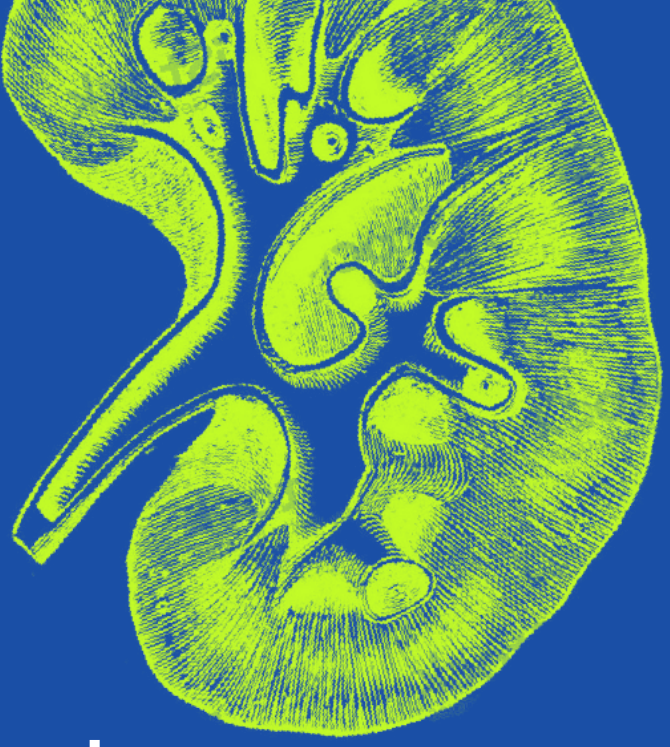


The Role Of Urine Output in Acute Kidney Injury and Hemodynamic Monitoring

A Literature Review

FIZEMEDICAL

#01

Manual VS. Digital, Automatic Urine Output Measurement

Digital, automated urine output measurement was found to be significantly more accurate, effective, and efficient than manual measurement. This emphasizes the potential of digital measurement in earlier detection of oliguric events and AKI, faster response time to patient deteriorating status, and nursing staff workload alleviation.

AUTOMATED VERSUS MANUAL URINE OUTPUT MONITORING IN THE INTENSIVE CARE UNIT

Minor et al. 2021

BACKGROUND

This prospective observational study compared the use of an electronic urine monitoring system to manual recording of a patient's hourly Urine output (UOP) in a cardiothoracic surgery ICU.

METHODS

Floor nurses recorded 187 hourly UO measurements from 44 patients as per standard of care, while a catheter sensor took measurements as well. Both UO measurement methods were monitored by weighing the urine collection bag via a digital scale and this was used as the gold standard for comparisons.

RESULTS

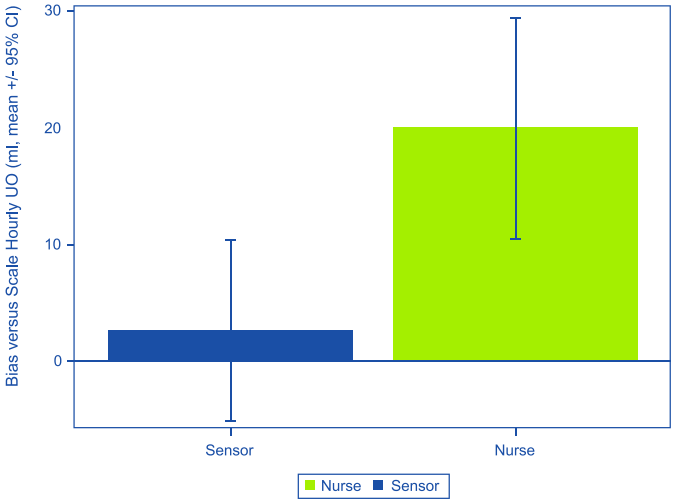
Automated measurement was found to be more efficient and accurate compared to manual measurement.
The mean measurement bias between the sensor and the scale was significantly lower than the mean bias between the nurse and the scale by 17.3 ml (95% CI: 7.0; 27.7; p-value: < 0.01) on average.

Automated measurements provided more timely data than the ones taken manually.
Manual recordings were frequently (39%) missed, often late, and were off by more than 17 ml/hr.

Automated measurement detected oliguric events more accurately.
The patients in this study produced 76.3 ml/hr of UO on average. The mean measurement bias between the sensor and the scale was significantly lower than the mean bias between the nurse's measurement and the scale by 17.3 ml (95% CI: 7.0; 27.7; p-value: < 0.01). A bias of 17 ml approaches 50% of the threshold value for oliguria diagnosis and is 22.3% of the total UO (76.3 ml/hr in this study). The sensor was able to detect several oliguric events lasting over one hour which were missed by the nurses, including one patient who met UO criteria for AKI.

CONCLUSIONS

The accuracy provided by manual monitoring of UO may be insufficient to detect AKI in some patients. Even in the absence of AKI, UO monitoring is essential for critically ill patients as oliguria is an early predictor of mortality in these patients.



