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ORIGINAL ARTICLE

Advanced hemodynamic monitoring: feasibility of leveraging non-invasive electrocardiometry in critically ill pediatric patients requiring continuous kidney replacement therapy

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Abstract

Background Hemodynamic instability occurs in children receiving continuous kidney replacement therapy (CKRT). Electrocardiometry can help characterize hemodynamics beyond traditional blood pressure (BP) and heart rate (HR). We aimed to assess the feasibility and correlations of hemodynamic measurements obtained using electrocardiometry in children receiving CKRT.

Methods Prospective single-center observational study of pediatric patients receiving CKRT between 11/2019 and 3/2021. Patients who received extracorporeal membranous oxygenation, ventricular assist device, pacemaker, apheresis, no invasive BP, and COVID-19 were excluded. Electrocardiometry measured cardiac index (CI), HR, stroke volume variability (SVV), stroke volume index (SVI), and systemic vascular resistance index (SVRI) continuously; data were aggregated into 1-h epochs, and correlation coefficients were computed using Spearman's rank test.

Results Seventeen patients with a median age of 43 months (IQR 13–122). Median weight and fluid overload at CKRT start were 13.9 kg (IQR 8.79–29.80) and 14.4% (IQR 2.4–25.6%) + 171.46 mL/kg (IQR 31.10–307.41), respectively. All measurements obtained via ICON were of high quality and no adverse events were identified. CI had a negative correlation with SVRI ($r = -0.67$) and had a positive correlation with SVI ($r = 0.83$) and mean arterial pressure (MAP) ($r = 0.63$). HR did not correlate with any hemodynamic variables, while MAP only correlated with SVI ($r = 0.63$).

Conclusions Electrocardiometry can assess the hemodynamic profile of children receiving CKRT. Compensatory cardiovascular changes remain intact in children receiving CKRT, as evidenced by correlations between SVI, SVRI, CI, and MAP. Future studies should investigate how this technology could enable more individualized CKRT prescriptions and improve patient outcomes.

Keywords Continuous kidney replacement therapy · Electrocardiometry · Hemodynamics · Hypotension

Introduction

Acute kidney injury (AKI) is common in critically ill pediatric patients and AKI requiring dialysis (AKI-D) occurs in 5.7% [1]. Continuous kidney replacement therapy (CKRT) is the treatment modality of choice in children as it allows for tighter solute management, slower fluid removal, and liberation from fluid restrictions. CKRT is theorized to have a favorable hemodynamic profile because it minimizes fluid and osmolar shifts [2]. Despite these posited benefits, pediatric AKI-D has poor short- and long-term outcomes such as impaired kidney recovery, chronic kidney disease (CKD), dialysis dependency, and death [1, 3–6]. Identifying modifiable factors targeted

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at better treatment delivery has the potential to improve outcomes.

A possible modifiable factor contributing to CKRT-associated morbidity are the hemodynamic perturbations that occur during therapy. Our current understanding of the hemodynamic impact of CKRT has recently evolved with evidence generated from critically ill adults. Regional myocardial wall motion abnormalities and decreased myocardial perfusion occur during CKRT in adults, ultimately resulting in a dose-dependent increase in mortality [7]. In addition to these cardiac changes, adults receiving CKRT are at risk for early hypotension, defined by a decrease in systolic blood pressure by ≥ 20 mmHg or a decrease in MAP by 10 mmHg within the first hour of CKRT initiation, in up to 64% of connections. Early hypotension has been associated with a 1.56-fold increase in mortality [8]. Every 10% increase in the number of days of hypotension during dialytic therapy is associated with a 14% increase in mortality [9]. In children, although the rate of hypotension at CKRT is lower, ranging from 27 to 56%, it is similarly associated with increased illness severity, higher vasoactive infusion scores (VIS), and lower central venous pressures (CVP)—all of which could contribute to the morbidity and mortality of CKRT [6, 10, 11]. Pediatric patients may be at increased risk for hemodynamic perturbations given the larger extracorporeal circuit volume compared to the estimated circulating blood volume [12, 13]. Despite this, there has been little investigation into the CKRT-induced changes in hemodynamic parameters such as cardiac index (CI), systemic vascular resistance index (SVRI), and other circulatory measures in pediatric patients.

