	
Other	2(15%)	2(4%)	
CRRT catheter size			.08
5F	1 (8%)	4 (6%)	
7F	11 (92%)	34 (50%)	
8F	0	20 (30%)	
9F	0	8 (12%)	
12.5F	0	1 (2%)	
CRRT modality			.82
CVVHD	9 (69%)	41 (58%)	
CVVH	2 (15%)	13 (18%)	
CVVHDF	2 (16%)	17 (24%)	

* Median (IQR)

TABLE VII

CRRT circuit data in children ≤10 kg

	≤5 kg (N = 170)	>5 kg (N-251)	*P* value
Anticoagulation protocol			<.001
Citrate	76 (45%)	155 (62%)	
Heparin	94 (55%)	96 (38%)	
Prime			<.001
Blood	164 (96.5%)	202 (80%)	
Saline	5 (3%)	29 (12%)	
Albumin	1 (0.5%)	20 (8%)	
Parameter			
Blood flow* (mL/kg/min)	12 (7.9–15.6)	6.6 (4.8–8.8)	<.001
Daily effluent volume* (mL/h/1.73 m^2)	3328 (2325–4745)	2321 (1614–2895)	<.001
Circuit life	28 (11–67)	37 (16–67)	.15

* Median (IQR)