Electrolyte Disorders

Detlef Bockenhauer, MD, PhD[a,*], Jakub Zieg, MD, PhD[b]

KEYWORDS

• Electrolyte disorders • Hyponatremia • Hypernatremia • Hypokalemia
• Hyperkalemia • Newborn • Renal physiology

KEY POINTS

- Electrolyte homoeostasis is maintained by the kidneys.
- Disorders of plasma sodium commonly reflect disorders of water.
- Sodium handling by the kidneys is determined by volume homeostasis rather than plasma sodium.
- Volume (ie, sodium) homeostasis and potassium homeostasis are interdependent.
- Low glomerular filtration rate and tubular immaturity contribute to an increased frequency of electrolyte abnormalities in the neonatal period.

INTRODUCTION

The evolution of life started in the sea, which contained a steady concentration of salts. The function of living cells is thus critically dependent on a constant electrolyte composition, and the evolution of life on land was only possible because of the development of kidneys, which provided this constant "internal milieu."[1] Disorders in the electrolyte composition of this milieu thus can have serious consequences and are associated with morbidity and mortality.[2] Abnormalities of plasma sodium and potassium are a frequent occurrence in neonates, especially in the neonatal intensive care unit (NICU). To provide adequate treatment, it is important to understand the underlying problem and physiology.[3] For instance, a common response to hyponatremia is to increase sodium supplementation. Yet most patients with hyponatremia do not have a sodium deficiency, but water excess. Increasing sodium administration in these patients may correct the hyponatremia but will result in volume overload, which has serious risks in the neonatal period, such as patent ductus arteriosus, bronchopulmonary dysplasia, and necrotizing enterocolitis.[4–6]

Disclosures: None.
[a] UCL Institute of Child Health, Great Ormond Street Hospital for Children NHS Foundation Trust, Nephrology Unit, 30 Guilford Street, London WC1 3EH, UK; [b] Department of Pediatrics, 2nd Faculty of Medicine, Charles University in Prague and Motol University Hospital, V Úvalu 84, 15006 Praha 5, Czech Republic
* Corresponding author.
E-mail address: d.bockenhauer@ucl.ac.uk

Clin Perinatol 41 (2014) 575–590
http://dx.doi.org/10.1016/j.clp.2014.05.007 **perinatology.theclinics.com**
0095-5108/14/$ – see front matter

This article reviews the physiology of renal water and electrolyte handling with respect to dysnatremias and dyskalemias in the context of the special circumstances of the transition from intrauterine to extrauterine life. In addition, some rare inherited disorders associated with neonatal electrolyte abnormalities are discussed.

Basics of Renal Water and Electrolyte Handling

In an average adult (surface area 1.73 m^2) with a glomerular filtration rate (GFR) of 100 mL/min, the kidneys produce 144 L of primary filtrate a day. Assuming a sodium and potassium concentration of 140 and 4 mmol/L, respectively, these 144 L contain approximately 20,000 mmol of sodium and 500 mmol of potassium. While most (60%–80%) of this is reabsorbed isotonically in the proximal tubule, there is still a large volume of water, sodium, and potassium delivered to the distal tubule, where decisions can then be made about either reabsorption or excretion. Urine osmolality can range from less than 50 to greater than 1000 mOsm/kg, so that, depending on intake and extrarenal losses, urine output can vary roughly between 500 mL and 20 L per day. Similarly, tubular sodium reabsorption can be adjusted so that sodium excretion may range from less than 10 to greater than 1000 mmol per day.[7] Potassium can even be secreted, so that potassium excretion may exceed the filtered amount.[8] Thus with normal kidney function renal water, sodium, and potassium excretion can be adjusted over a very wide range to provide homeostasis even under extreme circumstances. However, with decreased GFR the ability of the kidneys to maintain volume and electrolyte homeostasis diminishes, so that abnormalities can occur more easily.

The Special Circumstances of the Neonatal Kidney

Although the same physiologic principles apply to neonatal and adult kidneys, there are some important differences in the ability to maintain water and electrolyte homeostasis.

- *Neonatal kidneys have a low GFR*. GFR measured by creatinine clearance in preterm infants from 27 to 31 weeks of gestation without apparent kidney disease can be lower than 10 mL/min/1.73 m^2 in the first week of life, only increasing to greater than 15.5 mL/min/1.73 m^2 by 4 weeks of life.[9]
- *Urinary concentrating ability is not fully developed until about 1 year of age.* In fact, all neonates have a degree of physiologic nephrogenic diabetes insipidus, so that maximal urine concentration may not exceed 300 mOsm/kg, even in a term neonate.[10,11] It is because of this decreased urinary concentrating capacity that normal saline, which is commonly recommended as the basic intravenous fluid solution in older children,[12] is not suitable in the NICU, as it typically will be hypertonic in comparison with the baby's urine and thus may lead to hypernatremia.

The impaired ability of the neonatal, and especially premature kidneys to maintain electrolyte homeostasis is also reflected in the wider reference range for plasma electrolytes. For instance, plasma sodium levels between 125 and 150 mmol/L are usually considered normal in this age group.[13] This relative instability is further compounded by some factors specific to the transition from intrauterine to extrauterine life and the early neonatal period:

- *Extrarenal water losses are increased* because of the greater ratio of surface area to body mass, and will be further increased by the use of radiant heaters and ultraviolet therapy. Moreover, immature skin is more permeable to water, probably because of higher expression of water channels (aquaporins).[14,15]