Adequately monitoring hemodynamics during CKRT can be challenging. Echocardiography has been used in adults on CKRT; however, it is intermittent in nature and is unlikely to optimally and actively capture dynamic cardiovascular changes associated with fluctuations in CKRT course and prescription [7]. Transpulmonary thermodilution techniques, via pulmonary artery (PA) catheters [14], can have around 10% deviation in the measured cardiac output (CO) while on CKRT compared to measurements while off CKRT [15]. Non-invasive continuous hemodynamic monitoring devices, such as electrocardiometry or ultrasound, could thus allow clinicians to avoid more invasive techniques while still being able to obtain advanced hemodynamic measurements.

Electrocardiometry (ICON, Osypka Medical GmbH, Berlin, Germany) is a non-invasive hemodynamic monitor that uses changes in thoracic bioimpedance during different phases of the cardiac cycle to calculate stroke volume (SV) and aortic blood flow velocity. ICON is based on the theory that, during diastole, erythrocytes are randomly oriented, leading to increased bioimpedance, while during systole, erythrocytes align, leading to decreased impedance [16]. The CO, systematic vascular resistance (SVR), and thoracic fluid content (TFC) are derived from combining SV with heart rate (HR) and MAP obtained from standard bedside cardiorespiratory monitors. This technology has been validated against the current gold standard Ficks measurement (thermodilution) during cardiac catherization and echocardiography in heterogeneous pediatric populations [17–21]. It has also been studied in pediatric sepsis with strong associations between SV and SV variability (SVV) demonstrated in patients with fluid-responsive shock [22].

This study had two key objectives. First, we aimed to investigate the feasibility of using electrocardiometry to obtain advanced hemodynamic variables during the routine care of critically ill children receiving CKRT. Second, we aimed to identify the relations between hemodynamic parameters collected using ICON in pediatric patients on CKRT. We hypothesized that normal hemodynamic interactions and compensatory mechanisms will remain intact over the first 48 h of CKRT.

Methods

We conducted a single-center prospective observational study of children 1–18 years of age who recieved CKRT in the pediatric or cardiac intensive care unit at Texas Children's Hospital between November of 2019 to March of 2021. Patients who recieved extracorporeal membranous oxygenation, ventricular assist device, pacemaker, apheresis, and those without invasive BP measurement, or with confirmed COVID-19 infection were excluded. This study was approved by the Baylor College of Medicine (IRB protocol H-43712) and informed consent obtained prior to connection to ICON.

ICON was used to obtain continuous hemodynamic data during 48 h after CKRT initiation. This specific time period was selected to assess the impact on hemodynamics during the early high-risk period of CKRT with exposure to new non-biologic materials. Once consented, patients were connected to ICON using four leads on the neck and left side of the thorax; placement of these leads were determined by the age and size of the patient per the manufacturer's instructions. Hemodynamic variables were collected at 1-min intervals and subsequently aggregated using medians into 1-h epochs including: cardiac index (CI), heart rate (HR), thoracic fluid content (TFC), stroke volume (SV), stroke volume variability (SVV), and systemic vascular resistance index (SVRI). If no CVP was obtained during the time frame, CVP was set to 10 mmH_2O to calculate SVRI. The measurements obtained via ICON were compared to previously published literature that established normative weight-based ranges for these cardiovascular variables [23]. Further, the

measurements collected by the ICON device are unaffected by hematocrit/hemoglobin concentration. This is of particular importance in our cohort of patients undergoing CKRT, who may have a fluctuating fluid status and thus a dynamically variable erythrocyte concentration. However, such ICON measurements are affected by other entities impacting impedance transthoracically, namely, extravascular fluid or air in the chest, and is something important to consider in data interpretation [17].

