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RESEARCH

Risk of in-hospital Deterioration for Children with Single Ventricle Physiology

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Abstract

Children with single ventricle physiology (SV) are at high risk of in-hospital morbidity and mortality. Identifying children at risk for deterioration may allow for earlier escalation of care and subsequently decreased mortality.

We conducted a retrospective chart review of all admissions to the pediatric cardiology non-ICU service from 2014 to 2018 for children < 18 years old. We defined clinical deterioration as unplanned transfer to the ICU or inpatient mortality. We selected children with SV by diagnosis codes and defined infants as children < 1 year old. We compared demographic, vital sign, and lab values between infants with and without a deterioration event. We evaluated vital sign and medical therapy changes before deterioration events.

Among infants with SV (129 deterioration events over 225 admissions, overall 25% with hypoplastic left heart syndrome), those who deteriorated were younger ($p = 0.001$), had lower baseline oxygen saturation ($p = 0.022$), and higher baseline respiratory rate ($p = 0.022$), heart rate ($p = 0.023$), and hematocrit ($p = 0.008$). Median Duke Pediatric Early Warning Score increased prior to deterioration ($p < 0.001$). Deterioration was associated with administration of additional oxygen support ($p = 0.012$), a fluid bolus ($p < 0.001$), antibiotics ($p < 0.001$), vasopressor support ($p = 0.009$), and red blood cell transfusion ($p < 0.001$).

Infants with SV are at high risk for deterioration. Integrating baseline and dynamic patient data from the electronic health record to identify the highest risk patients may allow for earlier detection and intervention to prevent clinical deterioration.

Keywords Single ventricle · Clinical deterioration · Interstage · Early warning score

Background

Among pediatric cardiac patients, those with single-ventricle physiology (SV) have the highest morbidity and mortality rates, especially during the first year of life [1–3]. During the interstage period, or the time between the first two stages of single ventricle palliation, these infants have parallel circulation resulting in a fragile balance between systemic and pulmonary blood flow and are vulnerable to subtle changes in hemodynamic and respiratory status [2]. Children with SV admitted to the hospital often require unplanned transfer to the intensive care unit after clinical deterioration, which is associated with increased morbidity and mortality [4, 5]. However, studies have found that some unplanned ICU transfers may be preventable with opportunities to intervene prior to clinical deterioration if at-risk patients were to be detected earlier [6, 7].

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Multiple pediatric early warning scores have been developed to facilitate earlier detection of clinical signs and symptoms of deterioration in pediatric patients [8]. Detecting impending deterioration in children with SV is especially difficult due to limitations in technology for patient monitoring and their baseline vital sign abnormalities [9]. The Cardiac Children's Hospital Early Warning Score (C-CHEWS) was developed and validated at a single center to predict cardiac arrest or ICU transfer among inpatient pediatric patients with heart disease [10]. While this score had high sensitivity and specificity (67% and 94% respectively) for identifying those on acute care floors at risk for deterioration, the median lead time for a critical C-CHEWS score was only two hours, which may limit the ability to intervene to prevent deterioration [10].

In this study, we describe a cohort of pediatric cardiology patients who experience a clinical deterioration event in a non-ICU hospital environment. Further, we characterize infants with SV by assessing baseline difference between those who deteriorated and those who remained stable as well as by evaluating vital signs and medical therapy changes around a deterioration event. Our goal is to better identify at-risk children to guide resource allocation to optimize care and to reduce in-hospital morbidity and mortality.

Methods

Study Design and Population

We performed a retrospective cohort analysis of pediatric cardiology patients admitted to Duke Children's Hospital from 2014 to 2018. We included all admissions in which a patient spent time on the non-intensive care inpatient pediatric cardiology service. The study was approved by the Duke University Medical Center Institutional Review Board (Pro00102839).

