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Mini Review

First 48 Hours of Urine Output in Neonates: Physiological Transition, Clinical Uncertainty, and Evolving Diagnostic Perspectives

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Abstract

Urine output in the first 48 hours of life is widely regarded as a fundamental marker of neonatal renal adaptation; however, its interpretation remains complex due to the interplay between physiological immaturity and pathological processes. While most neonates void within the first 24 hours, early urine patterns are influenced by dynamic changes in renal hemodynamics, tubular function, and hormonal regulation. This review critically examines current evidence regarding the timing of first urination, normative urine output values, and their clinical significance. Particular emphasis is placed on the limitations of urine output as a diagnostic tool, especially in the early detection of neonatal acute kidney injury (AKI). Although reduced urine output is commonly used as an early indicator of renal dysfunction, emerging evidence suggests that reliance on urine output alone may underestimate subclinical kidney injury. A more nuanced interpretation, integrating clinical context and adjunctive biomarkers, is required to improve diagnostic accuracy and neonatal outcomes.

Keywords: Urine Output: Neonates: Physiological Transition:

Introduction

The assessment of urine output during the first 48 hours of life occupies a central role in neonatal clinical practice, yet its interpretation is often oversimplified. Traditionally, early urine passage has been viewed as a reassuring indicator of adequate renal function, whereas delayed urination has been considered a marker of pathology. However, this binary interpretation does not adequately reflect the complex physiological processes governing renal adaptation in the immediate postnatal period. The transition from placental to renal regulation of fluid and electrolyte balance is gradual and highly variable between individuals [1,2].

Available evidence consistently demonstrates that the majority of neonates pass urine within the first 24 hours, with reported rates exceeding 95% [1,3]. Nevertheless, these findings are derived primarily from observational studies with heterogeneous methodologies and inconsistent definitions of "first void," limiting their external validity. Moreover, the widely accepted threshold of 48 hours for defining abnormal delay is largely based on clinical convention rather than robust prospective data [2].

From a physiological standpoint, reduced urine output in the first 24 hours is expected rather than pathological. Neonatal kidneys are characterized by low glomerular filtration rate, elevated renal vascular resistance, and immature tubular function [4]. In addition, increased activity of antidiuretic hormone and the renin–angiotensin–aldosterone system promotes water conservation, resulting in relative oliguria [4,5]. The subsequent increase in urine output observed after 24–48 hours reflects improving renal perfusion and functional maturation.

Despite this physiological context, clinical practice frequently relies on fixed urine output thresholds (table1), typically <1 mL/kg/hour, to define oliguria. While pragmatic, such thresholds fail to account for developmental variability and may lead to misclassification of normal transitional states as pathological.

Importantly, urine output alone represents a relatively late and nonspecific indicator of renal dysfunction in neonates. A substantial proportion of critically ill infants may develop non-oliguric acute kidney injury, in which serum biochemical abnormalities and renal injury occur despite apparently adequate urine production. This limitation highlights the inadequacy of relying exclusively on quantitative urine thresholds for clinical decision-making. In addition, methods used to measure neonatal urine output, including diaper weight estimation and catheter-based monitoring, are themselves subject to variability and potential inaccuracy. The interpretation of early urine passage should therefore incorporate gestational age, birth weight, fluid balance, cardiovascular stability, and exposure to nephrotoxic agents. Premature neonates are particularly vulnerable because incomplete nephrogenesis and immature tubular handling predispose them to fluctuations in sodium and water balance. Emerging evidence also suggests that biomarkers such as cystatin C, kidney injury molecule-1, and neutrophil gelatinase-associated lipocalin may permit earlier recognition of renal injury before overt oliguria develops. Collectively, these observations support a more individualized and physiology-based approach to neonatal urine assessment rather than strict dependence on conventional urine output cutoffs.

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Table 1. Normal Urine Output in Neonates

Age	Expected Urine Output
First 24 hours	0.5 – 1.5 mL/kg/hour
24–48 hours	2 – 5 mL/kg/hour
After 48 hours	2 – 6 mL/kg/hour

Conversely, they may also fail to detect early kidney injury in non-oliguric neonates. This limitation is particularly relevant in neonatal acute kidney injury, where urine output alone lacks both sensitivity and specificity [6].

The role of urine output in the diagnosis of neonatal AKI remains contentious. Modified KDIGO (kidney disease-improving global outcomes) criteria Table 2 incorporate urine output as a key diagnostic parameter; however, their applicability in the early neonatal period is limited by confounding factors. Serum creatinine is influenced by maternal levels during the first days of life, while urine output is affected by fluid management and systemic hemodynamics [4,6].

Table 2. Neonatal AKI Classification (Modified KDIGO)

Stage	Serum Creatinine	Urine Output
Stage 1	↑ ≥0.3 mg/dL or 1.5–1.9× baseline	<1 mL/kg/hour
Stage 2	↑ 2.0–2.9× baseline	<0.5 mL/kg/hour
Stage 3	↑ ≥3× baseline or ≥2.5 mg/Dl	Anuria ≥12 hours

Emerging evidence suggests that novel biomarkers, including neutrophil gelatinase-associated lipocalin (NGAL) and cystatin C, may provide earlier and more accurate detection of renal injury, although their routine use remains limited in clinical practice [7] .

Distinguishing physiological oliguria from pathological states therefore requires careful clinical judgment. Persistent oliguria or anuria beyond 48 hours is uncommon and should prompt urgent evaluation for obstructive uropathy, congenital renal anomalies, or severe renal impairment Table 3. Importantly, urine output must be interpreted in conjunction with clinical context, including perinatal history, cardiovascular status, and laboratory findings [2,3].