Demographic variables of interest included age, weight, height, body surface area, primary diagnosis, primary comorbid condition, history of organ transplant, and date of admission. Illness severity was calculated daily using the Pediatric Logistic Organ Dysfunction 2 score (PELOD-2) [24, 25]. Furthermore, data to calculate the vasoactive inotropic score was collected at CKRT initiation [26]. Fluid overload (%) was calculated at the start of CKRT as ((net intake (mL/kg) – net output (mL/kg))/weight (kg) $\times 100$ [27]. The patient's fluid balance (FB) was extracted from the electronic health record at hourly intervals.

Missing data were imputed using multiple imputation with chained random forest models using the "missRanger" package in R and R studio. The number of trees used was chosen by verifying the clinical appropriateness of the numbers as well as considering the imputed dataset that was closest to the median of the original dataset. Patient and hemodynamic characteristics were reported using medians and interquariles for continuous measures and counts and proportions for categorical variables. Correlation coefficients were computed using the Spearman's rank test.

Results

Feasibility

Over the recruitment period, 41 children received CKRT. We were able to approach 31 patients and obtained consent from 19 (61.3%) patients, 12 (38.7%) patients/families did not consent. Two patients who consented (6.5%) were later excluded. The first patient initially provided consent but subsequently withdrew it during the connection to the ICON device. The second patient experienced clinical decompensation within a few hours of ICON connection, necessitating the placement of an invasive hemodynamic monitoring device, leading to the discontinuation of ICON (Fig. 1). Only 4.4% of the data were missing; we identified two reasons for missing data. First, during patient bathing and hygiene events (which usually occurred between the hours of 2200 and 0400), ICON leads were removed and not replaced until reassessed by the research team. Second, ICON leads were also removed during imaging studies, such as chest x-rays and computed tomography, and led to data collection interruptions. Since these interruptions occurred mainly during the day shift (0700–1900) and the research team was usually notified of these events, the reconnection to the ICON could occur as soon as the patient returned to the ICU. Additionally, we identified that CVP was not regularly obtained in our units, with 55% of patients missing CVP measurements in our study.

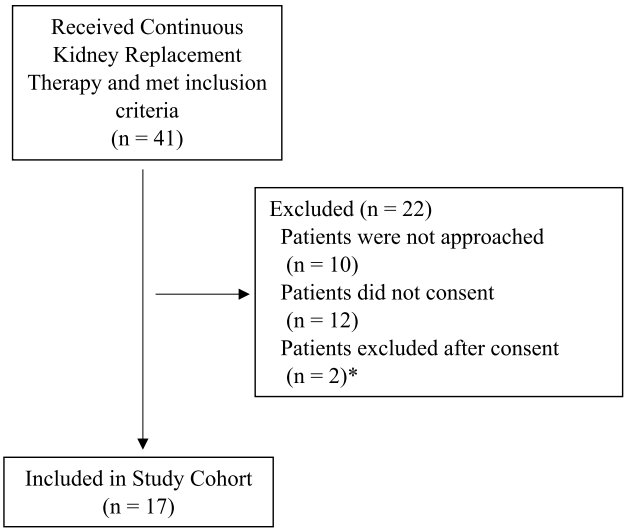

Fig. 1 Consort flow diagram of patient selection. *Two patients were excluded after consent; the first patient withdrew consent and the second patient experienced clinical decompensation necessitating invasive monitoring

Cohort demographics

We included 17 children who received CKRT with a median age of 43 months (IQR 13–122). Seven (41.2%) patients were male. The median weight and height were 13.9 kg (IQR 8.79–29.80) and 94 cm (IQR 77.00–124.40), respectively, with a median BSA of 0.63 m^2 (IQR 0.43–1.01). The most common reason for ICU admission was shock in 6 (35.3%), followed by oncological in 4 (23.5%). The most common indication for CKRT was fluid overload (FO) (94.1%) with or without AKI (88.2%). The median time between ICON connection and CKRT start was 24.5 h (IQR 21.50–41.00), with ICON trailing. The median fluid overload at CKRT start was 14.4% (IQR 2.4–25.6%). The median VIS at CKRT initiations was 4 (IQR 4–13.3) with a median PELOD-2 score of 7 (5–9). Seven (41.2%) patients died within 30 days of CKRT initiation (Table 1).