Study Outcomes and Definitions

The primary outcome was a clinical deterioration event. We defined deterioration as an unplanned transfer to the intensive care unit or a mortality on the non-intensive care inpatient service. We did not include planned admissions to the intensive care unit from the Emergency Department or the operating room, or transfers from the labor and delivery or neonatal wards. Patients could have more than one admission during the study period and could have more than one deterioration event per admission. We extracted patient data from the electronic health record (EHR) including demographics, vital signs, lab values, orders, and medication administration information. We defined emergent admission as those who presented through the emergency department. We defined urgent admission as those admitted due to clinical concern, such as following a clinic evaluation on the same day, but did not require emergency room care. We defined admission source based on how a child was initially admitted to the hospital for a given admission. We defined previous location as the service a child was on prior to the pediatric cardiology service. We defined SV based on diagnosis codes (see appendix in Supplemental Materials for full list of diagnosis codes) and confirmed the primary cardiac diagnosis as well ventricular dominance by manual review of echocardiogram reports. We also grouped infants based on surgical intervention: Norwood, PA band, shunt, Glenn, other, or none. Norwood included infants with Norwood repair with either RV-PA conduit or modified Blalock-Thomas-Taussig shunt. Shunt included infants with surgical systemic to pulmonary shunt or ductal stent for ductal dependent pulmonary blood flow. Glenn included infants with superior cavopulmonary anastomosis such as bidirectional Glenn and Kawashima. We grouped infants with left dominant as well as biventricular dominance together as left dominant [11]. We defined a sub-cohort of infants with SV based on age at admission less than one year.

We evaluated baseline vital sign and lab values on presentation to the inpatient pediatric cardiology service. We recorded baseline vital signs as the mean vital sign values during the first 24 h after presentation to pediatric cardiology. We recorded baseline lab values within 24 h prior to presentation to pediatric cardiology (e.g. if patient transferred from ICU or emergency department to the inpatient team). If no preceding lab value was obtained, we recorded the first lab value obtained in the first 24 h after presentation to pediatric cardiology.

We evaluated vital signs and Duke-Pediatric Early Warning Score (D-PEWS) over the 48 h preceding each deterioration event. We divided this period into eight consecutive six-hour blocks with the last block ending one hour prior to the deterioration time. For each block, we recorded mean vital signs or D-PEWS score for each patient. D-PEWS is our institutional bedside scoring system that assesses a score across three physiologic domains (behavior, cardiovascular, and respiratory) as well as parental and nursing concern (Supplemental Fig. 1), and it is based off the previously validated C-CHEWS warning score [10]. A higher score suggests more physiologic derangement and a score of three in any single domain or a composite score of four or higher triggers a rapid response evaluation to assess need for escalation of care or transfer to the ICU. The nursing team records a D-PEWS score every four hours for patients on the inpatient pediatric cardiology service.

We evaluated changes in therapy associated with each deterioration event. We grouped therapy by medication

class as identified within the electronic health record (antiarrhythmic, antibiotic, vasopressor). Fluid boluses included both lactated ringers IV bolus and sodium chloride 0.9% IV bolus. To account for therapies that may have been delivered in response to clinical changes leading to the deterioration event, the 24-hour deterioration window included the four hours prior to an event and the 20 h after an event. The 24-hour pre-deterioration window was the preceding 24 h (i.e. starting 28 h prior to an event). We defined therapy use as a binary outcome for each window if any qualifying medication or therapy was given.

Data Analysis

We performed manual verification on a randomly selected subset of twenty admissions to ensure accurate labeling of deterioration events as well as to verify key data points including patient age, vital signs, and lab values. We used standard summary statistics with counts and percentages for categorical variable and median and interquartile values (IQR) for continuous variables. We assessed differences between admissions with and without deterioration events using Chi squared and Wilcoxon rank-sum tests as the continuous outcomes were not normally distributed. We assessed changes in vital signs and D-PEWS prior to deterioration event using linear mixed models with random intercept and random slope. We used McNemar's test to assess changes in therapy with deterioration. We considered p values < 0.05 statistically significant. We did not correct for multiple comparisons as this was a hypothesis generating study. We performed all analysis using Python 3.8 (python.org: release date Oct. 14, 2019).