URINE OUTPUT MEASUREMENT IS MUCH MORE ACCURATE WHEN MEASURED BY A NOVEL ELECTRONIC URINOMETER THAN BY A CONVENTIONAL URINOMETER

Rott et al. 2020

BACKGROUND

Oliguria is defined as urinary output (UOP) <0.5ml/kg/hr and may lead to Acute Kidney Injury (AKI) and other poor outcomes. Currently, UOP measurement is done by visually inspecting the urine in a collection bag once every hour, while all other vital signs are automatically and continuously monitored.

OBJECTIVES

Assessing the FIZE kUO Electronic Urinometers' (FIZE kUO) accuracy as well as describing the importance of obtaining real-time measurement of UOP. The accuracy of the conventional Manual Urinometer (MU) was compared to the FIZE kUO.

METHODS

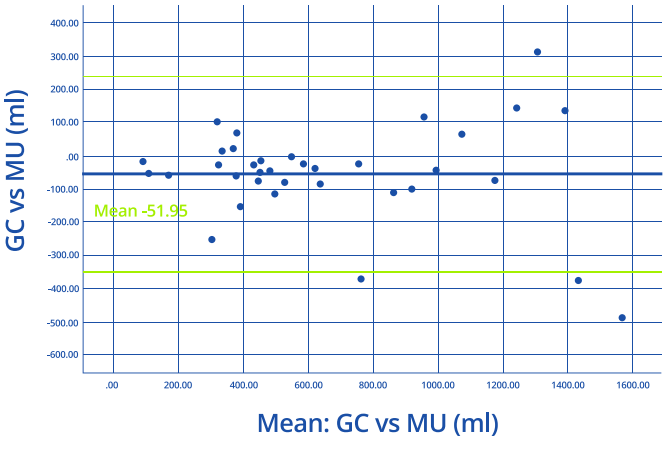
The FIZE kUO was attached to patients' foley catheter. The MU was connected to the tube exiting the FIZE kUO, leading to the urine collection bag. This allowed UOP to be measured by both methods in tandem. Finally, the collection bag was attached to a Graduated Cylinder (GC), which served as the gold standard.

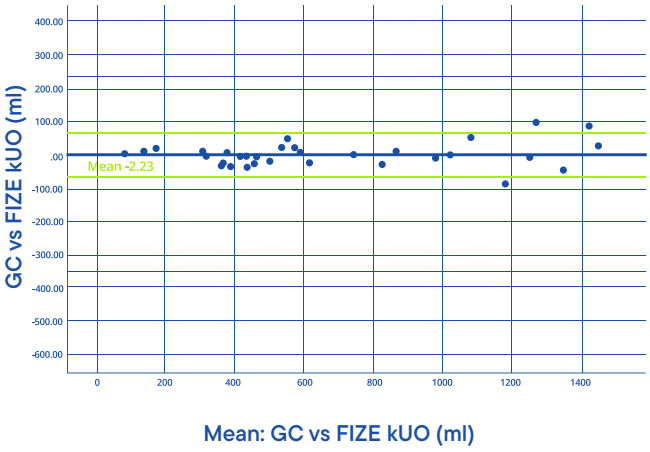
RESULTS

The FIZE kUO measurement was much more accurate than the MU measurement, when compared against the GC measurement. The average deviation from GC measurement was 4 ±3.0% for the FIZE kUO and 17 ± 23% for the MU (P<0.01).

CONCLUSION

This study demonstrated that the FIZE kUO provides a significantly more accurate assessment of urine output than the conventional MU when both are compared to gold standard measurements obtained by the GC. Utilizing the FIZE kUO has the potential to improve accuracy of this vital measurement, decrease manual labor, and provide automated digital data inputs into modern electronic medical record (EMR) systems. The FIZE kUO was found to have a significant positive potential impact on clinical decision making and patient outcomes by enabling early recognition of AKI and hemodynamic instability, thus decreasing overall mortality.