Hemodynamic profile

Over the first 48 h of ICON connection, median CI was 4.65 L/min/m^2 (IQR 3.53–5.97), median HR was 117 beats per minute (IQR 102–128), and median MAP was 78 mmHg (IQR 66–90). Over the same timeframe, the median SVI

Table 1 Demographics of the study population

Variable		Median [IQR] or *N* (%)
Study population, n		17
Age (months) at ICU admission		43.00 [13.00, 122.00]
Sex	Male	7 (41.2%)
	Female	10 (58.8%)
Weight (kg) at ICU admission		13.90 [8.79, 29.80]
Height (cm)		94.00 [77.00, 124.40]
Body surface area		0.63 [0.43, 1.01]
Vasoactive inotropic score (VIS)		4 [2–13.3]
PELOD-2 score		7 [5–9]
Admission reason	Respiratory	2 (11.8%)
	Post-operative	1 (5.9%)
	Shock	6 (35.3%)
	Cardiac post-operative	1 (5.9%)
	Digestive	2 (11.8%)
	Kidney	1 (5.9%)
	Hematologic/oncological	4 (23.5%)
History of transplant	No	15 (88.2%)
	Yes	2 (11.8%)
Chronic kidney disease	No	14 (82.4%)
	Yes	3 (17.6%)
Chronic kidney disease stage		
	Stage 3	3 (11.8%)
	Kidney failure	1 (5.9%)
Baseline creatinine (mg/dL)		0.31 [0.17, 0.55]
Time between CKRT start and icon connection (hours)		24.50 [21.50, 41.00]
Wt (kg) at start of CKRT		17.10 [11.20, 33.30]
Net fluid since ICU admission (mL)		3449.00 [304.00, 6090.50]
Net fluid at CKRT start (mL/kg)		171.46 [31.10, 307.41]
Fluid overload at CKRT start (%)		14.4% [2.4–25.6%]
CKRT reason	Fluid overload	16 (94.1%)
	AKI	15 (88.2%)
	Elevated ammonia	2 (11.8%)
Hemodialysis line location (%)	Jugular	10 (58.8%)
	Femoral	7 (41.2%)
Type of hemodialysis line (%)	Permanent	1 (5.9%)
	Temporary	16 (94.1%)
Hemoglobin at icon connection, unit		8.80 [8.20, 10.70]
30-day mortality	No	10 (58.8%)
	Yes	7 (41.2%)

was 39.22 mL/m^2 (IQR 30.57–49.50), median SVV was 14.63% (IQR 10.30–19.28), and median SVRI was 1153.03 dynes*s*m^2/cm^5 (IQR 960.62–1384.55). Median fluid balance was −0.77 mL/kg/hr (IQR −1.32 to −0.01 mL/kg/hr) and median TFC was 65.36 kΩ^{-1} (IQR 45.12–90.92). FB and TFC were weakly negatively correlated ($r = -0.11$). A graphical presentation of the hemodynamic data collected over the observation period can be seen in Fig. 2.

Previously determined normal ranges for CI, SVRI, SVI, and SVV for the median weight of the cohort (13.9 kg (IQR 8.79–29.80)) were referenced [23] (Table 2). All hemodynamic variables were within the weight-based normal range. A total of 629 (77%) of the SVV readings were above the fluid-responsive threshold of 10%, suggesting a preload-dependent state.