Results

Overall Patient Characteristics

The overall pediatric cardiology cohort included 1612 admissions across 992 children, with deterioration occurring in 288 admissions (18%). Overall, 26 patients died. The median age at admission was 1.6 years (IQR 0.3, 9.6), the median length of stay was 6.2 days (3.4, 14.8), and 418 admissions (26%) involved children with SV. Hypoplastic left heart syndrome was the most common single ventricle diagnosis (114 admissions [27%]). Children with a deterioration event were younger on admission (0.6 years [0.1, 5.5] v 2.0 years [0.4, 10.3]; $p < 0.001$), had longer length of stay (21.6 days [9.9, 61] v 5.2 days [3.2, 10.3]; $p < 0.001$), and were more likely to have SV (38% v 23%; $p < 0.001$) (Table 1). They were less likely to be admitted electively (17% v 40%; $p < 0.001$ across admission types) and less likely to have private insurance (37% v 44%; $p = 0.021$).

Children of any age with SV (relative risk 1.74 [95% CI 1.41–2.15]) and particularly infants with SV (RR 2.15 [1.72–2.68]) had significantly higher risk of deterioration compared to those with two-ventricle circulation.

Among this high-risk cohort of infants with SV, there were 227 total admissions across 100 infants with 75 admissions (33%) experiencing 129 total deterioration events. Overall, seven infants with SV died. Hypoplastic left heart syndrome (HLHS) was the most common primary cardiac diagnosis (57 admissions [25%]). Deterioration was relatively more common for infants following Norwood repair (39% v 14%) and less common following Glenn (8% v 22%) or PA band (8% v 17%) ($p = 0.001$ across surgical repair types). Infants with SV who deteriorated were younger at the time of hospital admission (0.0 years [0.0, 0.4] v 0.4 years [0.0, 0.7]; $p < 0.001$), had longer length of stay (60.8 days [26.1, 117.9] v 7.9 days [3.9, 19.9]; $p < 0.001$), and were less likely to be admitted electively (11% v 32%; $p < 0.001$ across admission types) compared to infants with SV who did not deteriorate (Table 2). Infants most commonly transferred to the pediatric cardiology service from the ICU (49% of infants). No difference in rate of deterioration was seen based on previous location prior to the pediatric cardiology service ($p = 0.36$).

HLHS was more common (32% v 22%) and tricuspid atresia was less common (8% v 18%) for infants with a deterioration event, but this association was not significant when evaluated across all cardiac diagnoses ($p = 0.08$). There was no significant association with ventricular dominance ($p = 0.43$).

Infants with SV Baseline Characteristics

Upon presentation to the inpatient pediatric cardiology service, infants with SV who deteriorated had younger age (median 0.2 years [0.1, 0.4] v 0.4 years [0.1, 0.7]; $p = 0.001$), lower baseline oxygen saturation (80% [78%, 88%] v 83% [79%, 91%]; $p = 0.022$), and higher baseline respiration rate (46 breaths per minute [39, 52] v 43 [37, 49]; $p = 0.022$), heart rate (141 beats per minute [129, 151] v 135 [125, 146]; $p = 0.023$) and hematocrit (43.4 [40.6, 47.6] v 40.3 [37.0, 45.7]; $p = 0.008$) (Table 3). No difference was seen in baseline systolic blood pressure ($p = 0.12$), white blood cell count ($p = 0.10$), sodium ($p = 0.53$), or creatinine ($p = 0.46$).

Characteristics Around Deterioration

Infants with SV experienced 129 deterioration events. Deterioration occurred a median of 8.0 days (2.0, 23.1) after presentation to the pediatric cardiology service (Table 4).

Table 1 Characteristics of all pediatric cardiology patients. Data presented as median (interquartile values) and count (percentage). HLHS: hypoplastic left heart syndrome; uAVCD: unbalanced atrioventricular canal defect; DILV: double inlet left ventricle