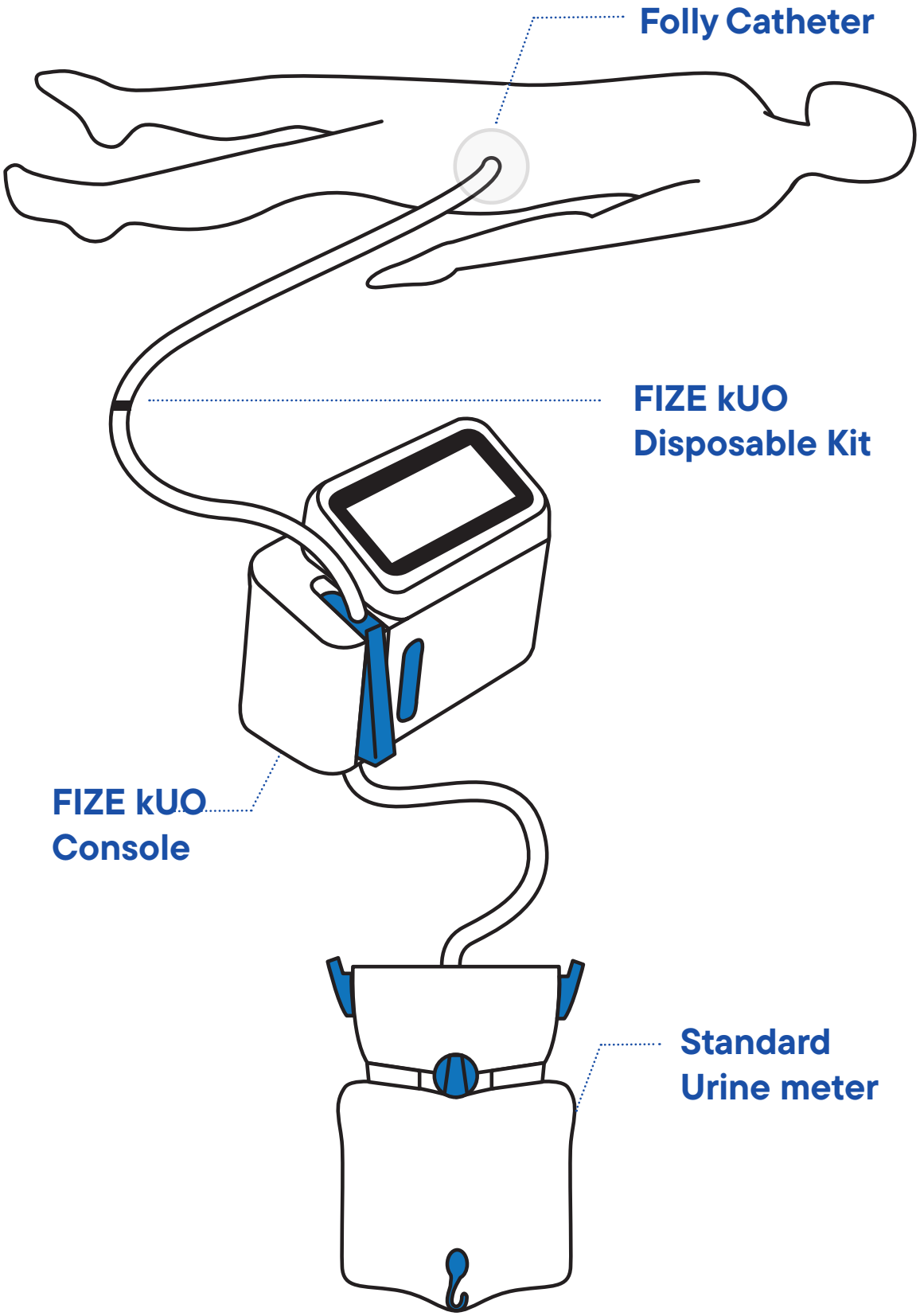


Figure 1: Experiment Setup (FIZE Medical Ltd).

IMPROVING FLUID OUTPUT MONITORING IN THE INTENSIVE CARE UNIT

Kushnir et al 2022

BACKGROUND

Changes in UOP are used to prescribe and assess the effectiveness of diuretic medications as well as identifying early signs of acute kidney injury (AKI) is based on changes in urine output (UOP). Given the numerous demands on nurses, this important task is easy to overlook and can result in incomplete, inaccurate, or late measurements, which in turn may result in compromised patient care.

OBJECTIVES

This study aimed to assess the frequency and severity of incomplete, inaccurate, or late UOP measurements, as well as to identify and evaluate the attitudes of nursing staff and physicians regarding procedures for UOP measurements.

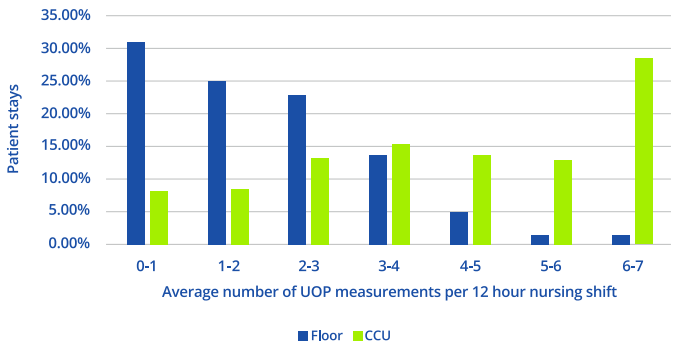
METHODS

Documentation timeliness (time between expected and observed UOP recording) was assessed over a 3-month period by analyzing UOP documentation in 254 patients' medical records in a cardiac intensive care unit (CICU), and a cardiothoracic-intensive care unit (CTICU). Lateness of UOP documentation was assessed by the number of minutes between the time UOP was recorded, and the time it was documented in the electronic medical record (EMR). In addition, a survey of 25 nurses, 7 nurse practitioners, and 13 cardiologists was conducted to assess their opinions regarding UOP.

RESULTS

Manual UOP measurements and reporting were often inaccurate and late.
Less than 30% of patients had UOP recorded every hour and on average these measurements were recorded over an hour late. 60% of admissions had UOP recorded in the EMR less frequqntly than every 2 hours.
90% were recorded over 20 minutes late ~10% of patients in the CICU had only ≤2 measurements recorded per nursing shift.

Survey findings showed positive staff attitudes towards automation of UOP monitoring.
An overwhelming majority of nurses surveyed (24/25) believed that automated UOP monitoring should be developed and would improve workflow. 95% (19/20) believed that accurate and timely UOP reporting would improve diuretic prescribing practices. More than half of physicians (13/19) believed that automated, hourly UOP recordings could lead to a decrease in CICU/CTICU length of stay.