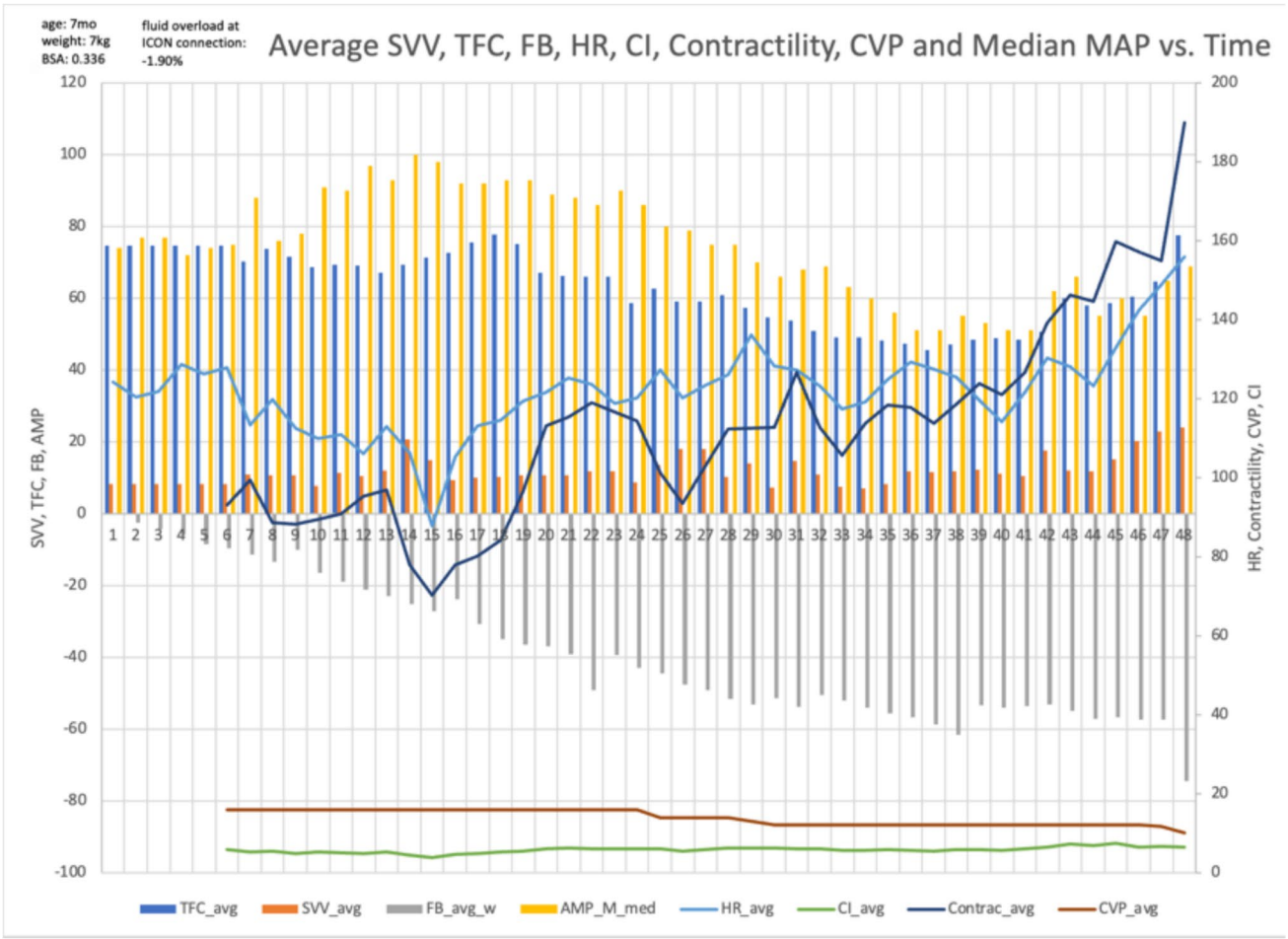

Fig. 2 Average SVV, TFC, FB, HR, CI, contractility, CVP, and median MAP vs. time from an individual patient. HR (in light blue) and contractility (in black) increase as FB (in grey) decreases from hour 30 to hour 48. This is preceeded by an increase in SVV (in orange) around hour 26. This patient demonstrates the hemodynamic response that is expected: an increase in HR and a decrease in MAP (denoted AMP in this figure), both of which indicate a state of hemodynamic instability, is preceded by an increase in SVV