		Admissions Without Deterioration (n = 1324)	Admissions With Deterioration (n = 288)	p-value
Age at admission (years)		2.0 (0.4, 10.3)	0.6 (0.1, 5.5)	**< 0.001**
Length of stay (days)		5.2 (3.2, 10.3)	21.6 (9.9, 61.0)	**< 0.001**
Male		735 (56)	173 (60)	0.18
Race				0.17
	White	693 (53)	137 (48)	
	Black	372 (28)	84 (29)	
	2 or More	103 (8)	33 (12)	
	Other	140 (11)	32 (11)	
Hispanic		110 (9)	29 (10)	0.45
Single Ventricle		309 (23)	109 (38)	**< 0.001**
Primary Cardiac Diagnosis				0.22
HLHS	77 (25)	37 (34)		
Tricuspid Atresia	41 (13)	13 (12)		
uAVCD	53 (17)	16 (15)		
DILV	23 (7)	12 (11)		
Other	115 (37)	31 (28)		
Ventricular Dominance				1
Left dominant	155 (50)		54 (50)	
Right dominant	154 (50)	55 (50)		
Admission source				**< 0.001**
	Born in hospital	85 (6)	46 (16)	
	Clinic	53 (4)	15 (5)	
	Home	881 (67)	141 (49)	
	Transfer from another hospital	270 (20)	78 (27)	
Admission type				**< 0.001**
	Emergency	329 (25)	103 (36)	
	Newborn	86 (7)	45 (16)	
	Elective	524 (40)	49 (17)	
	Urgent	385 (29)	91 (32)	
Payor type				**0.021**
	Private	584 (44)	105 (37)	
	Public	740 (56)	183 (64)	

Twelve patients deteriorated within 24 h of presentation. While there were no significant or consistent changes across individual vital signs during the 48 h prior to a deterioration event – including respiration rate ($p = 0.31$), heart rate ($p = 0.98$), systolic blood pressure ($p = 0.94$), or oxygen saturation ($p = 0.55$) – there was a significant increase in composite D-PEWS scores (median 1.0 [1.0, 2.0] 12 h before event v 2.0 [1.0, 2.5] 6 h before; $p < 0.001$).

We evaluated the therapy infants with SV received with deterioration compared to the preceding 24 h (Table 5). Seventy-nine infants (61%) previously receiving oxygen support received an increase in oxygen support and 23 infants (18%) were newly started on supplemental oxygen. Deterioration was associated with an increase in the overall number of infants receiving any supplemental oxygen (98 infants with deterioration v 83 infants in preceding 24 h; $p = 0.012$), receiving fluid boluses (30 infants v 2 infants; $p < 0.001$), receiving a packed red blood cell transfusion (22 infants v 2 infants; $p < 0.001$), receiving antibiotics (69 infants v 51 infants; $p < 0.001$), and receiving vasopressor support (12 infants v 2 infants; $p = 0.009$). No significant difference was seen in the use of antiarrhythmic therapy (16 infants v 14 infants, $p = 0.62$).

Discussion

In this study, we describe deterioration events in pediatric cardiology patients, who are at high risk for clinical deterioration, with an event occurring in 18% of admissions. Infants with SV represent an even higher risk population, with deterioration occurring in one-third of admissions. Our

Table 2 Characteristics of all infants with single ventricle physiology. Data presented as median (interquartile values) and count (percentage). HLHS: hypoplastic left heart syndrome; uAVCD: unbalanced atrioventricular canal defect; DILV: double inlet left ventricle

		Admissions Without Deterioration (n = 152)	Admissions With Deterioration (n = 75)	p-value
Age at admission (years)		0.4 (0.0, 0.7)	0.0 (0.0, 0.4)	**< 0.001**
Length of stay (days)		7.9 (3.9, 19.9)	60.8 (26.1, 117.9)	**< 0.001**
Male		99 (65)	46 (61)	0.68
Race				0.54
	White	92 (61)	41 (55)	
	Black	28 (18)	20 (27)	
	2 or More	13 (9)	5 (7)	
	Other	19 (13)	9 (12)	
Hispanic		10 (7)	8 (11)	0.43
Primary Cardiac Diagnosis				0.08
HLHS	33 (22)	24 (32)		
Tricuspid Atresia	27 (18)	6 (8)		
uAVCD	21 (14)	14 (19)		
DILV	14 (9)	10 (13)		
Other	57 (38)	21 (28)		
Surgical Repair				**0.001**
Norwood		21 (14)	29 (39)	
Shunt		37 (24)	21 (28)	
PA Band		26 (17)	6 (8)	
Glenn		34 (22)	6 (8)	
Other		22 (15)	9 (12)	
None		11 (7)	4 (5)	
Ventricular Dominance				0.43
Left dominant	85 (56)		37 (49)	
Right dominant	67 (44)	38 (51)		
Admission source				**< 0.001**
	Born in hospital	26 (17)	31 (41)	
	Clinic	11 (7)	3 (4)	
	Home	90 (59)	24 (32)	
	Transfer from another hospital	23 (15)	13 (17)	
Previous location				0.36
ICU		74 (49)	38 (51)	
New Admission		46 (30)	18 (24)	
Emergency Department		23 (15)	10 (13)	
Other		9 (6)	9 (12)	
Admission type				**< 0.001**
	Emergency	44 (29)	22 (29)	
	Newborn	26 (17)	31 (41)	
	Elective	49 (32)	8 (11)	
	Urgent	33 (22)	13 (17)	
Payor type				0.44
	Private	54 (36)	22 (29)	
	Public	98 (65)	53 (71)	