CONCLUSION

Manual measurement and documenting UOP poses a significant burden on nursing staff which impacts clinicians' orders that rely on up to date UOP information. Delays in recording UOP measurement effect the timeline for assessment of medication effectiveness, which can be detrimental for critically ill patients for whom volume status and diuretic dose must be consistently reevaluated (i.e. intravenous furosemide exhibits its maximal effect on UOP within 1 hour). Automated UOP monitoring and documentation would improve timeliness of UOP recordings in the EMR and remove this documentation burden from nurses, thus reducing their workload.

#02

Importance of Urine Output Monitoring in Acute Kidney Injury and Fluid Overload

Acute Kidney Injury (AKI) stands out as one of the more prevalent and serious morbidities among hospitalized patients (Hobson et al., 2015). AKI can result in several severe outcomes, with an estimated mortality rate of ~20% for hospitalized patients (Selby et al., 2012; Susantitaphong et al., 2013). Therefore, monitoring for the development of AKI is of utmost importance.

Urine output has been identified as the most cost-effective and impactful biomarker for detecting and managing AKI (Goldstein et al., 2020). Diligent monitoring of urine output has demonstrated reductions in morbidity and mortality, as well as shortened length of stay and decreased overall cost of care.

In pediatric patients, for nearly one in five patients exhibiting stage 2-3 AKI the only indication was changes in urine output, which could have led to misclassification as not having severe

AKI. Patients with AKI who presented higher serum creatinine levels along with oliguria were found to have a fivefold increase in the likelihood of death within 28 days (Kaddourah, 2019). In adults, intensive urine output monitoring has shown improvements in AKI detection, resulting in reduced 30-day mortality rates and more than 50% reduction in fluid overload (Jin, 2017).

INTENSIVE MONITORING OF URINE OUTPUT IS ASSOCIATED WITH INCREASED DETECTION OF ACUTE KIDNEY INJURY AND IMPROVED OUTCOMES

Jin et al 2017

BACKGROUND

The National Confidential Enquiry into Patient Outcomes and Death from the United Kingdom published a report showing that in 30% of patients who died of Acute Kidney injury (AKI), the condition was predictable and avoidable. Careful monitoring of urine output (UOP) could lead to earlier recognition of AKI, yet standards for UOP monitoring vary widely between ICUs.

OBJECTIVES

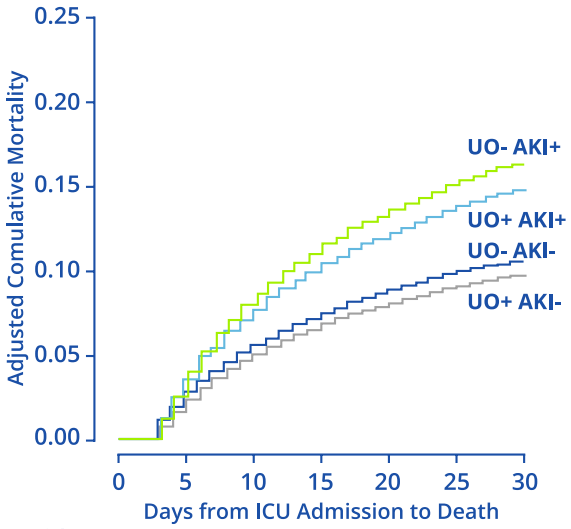
This study aimed to compare the risk of AKI and outcomes associated with AKI in patients who had and those who did not have daily monitoring of serum creatinine (SC) and hourly monitoring of UOP.

METHODS

15,724 adults admitted to ICUs from 2000 to 2008 were included in this retrospective cohort study. Intensive UOP monitoring was defined as hourly recordings and no gaps >3 hours for the first 48 hours after ICU admission. For all analyses, moderate to severe AKI was defined as stage 2-3. For UOP criteria, were collected at least every 6 hours to stage AKI regardless of whether the patient had intensive or non intensive UOP monitoring.

RESULTS

Intensive UOP monitoring improved AKI detection.
Intensive monitoring for UOP was conducted in 4,049 patients (26%), in whom significantly higher rates of AKI (OR, 1.22; P < .001) were present. In contrast, intensive monitoring of SC was only marginally significant (OR, 1.11; 95% CI, 1.00-1.24; P ¼ .05)



No. at risk							
UO+ AKI-	1,518	1,493	1,447	1,424	1,411	1,392	1,380
UO+ AKI+	2,528	2,474	2,315	2,222	2,155	2,089	2,035
UO- AKI-	4,212	4,106	3,993	3,912	3,877	3,835	3,801
UO- AKI+	7,461	7,160	6,600	6,287	6,061	5,908	5,792

"UO+/- indicates whether or not UOP was monitored.
AKI+/- indicates whether AKI stage 2-3 was present"

Intensive UOP monitoring improved survival. For patients experiencing AKI, in unadjusted analyses intensive monitoring for UOP was strongly associated with improved survival to 30 days compared with patients with less intensive UOP monitoring (HR, 0.85; 95% CI, 0.77-0.94; P = .001), and this held after adjusting for age and severity of illness (HR, 0.90; 95% CI, 0.81-0.99; P = .04).