Table 2 Normal ranges for hemodynamic parameters according to weight

Variable	< 10 kg	10–14.9 kg	15–19.9 kg	20–29.9 kg	30–49.9 kg	50–74.5 kg	> 75 kg
CI	4.43 (na)	5.01 (3.70–7.55)	4.93 (3.60–6.72)	4.69 (3.35–6.87)	4.37 (2.43–6.29)	3.37 (1.83–5.68)	2.91 (1.39–4.91)
SVRI	1056 (na)	1072 (693–1563)	1142 (775–1654)	1250 (833–1754)	1451 (998–2609)	2071 (1166–4407)	2703 (1328–5604)
SVV	9.0 (na)	10.6 (1.0–84.5)	10.4 (1.5–27.6)	9.0 (1.3–24.6)	12.4 (2.5–36.0)	18.2 (5.3–44.9)	18.8 (8.9–50.0)
SVI	37.0 (na)	49.3 (33.7–68.8)	54.0 (40.3–74.6)	56.5 (42.1–76.9)	55.4 (35.3–72.9)	47.1 (26.3–68.8)	39.3 (21.1–58.7)

Values obtained from Cattermole et al. [23]

Hemodynamic correlations

There was a strong positive correlation between SVI and CI ($r = 0.83$) and a moderate correlation between SVI and MAP ($r = 0.63$). We identified a moderate inverse relationship between CI and SVRI ($r = -0.67$). All other significant correlations were weak ($r < 0.5$ or > -0.5) (Table 3).

Correlations with mean arterial pressure

MAP was significantly correlated with all of the ICON hemodynamic variables, with the exception of TFC ($r = 0.03$). Despite these significant correlations, MAP only had a moderate positive correlation with SVI ($r = 0.63$). The remaining correlations were weak with $r < 0.5$ (CI ($r = 0.49$), SVRI ($r = 0.16$), SVV ($r = -0.08$), HR ($r = -0.28$),

Table 3 Spearman correlation coefficients of hemodynamic variables in the entire cohort

	CI	SVRI	SVV	SVI	HR	MAP	TFC	FB
CI		-0.67125	-0.39617	0.829385	0.062414	0.490797	-0.21474	-0.05685
SVRI			0.319199	-0.42916	-0.33996	0.161379	0.224611	-0.09873
SVV				-0.36699	0.225469	-0.07817	0.526204	-0.23527
SVI					-0.2856	0.627847	-0.06535	-0.09288
HR						-0.28083	0.006598	-0.06615
MAP							0.026037	-0.27124
TFC								-0.11251
FB								

*grey=significant correlation ($p<0.05$)

FB ($r = -0.27$)). The number and strength of significant correlations observed were expected, given that any measured value of MAP is intrinsically influenced by interactions between multiple measured hemodynamic variables.

Correlations with heart rate

HR had no strong correlations with any of the ICON hemodynamic variables. HR was weakly positively correlated with SVV ($r = 0.23$) and weakly negatively correlated with SVRI ($r = -0.34$), SVI ($r = -0.29$), and MAP ($r = -0.28$). HR also showed no significant correlation with CI ($r = 0.06$), TFC ($r = 0.01$), or FB ($r = -0.07$).

Discussion

We demonstrated the feasibility of utilizing ICON to measure hemodynamics parameters in critically ill children receiving CKRT with minimal missing data while also maintaining high-quality measurements. Pediatric CKRT patients in this study had normal CI, SVRI, SVI, and SVV over the first 48 h of CKRT [23]. When we evaluated the relations between hemodynamic parameters during CKRT, we found that, as expected, SVI had a positive correlation with CI (r = 0.83) and MAP ($r = 0.63$), while CI had a negative correlation with SVRI ($r = -0.67$). These hemodynamic correlations suggest that cardiovascular coupling mechanisms remain intact in pediatric patients receiving CKRT. Given these findings, we propose that this technology could be used to monitor and identify pathologic hemodynamic changes in individual children receiving CKRT.