analysis suggests that there are baseline differences between infants with SV who deteriorate and those who do not. There were also significant changes in respiratory support and medication administrations with deterioration.

Our data demonstrated a significant increase in our institutional warning score, D-PEWS, prior to a deterioration event. Notably when averaged across all infants with SV, we did not see a significant change in any single vital sign prior to event, suggesting a composite warning score has higher sensitivity for detecting a deterioration event. However, our data also highlight the limitations of the current warning system. The D-PEWS score increase was predominately in the final six-hour window before a deterioration event, which is similar to prior assessment of the C-CHEWS warning score

Table 3 Baseline vital signs and lab values of infants with single ventricle physiology (SV infants) on presentation to the non-intensive care pediatric cardiology service grouped by those who did and did not have a subsequent deterioration event. Data presented as median (interquartile values)

	SV infants without deterioration	SV infants with deterioration	p-value
Heart rate (beats per minute)	135 (125, 146)	141 (129, 151)	**0.023**
Respiration rate (breaths per minute)	43 (37, 49)	46 (39, 52)	**0.022**
Oxygen saturation (%)	83 (79, 91)	80 (78, 88)	**0.022**
Systolic blood pressure (mmHg)	91 (82, 97)	89 (80, 96)	0.12
Age (years)	0.4 (0.1, 0.7)	0.2 (0.1, 0.4)	**0.001**
Weight (kg)	5.3 (3.8, 6.9)	4.5 (3.5, 5.4)	**0.04**
White blood cell count	11.8 (8.5, 14.4)	12.4 (10.5, 18.2)	0.10
Hematocrit	40.3 (37.0, 45.7)	43.4 (40.6, 47.6)	**0.008**
Sodium	138 (136, 140)	138 (136, 140)	0.53
Creatinine	0.3 (0.2, 0.4)	0.3 (0.2, 0.5)	0.46
Anion gap	10 (9, 13)	11 (9, 13)	0.25

that demonstrated that a critically elevated score occurred a median of only two hours before an event [10]. This short time horizon may limit the efficacy of interventions to prevent deterioration from occurring. Additionally, the median score was increased to only 2.0. In our scoring system, a composite D-PEWS score must be at least 4 to trigger evaluation from a rapid response team. Thus, most infants with SV would not be detected before deterioration events, suggesting that a more robust risk assessment tool to allow for earlier identification of at-risk patients is needed.

Our data also help to describe the etiology of deterioration in infants with SV. Desaturation is a major cause of deterioration with 18% of events requiring new respiratory support and 61% of events requiring an increase in respiratory support. A recent study of children with congenital heart disease in Japan similarly identified desaturation as the most common cause for rapid response team evaluation [12]. This likely reflects the delicate balance between systemic and pulmonary blood flow in the parallel circulation of infants with SV physiology [13]. Acute arrhythmia was uncommon in our cohort, as we did not identify a significant increase in antiarrhythmic usage. It is possible that infants who already required one antiarrhythmic medication acutely required addition of a second therapy, such as addition of a second agent for treatment of supraventricular tachycardia, which would not be captured in our analysis. Additionally, only a small subset (9%) of infants with SV required initiation of vasopressor therapy suggesting that acute low cardiac output was uncommon once patients were on the non-ICU team.