Intensive UOP monitoring improved fluid volume.
With or without AKI, patients with intensive UOP monitoring also received less fluids, leading to less cumulative fluid volume (2.98 L vs 3.78 L; P < .001) and less fluid overload (2.49% vs 5.68%; P < .001) over the first 72 hours of ICU stay.

CONCLUSION

Intensive monitoring of UOP was associated with improved detection of AKI and reduced 30-day mortality in patients experiencing AKI, as well as less fluid overload for all patients. Intensive monitoring of UOP is critical, especially for patients at high risk of AKI or fluid overload, or both.

OLIGURIA AND ACUTE KIDNEY INJURY IN CRITICALLY ILL CHILDREN: IMPLICATIONS FOR DIAGNOSIS AND OUTCOMES

Ahmad Kaddourah 2019

BACKGROUND

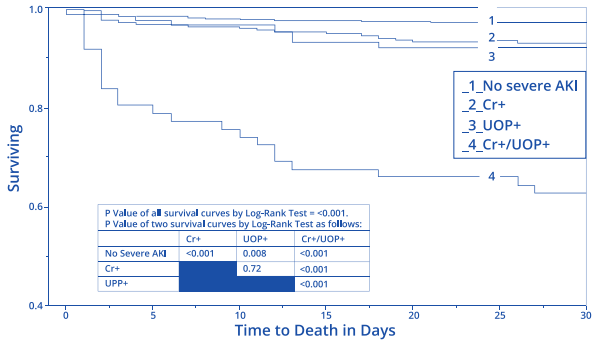
Acute Kidney Injury (AKI) is common in hospitalized children and is associated with poor prognostic outcomes. Although oliguria is often characteristic of AKI, the role of urine output (UOP) criteria in AKI diagnosis has not been studied extensively.

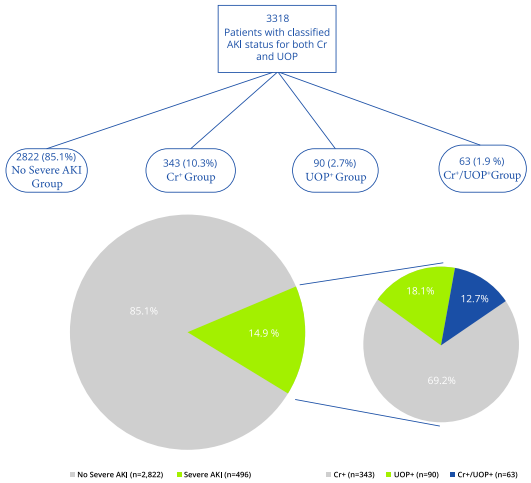
OBJECTIVES

Evaluating the criteria by which AKI is defined, assessing assess the significance of changes in serum creatinine (SC) and UOP, and determining their impact on the diagnosis and outcome of severe AKI. This study also describes the epidemiology of oliguric AKI and the contribution of UOP criteria to severe AKI ascertainment and outcomes.

METHODS

Critically ill children enrolled in the Assessment of Worldwide Acute Kidney Injury, Renal Angina and, Epidemiology (AWARE) database were divided into four cohorts: non-severe AKI (n=2822 [85.1%]), severe AKI by SC criteria only (n=343 [10.3%]), severe AKI by UOP criteria only (n=90 [2.7%]), and severe AKI by both SC and UOP criteria (n=63 [1.9%]). These groups were assessed for correlation with the following: 28-day mortality, PICU length of stay (LOS), and utilization of renal replacement therapy, mechanical ventilation and extracorporeal membrane oxygenation (ECMO).





RESULTS

SC based diagnoses missed many cases of AKI with changes only in UOP.
Nearly one in five critically ill children with Acute Kidney Injury do not experience an increase in SC. These cases of AKI, identified only by UOP criteria, are associated with comparably poor outcomes to those diagnosed by changes in SC.

Children meeting both criteria had worse outcomes than those meeting only one.
Severe AKI occurred in 496 of 3,318 children (14.9%); 343 (69.2%) filled SC criteria only, 90 (18.1%) filled UOP criteria only, and 63 (12.7%) filled both criteria. While 28-day mortality did not differ between those with stage 2/3 AKI by SC alone vs. UOP alone, patients who met Stage 2 or 3 by both criteria experienced a 5-fold increased 28-day mortality rate (38.1% in comparison to SC criteria alone (6.7%) or UOP criteria alone (7.8%)). Compared with patients without severe AKI, the relative risk of receiving dialysis increased from 9.1 (95% CI, 3.9–21.2) using SC criteria only, to 28.2 (95% CI, 11.8–67.7) using UOP criteria only, to 165.7 (95% CI, 86.3–318.2) using both criteria.

CONCLUSION

Oliguria represents a risk factor for poorer outcomes in children who develop AKI. Utilizing both the SC and UOP criteria leads to a more comprehensive assessment and identifies a subset of children with AKI who are at higher risk for morbidity and mortality. Current guidelines for identifying AKI are primarily SC based and do not always take UOP into account although AKI defined by oliguria often portends a higher risk of morbidity and mortality. Thus AKI diagnosis is missed in a substantial proportion of patients where UOP is not assessed. Utilizing UOP to assess and recognize oliguria, in addition to SC based AKI diagnostic measures, is crucial for management of critically ill patients with or at risk off AKI.