We demostrated feasibility in using ICON to obtain hemodynamic data in children receiving CKRT as supported by the correlations SVI and CI ($r = 0.83$), SVI and MAP ($r = 0.63$), and CI and SVRI ($r = -0.67$). In this study, there was only 4.4% missing data, which was associated with routine clinical care in our pediatric and cardiac intensive care units. CVP was missing in 55% of our study population, likely deemed not clinically indicated by the bedside team. In the absence of measured CVP, a value of 10 mmHg was imputed and used to calculate the SVRI. While this approach enabled SVRI estimation, the imputed value could have introduced a bias: it could underestimate SVRI in patients with intravascular volume depletion and overestimate it in those with intravascular volume overload. In critically ill patients, continuous CVP measurements are often challenging to obtain, due to numerous medication requirements leading to limited central venous access. However, in future studies utilizing ICON, we aim to prioritize the collection of CVP data more consistently in patients with central venous access. This will enable more precise SVRI calculations, ultimately improving our understanding of the hemodynamic profiles in this patient population.

What is known about the adverse impact of dialysis on the cardiovascular system largely is derived from intermittent hemodialysis (iHD), whose rapid fluid shifts increase risk of cardiovascular-related morbidity and mortality [28]. The negative cardiovascular impact of iHD is thought to be related to acute fluid shifts that occur during dialytic therapy, leading to microvascular dysfunction and subclinical myocardial ischemia [29]. Evidence via single photon emission computed tomography has illustrated silent ST-segment depressions and decreases in myocardial perfusion in adults [30, 31]. More concerning, however, is the increase in myocardial strain that occurs in asymptomatic children during routine outpatient iHD [32]. These repeated bouts of myocardial ischemia during iHD could lead to progressive myocardial strain/dysfunction and contribute to the high rates of cardiovascular morbidity and mortality noted in this

population. Although CKRT provides slow fluid and solute removal, similar changes to myocardial function as described during iHD are also seen in adults receiving CKRT [7, 33]. In children requiring CKRT, up to 50% experience relative hypotension in the hour after CKRT initiation [5, 6, 34]. Given the significant risk of hemodynamic and cardiovascular compromise during hemodialysis, future research should further explore the potential utility of electrocardiometry. Specifically, these studies should evaluate its role not only in advancing our understanding of cardiovascular dysfunction during dialysis but also in mitigating hemodynamic instability in this vulnerable patient population.

Conventionally, current clinical measures of cardiovascular performance during CKRT rely on changes in HR and MAP. However, these measures did not strongly correlate with CI or SVRI in our study [7] suggesting that the traditional clinical hemodynamic monitors fail to capture the full hemodynamic picture. The weak and limited significant correlations identified with HR were not unexpected, as HR may fluctuate in response to changes in various hemodynamic variables measured but its value is not directly determined by these variables. HR is also commonly affected by various pharmaceuticals, particularly those used in the critically ill patient population. One such agent named dexmedetomidine, frequently utilized for sedation in patients requiring mechanical ventilation, can cause HR to significantly decrease and thus could be contributing to the limited correlations with respect to HR observed in this study [35, 36].

Using electrocardiometry has the benefit to non-invasively provide a continuous way to assess hemodynamic changes during CKRT. If validated in larger cohorts, ICON has the potential to provide dynamic biofeedback during CKRT, allowing a patient-driven model of fluid removal. Biofeedback fluid-titration methods used in CKRT are based primarily on ultrafilration (UF) and relative blood volume (RBV) algorithms, rather than the direct patient-centered hemodynamic variables measured by ICON [37]. If successfully integrated into a clinical algorithm, the additional variables such as SVV and CI could be utilized to predict overt intradialytic hypotension and subsequently allow clinicians to adjust CKRT prescription before further cardiovascular strain occurs. The limitation of the ICON device must be acknowledged, as performance can vary based on patient factors and clinical context. A meta-analysis by Sanders et al. reported a 42% mean percentage error in pediatric patients, which is above the 30% acceptable threshold, though is still comparable to other non-invasive tools. Some have proposed raising the acceptable error margin to 45% for such devices. Ultimately, ICON's continuous monitoring should complement, not replace, other tools for monitoring and clinical decision making.