Our study identified small but statistically significant baseline differences in oxygen saturation, respiration rate, heart rate, and hematocrit between infants with SV who deteriorated and those who did not. Previous studies evaluated precursors for cardiopulmonary resuscitation or intubation for interstage infants with SV in the cardiac intensive care unit and similarly found lower oxygen saturation and higher heart rates to be risk factors [9, 14]. These previous studies had not identified respiration rate as a significant predictor. This discrepancy is possibly due to the difference in patient population, as prior studies analyzed children admitted to the intensive care unit, who likely have different baseline physiology.

Optimizing clinical deterioration detection systems will require integrating both vital sign and lab value data as well as additional information from the EHR such as changes in medication or respiratory support. Accordingly, event detection models that incorporate a child's underlying cardiac physiology as well as vital sign parameters may have increased performance [15]. Combining EHR derived data with machine learning methods has shown early promise for improved event detection in the pediatric cardiac intensive care unit [16–18]. Further work will be required to develop more sophisticated clinical deterioration models for children and infants on non-ICU services, which likely have distinct arrays of available patient information. Automatic extraction of data from the EHR may also help lessen the workload on bedside nurses required by manual scoring-based warning systems. It will be critical to identify the highest risk infants, especially as care bundles targeting high-risk patients have reduced the incidence of in-hospital cardiac arrest [19].

Our data indicated deterioration was relatively more common in patients with Medicaid or Medicare insurance, suggesting a difference in outcomes based on the socioeconomic status of patients' families. This finding is similar to previous studies that have linked race, socioeconomic factors, and insurance type with divergent outcomes [20–22]. This disparity could potentially be due to differences in outpatient care prior to admission, in initial triage, or in care provided during hospitalization. Additionally, a recent study suggests that maternal metabolic syndrome and maternal placental syndrome may be significant prenatal contributors to disparities in outcomes for children with congenital heart disease [23]. We did not identify a difference in outcomes by payor type among the small cohort of infants with SV,

Table 4 Change in vital signs prior to deterioration for infants with single ventricle physiology. We divided this period into eight consecutive six-hour blocks with the last block ending one hour prior to the deterioration time, and we recorded the mean vital sign or D-PEWS score for each patient during each block. Data presented as median (interquartile values) across 129 deterioration events

Vital sign	48 h before	24 h before	12 h before	6 h before	p value
Respiration rate (breaths per minute)	46 (37, 55)	48 (40, 54)	46 (41, 53)	48 (40, 58)	0.31
Heart rate (beats per minute)	142 (132, 152)	144 (134, 153)	141 (129, 149)	145 (134, 153)	0.98
Systolic blood pressure (mmHg)	92 (85, 98)	93 (85, 99)	93 (86, 99)	94 (85, 100)	0.94
Oxygen saturation (%)	82 (77, 85)	81 (78, 85)	81 (77, 86)	81 (76, 85)	0.55
D-PEWS	1.0 (0.0, 1.8)	1.0 (0.5, 2.0)	1.0 (1.0, 2.0)	2.0 (1.0, 2.5)	**< 0.001**

possibly because more hospital resources are provided to these infants through a single ventricle monitoring program [24]. Further research is essential to ensure all children receive equitable care and outcomes regardless of socioeconomic or racial background.

We recognize limitations in our study. While we selected infants based on SV physiology with grouping by primary cardiac diagnosis, we acknowledge these are not homogenous groups. We did not incorporate additional anatomic or functional information, such as degree of AV valve regurgitation, systemic ventricle systolic function, or type of pulmonary blood source, which have been shown to contribute to patient morbidity and mortality [25, 26]. Incorporating these parameters would likely allow for detection of further difference between groups and would strengthen a clinical deterioration detection system. Additionally, our definition of clinical deterioration is based on patient transfer records. While we evaluated changes in therapy around deterioration, we were unable to determine the exact cause for acute deterioration for each patient. As a result, there may be additional clinical changes we were unable to recognize. Finally, as a single center study, our results may not be applicable for all pediatric medical centers. Other institutions may have different thresholds for transferring a patient to the ICU or different baseline levels of acuity on non-ICU pediatric cardiology teams, which could affect the characteristics of those patients who deteriorate [27, 28].