#03

Urine Output's Vital Role in Shock Prediction

Cardiovascular shock is a potentially life-threatening condition requiring immediate and multifaceted intervention. While there are several types of shock, depending on underlying causes, all shock states involve changes to intravascular volume leading to severe and widespread tissue damage due to hypoperfusion. Shock is associated with poor outcomes, and carries a high mortality rate ranging between 20-50% (JAMA 2022). Prompt and thorough response to shock is paramount for positive patient outcomes and survival.

While several vital signs are used in practice to identify, assess, and track shock progression, urine output (UOP) has emerged as a critical forteller of changes to intravascular volume, specifically during shock (Brotfain 2019, Kleinpell 2020, and Kowalski 2023). In all types of shock, from the very beginning of its development, the kidneys experience changes to renal blood flow. These changes result in consistent, measurable, and accurate decreases in UOP, which can often be the only indicator of the underlying crisis; showing up long before changes are witnessed in the cardiovascular system. This sentinel signal is vital for rapid identification, allowing optimal tissue preservation and recovery in the development of shock states.

THE VITAL ROLE OF URINE OUTPUT IN SHOCK PREDICTION

Shock is a life-threatening condition, in which profound circulatory disturbances cause widespread tissue hypoxia due to inadequate tissue perfusion failing to meet the metabolic needs and oxygen consumption requirements of cells. As shock progresses, it becomes increasingly more complex and challenging to treat, and may eventually result in irreversible damage, multiorgan failure (MOF), and death. It is of utmost importance for the clinician to work to simultaneously identify and treat the underlying cause of shock, as well as manage its effects and prevent further damage or death (Gaieski DF, Mikkelsen ME. Definition, classification, etiology, and pathophysiology of shock in adults. In: UpToDate, Gong M (Ed), Wolters Kluwer. Accessed on Dec 24, 2023).

Generally, there are four categories of shock: distributive, hypovolemic, cardiogenic, and obstructive. Distributive shock is the most common type, and pertains to a state of relative hypovolemia resulting from pathological redistribution of the absolute intravascular volume (Standl 2018). Septic shock, a form of distributive shock, is the most common form of shock among patients admitted to the intensive care unit, followed by hypovolemic and cardiogenic shock (Gaieski DF, Mikkelsen ME. Definition, classification, etiology, and pathophysiology of shock in adults. In: UpToDate, Gong M (Ed), Wolters Kluwer. Accessed on Dec 24, 2023). Septic shock carries a mortality rate between 40 to 50% (Haseer Koya, 2023).

However, the prevalence of different types of shock depends on the clinical setting and the population it serves. While septic shock is most prevalent in the ICU, level-1 trauma centers will likely see a higher percentage of hemorrhagic shock, a type of hypovolemic shock (Gaieski DF, Mikkelsen ME. Definition, classification, etiology, and pathophysiology of shock in adults. In: UpToDate, Gong M (Ed), Wolters Kluwer. Accessed on Dec 24, 2023).

STAGES AND PROGRESSION OF SHOCK

All four types of shock progress similarly through four stages as the patient's condition worsens.

STAGE I The first stage, known as initial or pre-shock, is characterized by initiation of compensatory mechanisms to counter the decrease in tissue perfusion, as cells switch to anaerobic respiration, causing a rise in lactic acid (Kleinpell 2020). In this stage cardiac output (CO) remains stable, because although stroke volume (SV) decreases, heart rate (HR) increases to compensate. At this point, other than potentially a slight decrease in blood pressure (BP) and tachycardia, the majority of clinical manifestations are vague physical signs: altered level of consciousness, and in most types of shock, peripheral vasoconstriction, manifesting as cool clammy skin (Kleinpell 2020). These changes are non-specific and may be too subtle to detect, as the compensatory mechanisms mask the underlying deterioration (Ahrens 2015 and Kleinpell 2020). However, an additional and critical sign emerging already in the initial stage of shock is a change in urinary output (UOP) (Brotfain 2019, Kleinpell 2020, and Kowalski 2023).

While all other vital signs and biomarkers currently being utilized clinically are non-specific, not entirely reliable, difficult to measure, or simply appear after injury was already incurred, UOP is notable in that it is a direct meter for fluid and hemodynamic status, reliable and easy to obtain, and serves as a sentinel, acting as a preliminary warning sign for impending deterioration.

STAGE II In the second phase of shock, known as the compensatory stage, hemostatic mechanisms mediated by the sympathetic nervous system (SNS) are triggered in an attempt to maintain arterial BP despite decreasing CO, and improve tissue perfusion (Pasman 2015). Capillary hydrostatic pressure decreases, shifting fluid to the intravascular space; neurohormonal cascades are initiated, catecholamines are released, increasing cardiac contractility; the reninangiotensin-aldosterone system (RAAS) is activated, resulting in water retention and shunting blood to vital organs; antidiuretic hormone causes additional sodium and water retention, further decreasing UOP (Kleinpell 2020); and finally, intrarenal blood flow is disturbed, as a result significantly lowering UOP (Lankadeva 2016). However, despite a fulminant renal response, other vital signs and biomarkers are still insufficient for recognizing the underlying crisis at this point, as multifaceted compensation masks the fall in CO. Since UOP begins to exhibit clear changes in response to the very initiation of these compensatory mechanisms, as mentioned above, focusing on precise monitoring of UOP is paramount in early and accurate detection and progression of shock.