TFC is an estimation of extravascular, intravascular, and intrapleural fluid components of the thorax [38]. Currently, the gold standards for assessing fluid status are net intake/output, fluid balance, and daily weights. Although useful, these measures focus more on the quantitative fluid status of the patient rather than their clinical response to such fluid exposure. TFC and other bioimpedance metrics have thus been postulated as a possible and more comprehensive, clinical measure of fluid status [39, 40]. However, the utility of TFC as a metric can be greatly affected by its inherent imprecision in not accounting for all components of fluid overload (FO). In our analysis, TFC did not significantly correlate with hemodynamic variables of the study and did not have the expected correlation to FB ($r = -0.11$). Thus, our data suggest that TFC might not be reliable for evaluating fluid status in children receiving CKRT. This is consistent with previous studies showing that bioimpedance methods of estimating fluid status, such as total body, intracellular, and extracellular water, during iHD are not superior to traditional clinical judgment with respect to volume management [40]. Our recent investigation has also revealed significant differences in CI, SVRI, and SVV comparing patients with and without FO > 15% at the initiation of CKRT [41]. These findings, in conjunction with the results identified in this study, potentially suggest that TFC measurements may be influenced by fluid overload, which requires further investigation.

Our study has several limitations. First, it is a single-center study with a smaller, limited sample size and therefore our study cohort may not representatively reflect the larger pediatric CKRT population. Second, our aggregatation of data into 1-h intervals led to a loss of granularity, which may have lead to inability to capture all hemodynamic fluctuations that occurred during our observation period. Data was aggregated into such 1-h epochs in order to reduce signal noise and optimize modeling in this feasibility study. However, a shorter time window, especially in the immediate period following CKRT initiation, may capture clinically relevant dynamics otherwise obscured and is critical to explore in future hypothesis-driven studies. Third, 55% of CVP measurements were missing and required imputation which as previously stated could have led to either an under- or over-estimation of the patient's calculated SVRI; given the small sample size and overarching goal to investigate feasibility of using ICON in children receiving CKRT, we did not complete a sensitivity analysis. Fourth, 4.4% of our advanced hemodynamic data was missing and required imputation; missingness was non-random in nature as ~ 70% was associated with evening patient baths. Fifth, the median time to ICON connection was 24.50 h which did not allow us to capture the initial connection to CKRT. The initial 24 h has been previously described as a high-risk timepoint for hemodynamic compromise and

future studies should target this time frame. Sixth, pharmaceutical agents utilized during hospitalization and while on CKRT that affect HR could have contributed to the limited and disparate correlations observed with HR specifically. Seventh, this study's inability to compare ICON data to reference standards like echocardiography or thermodilution limits assessment of measurement accuracy and clinical utility. However, ICON/electrocardiometry has been previously compared to data collected by cardiac MRI, ECHO, and pulmonary catheterization, and researchers have identified good correlations. As a feasibility study, the goal was to assess the practicality of continuous, non-invasive monitoring in pediatric CKRT patients rather than validate absolute accuracy. Thus, we did not utilize these other modalities (ECHO, cardiac MRI, etc.) to compare our ICON values, which should be the one of the targets of future research. Future studies should also prioritize device validation using continuous data early in CKRT to better determine the clinical applicability of ICON-derived metrics.

Conclusion

Our findings suggest that electrocardiometry can be used to continuously monitor advanced hemodynamic measures in children receiving CKRT. Using electrocardiometry, we identified that expected compensatory cardiovascular changes remain intact in pediatric patients during CKRT as evidenced by correlations between SVI, SVRI, CI, and MAP. However, current hemodynamic variables we obtain at bedside (MAP and HR) did not strongly correlate with CI. Validation in a larger sample size can allow clinicians to apply real-time hemodynamic data to provide individualized CKRT prescriptions in our quest to provide personalized precision medicine in the ICU.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00467-025-06860-9.

Data availability The data used in this study were obtained from retrospective chart reviews of patient medical records and are not publicly available due to privacy and institutional regulations.

Declarations

Conflict of interest The authors declare no competing interests.

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