Table 3. Differential Diagnosis of Oliguria/Anuria in Neonates

Category	Causes
Pre-renal	Dehydration, hypovolemia, sepsis, shock
Renal	Acute kidney injury, congenital renal dysplasia
Post-renal	Posterior urethral valves, ureteral obstruction
Functional/Physiological	Transitional oliguria, delayed feeding

A significant limitation of the current literature is the lack of high-quality prospective studies defining normative urine output patterns across diverse neonatal populations. Most available data are derived from older or small-scale studies, with limited stratification by gestational age or clinical condition. This gap hinders the development of precise diagnostic thresholds and may contribute to variability in clinical practice [6]. Figure 1, Clinical Approach to Absence of Urine in the First 48 Hours of Life

From a clinical perspective, urine output should not be used as an isolated diagnostic marker but rather as part of a comprehensive assessment strategy. Integration with clinical examination, biochemical parameters, and imaging studies is essential. Additionally, greater emphasis should be placed on trends over time rather than single measurements, allowing for more accurate differentiation between physiological adaptation and evolving pathology.

In conclusion, while urine passage in the first 48 hours remains a valuable clinical indicator, its interpretation requires a nuanced understanding of neonatal physiology and clinical context. Future research should focus on refining diagnostic criteria, validating novel biomarkers, and establishing population-specific reference ranges to enhance early detection of renal dysfunction and improve neonatal outcomes.

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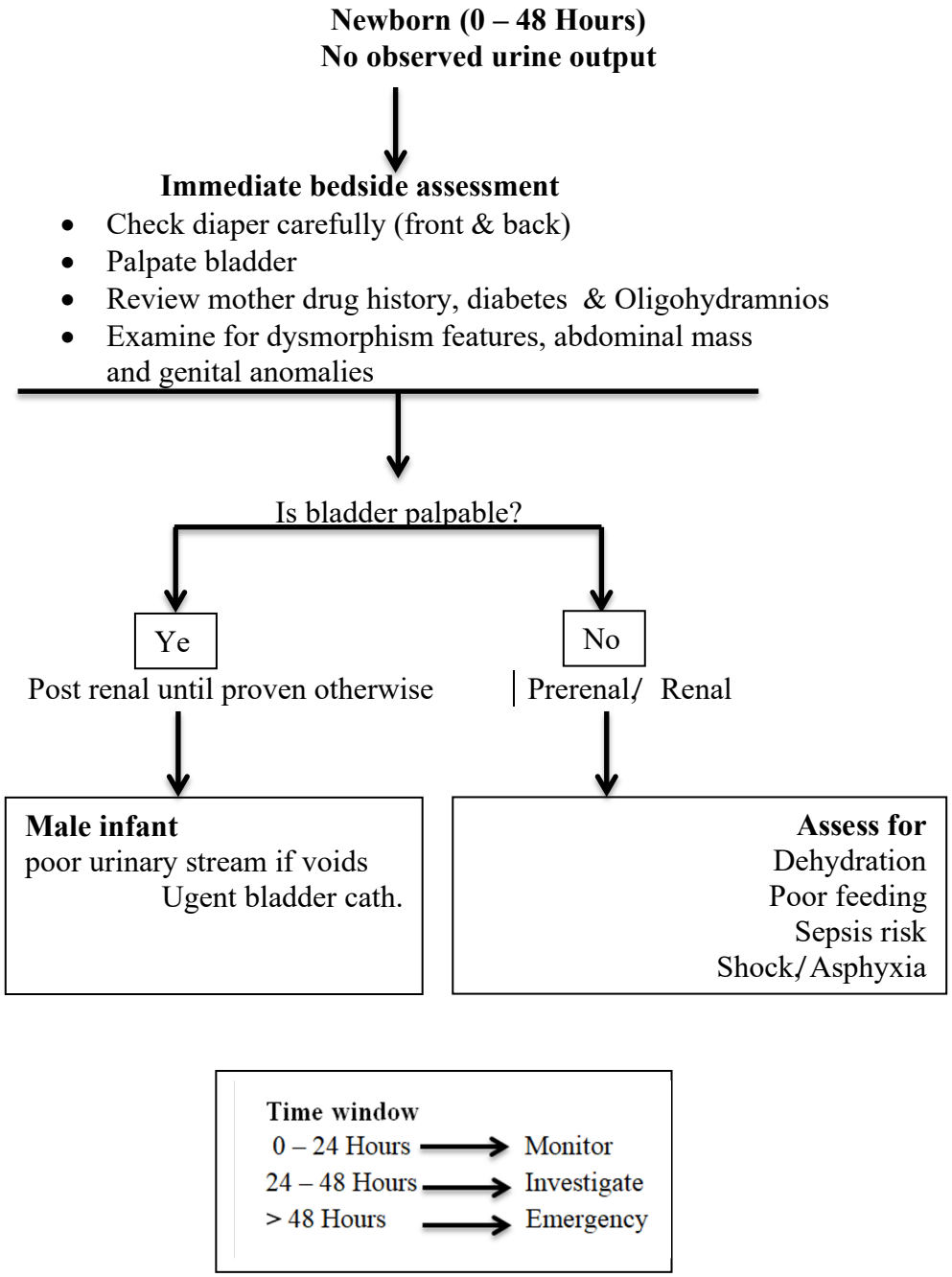

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