STAGE III Once compensatory mechanisms can no longer be maintained, a more severe clinical picture begins to emerge. This phase is known as decompensation, or the progressive stage. During this stage, most of the classic signs and symptoms of shock appear, due to early organ dysfunction. CO decreases as HR is no longer able to compensate for decreased SV (Haseer Koya 2023). Compensatory attempts now fail to sustain adequate perfusion to the tissues, and impaired oxygenation causes hypoxic injury to the cells. Metabolic acidosis and severe electrolyte imbalances may ensue, worsening cellular damage and patient outcomes.

STAGE IV If earlier stages were not identified, or if interventions were insufficient, decompensation will be followed by the final stage, known as refractory shock. In this stage, end-organ damage and cellular necrosis cause irreversible organ dysfunction, MOF, and death (Haseer Koya 2023 and kleinpell 2020).

SEPTIC SHOCK

Septic shock is a subset of sepsis in which particularly profound circulatory, cellular, and metabolic abnormalities lead to significantly worse outcomes than with sepsis alone (Singer 2016). Unlike in other shock states, a dysregulated host response to infection causes vasodilation which increases capillary membrane permeability thus allowing for fluid shifts, decreasing intravascular blood volume, in addition to forming micro emboli further impacting perfusion (Singer 2016 and Kleinpell 2020).
In septic shock both renal hypoperfusion and hyperperfusion may occur despite general vasodilation. However, regardless of blood supply to the kidneys, an intrarenal decrease in blood flow is observed in sepsis.

The relationship between global and intrarenal blood flow, and the effects on UOP

Absolute (hypovolemia) and relative (hemodynamic perturbation) reductions in effective blood volume may lead to a reduced renal blood flow (RBF). However, this reduction alone does not usually affect glomerular filtration rate (GFR). In fact, in sepsis-associated AKI, RBF may be preserved or even increased (Fani 2018). What seems to have a greater influence than global RBF is intrarenal blood flow. In practice we observe that blood flow within the kidneys themselves is not directly correlated to systemic blood flow. A decrease in intrarenal blood flow leads to decreased UOP regardless of RBF. These changes to UOP and UFRV are clinically indispensable, and are observed already in the initial stage of shock, before any other vital sign shows reliable and measurable changes (Brotfain 2019 and 2020, Kleinpell 2020, and Kowalski 2023).

HYPOVOLEMIC SHOCK

Hypovolemic shock is considered the second most common form of shock (Standl 2018 and Haseer Koya, 2023). Commonly hypovolemic shock is due to rapid blood loss; other causes include profound dehydration, massive GI losses, and capillary fluid leak into interstitial spaces, as seen in pancreatitis, bowel obstruction, and ascites. In hypovolemic shock, CO falls due to decreasing venous return to the heart - preload (Kleinpell 2020). The amount of volume loss, rate of loss, and underlying cause affect the presentation and severity of shock (Kowalski 2023).

Initial stage Up to 15% (750 ml) of volume is lost, changes to CO are adequately compensated by vasoconstriction and increased HR. At this point the patient is largely asymptomatic, although changes to UOP can already be observed.

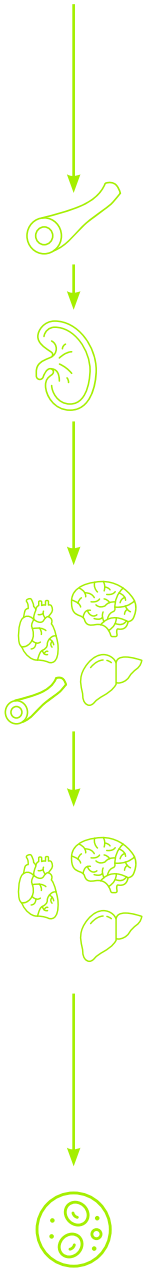
Compensatory stage
15-30% (750-1500 ml) of volume is lost. CO decreases and leads to hypoxemia. HR and RR increase, and a noticeable reduction in urine output is observed.

Progressive stage 30% to 40% (1500-2000 ml) of volume is loss. Compensatory mechanisms fail, arrhythmias can develop, causing myocardial ischemia. Respiratory distress can occur.

Refractory stage Over 40% (more than 2000 ml) of volume is lost. Organ failure and cardiac arrest may ensue, becoming immediately life threatening. While in hemorrhagic shock UOP may not begin to decrease until the compensatory stage, it remains the single most important indicator for monitoring fluid status in a patient. Additionally, BP is not an adequately reliable to detect the beginning of shock, as the body's compensatory mechanisms keep BP typically within normal limits until up to 30% of blood volume has already been lost (Kowalski 2023).