Conclusion

Infants with SV are at high risk for clinical deterioration. There are baseline differences in admission characteristics, vital signs, and lab values between infants with SV who have a deterioration and event and those who remain stable. Additionally, there are significant changes in oxygen support and medical therapy associated with deterioration events. Integrating baseline and dynamic patient data from the Electronic Health Record to identify the highest risk patients may allow for earlier detection and intervention and prevent clinical deterioration.

Table 5 Change in therapy with deterioration for infants with single ventricle physiology. Assessed use of therapy in the 24-hour window associated with deterioration and the preceding 24 h window. Data presented as count (percentage) across 129 deterioration events. pRBC: packed red blood cell

Therapy	Before Deterioration	Deterioration	New administrations	p-value
Antiarrhythmic	14 (11)	16 (12)	3 (2)	0.62
Antibiotic	51 (40)	69 (53)	20 (16)	**< 0.001**
Fluid Bolus	2 (2)	30 (23)	28 (22)	**< 0.001**
Vasopressor	2 (2)	12 (9)	11 (9)	**0.009**
pRBC transfusion	2 (2)	22 (17)	22 (17)	**< 0.001**
Supplemental oxygen	83 (64)	98 (76)	23 (18)	**0.012**

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Declarations

Competing Interests The authors declare no competing interests.

Conflict of Interest The authors have no financial disclosures or conflicts of interest to report.

IRB Approval The study was approved by the Duke University Medical Center Institutional Review Board (Pro00102839).

References

1. Fixler DE et al (2010) Mortality in first 5 years in infants with functional single ventricle born in Texas, 1996 to 2003. Circulation 121(5):644–650. https://doi.org/10.1161/CIRCULATIONAHA.109.881904
2. Ahmed H et al (2020) Development of a validated risk score for interstage death or transplant after stage I palliation for single-ventricle heart disease. J Thorac Cardiovasc Surg 160(4):1021–1030. https://doi.org/10.1016/j.jtcvs.2019.11.001
3. Mascio CE et al (2019) Thirty years and 1663 consecutive Norwood procedures: has survival plateaued? J Thorac Cardiovasc Surg 158(1):220–229. https://doi.org/10.1016/j.jtcvs.2018.12.117
4. Penk JS et al (2015) Unplanned admissions to a pediatric cardiac critical care unit: a review of 2 years' experience. Pediatr Crit Care Med 16(2):155–160. https://doi.org/10.1097/PCC.0000000000000316
5. Bavare AC et al (2017) Acute Decompensation in Pediatric Cardiac Patients: outcomes after Rapid Response events. Pediatr Crit Care Med 18(5):414–419. https://doi.org/10.1097/PCC.0000000000001117
6. Miles AH, Spaeder MC, Stockwell DC (2016) Unplanned ICU transfers from Inpatient units: examining the prevalence and preventability of adverse events Associated with ICU transfer in Pediatrics. J Pediatr Intensive Care 5(1):21–27. https://doi.org/10.1055/s-0035-1568150
7. Endacott R et al (2007) Recognition and communication of patient deterioration in a regional hospital: a multi-methods study. Aust Crit Care 20(3):100–105. https://doi.org/10.1016/j.aucc.2007.05.002
8. Lambert V et al (2017) Paediatric early warning systems for detecting and responding to clinical deterioration in children: a systematic review. BMJ Open 7(3):e. https://doi.org/10.1136/bmjopen-2016-014497
9. Rusin CG et al (2016) Prediction of imminent, severe deterioration of children with parallel circulations using real-time processing of physiologic data. J Thorac Cardiovasc Surg 152(1):171–177. https://doi.org/10.1016/j.jtcvs.2016.03.083
10. McLellan MC, Gauvreau K, Connor JA (2014) Validation of the Cardiac Children's hospital early warning score: an early warning Scoring Tool to prevent cardiopulmonary arrests in children with Heart Disease. Congenit Heart Dis 9:194–202. https://doi.org/10.1111/chd.12132
11. King G et al (2022) Natural and modified history of atrioventricular valve regurgitation in patients with Fontan circulation. J Am Coll Cardiol 79(18):1832–1845. https://doi.org/10.1016/j.jacc.2022.02.022
12. Haga T et al (2022) Characteristics of In-Hospital patients with congenital heart Disease requiring Rapid Response System Activations: a japanese database study. Congenit Heart Dis 17(1):31–43. https://doi.org/10.32604/chd.2022.017407
13. Olive MK, Owens GE (2018) Current monitoring and innovative predictive modeling to improve care in the pediatric cardiac intensive care unit. Transl Pediatr 7(2):120–128. https://doi.org/10.21037/tp.2018.04.03
14. Rusin CG et al (2021) Automated prediction of cardiorespiratory deterioration in patients with single ventricle. J Am Coll Cardiol 77(25):3184–3192. https://doi.org/10.1016/j.jacc.2021.04.072
15. Garcia-Canadilla P et al (2022) Machine learning-based Systems for the anticipation of adverse events after Pediatric Cardiac surgery. Front Pediatr 10. https://doi.org/10.3389/fped.2022.930913
16. Ruiz VM et al (2022) Early prediction of clinical deterioration using data-driven machine-learning modeling of electronic health records. J Thorac Cardiovasc Surg 164(1):211–222. https://doi.org/10.1016/j.jtcvs.2021.10.060
17. Ruiz VM et al (2019) Early prediction of critical events for infants with single-ventricle physiology in critical care using routinely collected data. J Thorac Cardiovasc Surg 158(1):234–243. https://doi.org/10.1016/j.jtcvs.2019.01.130
18. Kim SY et al (2019) A deep learning model for real-time mortality prediction in critically ill children. Crit Care 23(1):279. https://doi.org/10.1186/s13054-019-2561-z
19. Alten J et al (2022) Preventing Cardiac arrest in the Pediatric Cardiac Intensive Care Unit through Multicenter collaboration. JAMA Pediatr 176(10):1027–1036. https://doi.org/10.1001/jamapediatrics.2022.2238
20. DiBardino DJ et al (2012) Effect of sex and race on outcome in patients undergoing congenital heart surgery: an analysis of the society of thoracic surgeons congenital heart surgery database. Ann Thorac Surg 94(6) 2054-9; discussion 2059. https://doi.org/10.1016/j.athoracsur.2012.05.124