STAGES OF SHOCK PROGRESSION AND THEIR ASSOCIATED CLINICAL SIGNS

INJURY



INITIAL STAGE

Impaired tissue perfusion

Occult vasoconstriction
or sepsis-induced vasodilation

Intrarenal shunting

UOP ⇩ UFR ⇩ UFRV ⇩
Kidneys initiate compensatory mechanisms to maintain CO

Clinical signs:
Changes in UFR & UFRV

COMPENSATORY STAGE

Systemic compensation

Increased cardiac contractility, water and sodium retention, vasoconstriction*

*not present in sepsis

Clinical signs:
Clinically significant oliguria, slight non specific & unreliable changes toin BP, HR, MAP.

PROGRESSIVE OR DECOMPENSATORY STAGE STAGE

Compensation mechanisms fail

CO ⇩, early organ dysfunction

Clinical signs:
Severe oliguria or anuria, life threatening changes to BP, HR, MAP. Metabolic acidosis & electrolyte imbalances appear.

FINAL OR REFRACTORY STAGE

Hypoxic cell death

End organ damage, death

Clinical signs:
Severe oliguria or anuria, life threatening changes to BP, HR, MAP. Uncompensated metabolic acidosis & severe electrolyte imbalances appear.

THE PROBLEM WITH COMMON HEMODYNAMIC MARKERS IN EARLY SHOCK DETECTION

Vital signs and biomarkers used to monitor and assess for shock in intensive care settings, include blood pressure (BP), stroke volume (SV), mean arterial pressure (MAP), heart rate (HR), arterial blood lactate concentration (Lact), and shock index (Rady, 1994). Although urine output (UOP) is one of the most critical vital signs used for hemodynamic instability (Rivers 2001 and Rhodes 2016), in practice it is often overlooked. Recent data shows that UOP may serve as one of the earliest prognosticators for emerging hemodynamic changes (Macedo 2011 and Brotfain 2017). Yet, despite evidence hailing UOP as an early predictor for hypoperfusion, accurate and continuous measurement methods for UOP are not widespread in the clinical setting.

Blood Pressure (BP)

Although BP is widely used as the primary measure for shock progression, in order to truly understand the circulatory status and cardiac function of a patient, the truest measure of the heart's effectiveness is obtained only by SV measurement. Although CO and BP are essential and fundamental signs of cardiovascular (CV) function, BP cannot serve as an approximation for SV; as measurements for flow and pressure are not interchangeable (Phillips 2017).

Heart Rate (HR)

Heart rate is considered to be one of the most obvious signs of hemodynamic imbalances and disturbances to homeostasis. However, it is as nonspecific as it is overt. Aside from the release of epinephrine and norepinephrine in order to increase HR, and compensate for CO decrease, the presence of pain and agitation could themselves cause tachycardia (Kleinpell 2020).

Mean Arterial Pressure (MAP)

MAP has traditionally been the primary measure for shock evaluation, however as previously discussed, compensatory mechanisms delay clinically significant CV findings until later stages of shock. In addition, currently most MAP devices utilized in ICU are invasive, posing an additional risk for iatrogenic infection. Despite non-invasive MAP devices becoming increasingly available, UFR and UFRV have been shown to be more responsive to dynamic changes in fluid status, and appear earlier than changes relying on CV function. This is true both in predicting impending BP drops while compensatory mechanisms hold steady, and in fluid resuscitation assessment, as UFR and UFRV increase prior to clinically significant changes in MAP (Brotfain 2020).

Stroke Volume (SV)

In theory, the most accurate measure for fluid status and management is SV. However, even proponents of utilizing SV as the most accurate indicator of CO agree that most current methods have significant flaws (Ahrens 2015 and Phillips 2017). Although SV is the most accurate measure of cardiac function and fluid status, obtaining SV measurements is severely limited by cost, the need for exact and frequent calibrations requiring specially trained practitioners, and is most accurate when invasive, putting the patient at further risk (Johnson 2015 and Phillips 2017). Finally, dynamic, non-invasive SV measurements, seen as most accurate, require patients to be on fully controlled mechanical ventilation without spontaneous efforts, which is seldom the case in ICU patients (Cecconi 2014).

Lactate and pH

Shock is associated with acidosis. Due to inadequate oxygen utilization by the cells, cellular dysoxia occurs, with increased blood lactate levels (Cecconi 2014). Common markers for measuring pH levels include arterial pH and serum bicarbonate. However, these markers may not be reliable in reflecting present lactic acidosis, as compensatory mechanisms such as tachypnea and alkalosis may mask the presence of lactic acidosis. In addition, serum biochemistry is not measured continuously, therefore cannot reliably serve as an early shock indicator.

UOP AS A PRIMARY AND INVALUABLE MEASURE FOR SHOCK

A decrease of urine output below 0.5 ccs/kg/hr suggests renal hypoperfusion and could be the first sign of initial shock or alterations to blood flow in all types of shock (Bajwa 2004 and Kowalski 2023). As the kidneys place a high demand on perfusion, they are especially susceptible to hypoxia and ischemia (Kwiatkowska 2023 and Evans 2019), and are therefore one of the first sites impacted by changes in circulating volume (Kleinpell 2020). This is expressed through UOP and urine flow rate variability (UFRV), responding to hypovolemia earlier than other standard signs (Brotfain 2019 and Shalman 2020).

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