21. Benavidez OJ et al (2007) Complications and risk factors for mortality during congenital heart surgery admissions. Ann Thorac Surg 84(1):147–155. https://doi.org/10.1016/j.athoracsur.2007.02.048
22. Williamson CG et al (2023) Insurance-based disparities in congenital Cardiac Operations in the era of the Affordable Care Act. Pediatr Cardiol. https://doi.org/10.1007/s00246-023-03136-7
23. Santana S et al (2022) Adverse maternal fetal environment partially mediates disparate outcomes in non-white neonates with major congenital heart disease. J Pediatr. https://doi.org/10.1016/j.jpeds.2022.06.036
24. Gardner MM et al (2019) Association of a Home Monitoring Program with Interstage and Stage 2 outcomes. J Am Heart Assoc 8(10). https://doi.org/10.1161/JAHA.118.010783
25. Tabbutt S et al (2012) Risk factors for hospital morbidity and mortality after the Norwood procedure: a report from the Pediatric Heart Network single ventricle Reconstruction trial. J Thorac Cardiovasc Surg 144(4):882–895. https://doi.org/10.1016/j.jtcvs.2012.05.019
26. Schwartz SM et al (2014) Risk factors for prolonged length of stay after the stage 2 procedure in the single-ventricle reconstruction trial. J Thorac Cardiovasc Surg 147(6):1791–1798. https://doi.org/10.1016/j.jtcvs.2013.07.063
27. Chapman SM et al (2016) Systematic review of paediatric track and trigger systems for hospitalised children. Resuscitation 109:87–109. https://doi.org/10.1016/j.resuscitation.2016.07.230
28. Trubey R et al (2019) Validity and effectiveness of paediatric early warning systems and track and trigger tools for identifying and reducing clinical deterioration in hospitalised children: a systematic review. BMJ Open 9(5):e. https://doi.org/10.1136/bmjopen-2018-022105