Urine Electrolytes and its Osmolality

An Essay

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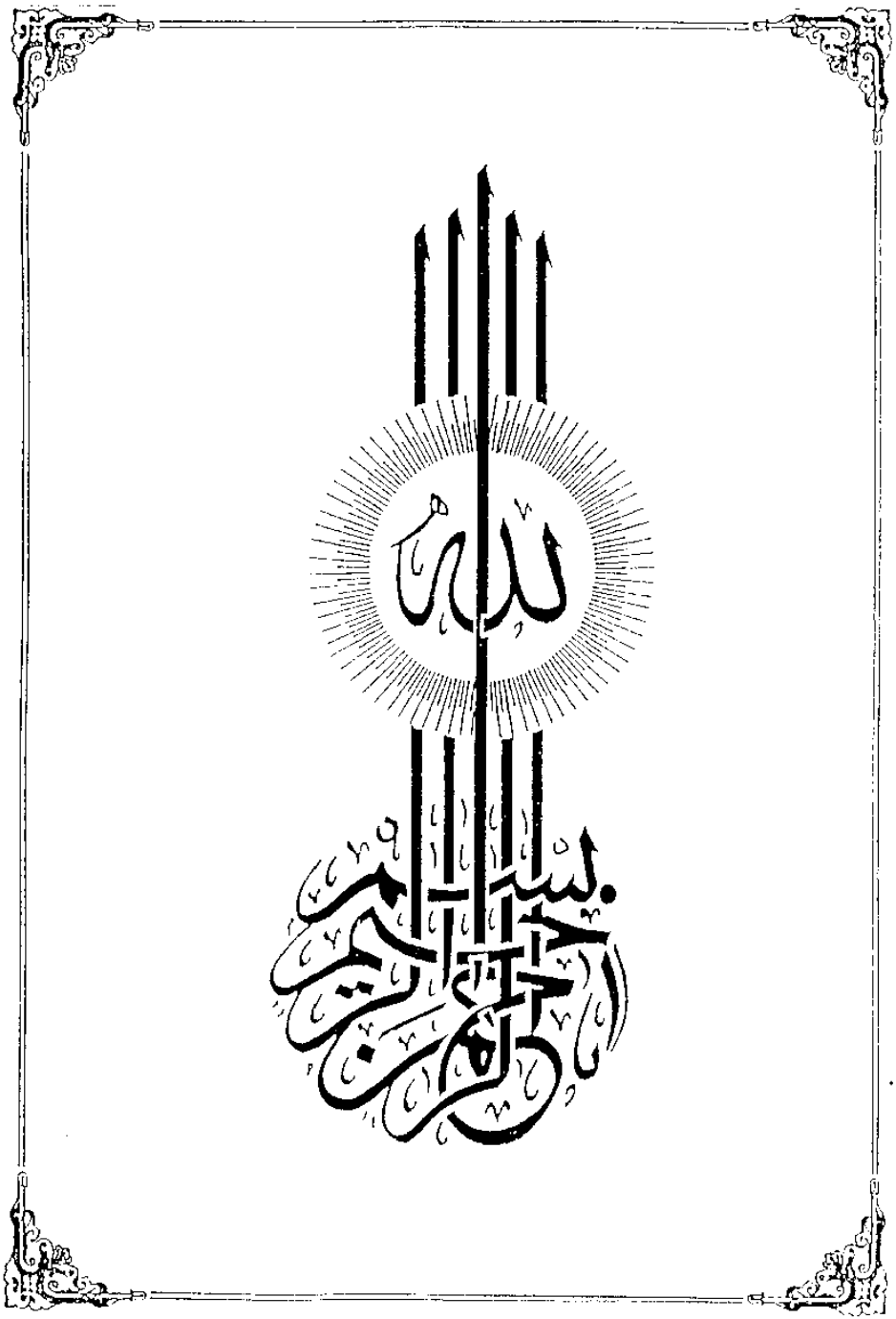

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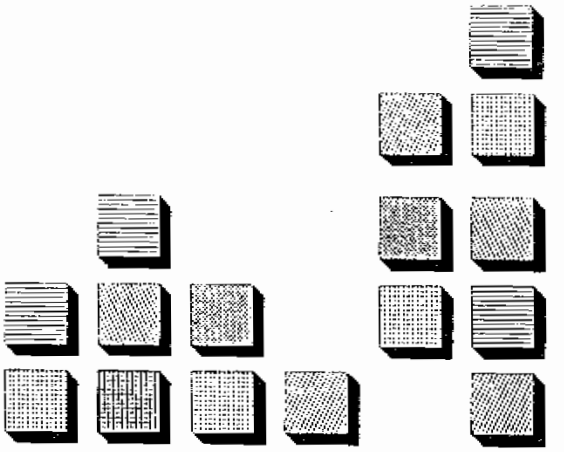


INTRODUCTION

Recent studies have revealed that the assessment of urine electrolytes and osmolality can provide valuable information and important clues to the diagnosis and management of disorders of fluid, electrolytes and/or acid-base metabolism. It can also give important complementary information in certain pathological condition, when interpreted in conjunction with the history, physical examination and other laboratory data **(Behrman et al., 1992)**.

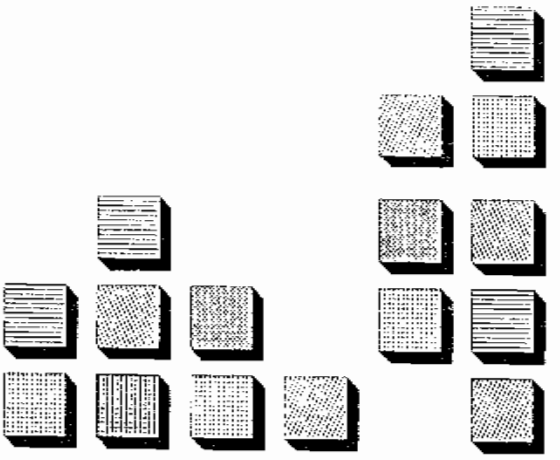
Although there are usual ranges "for solute and water excretions, there are no normal values. Thus values must be interpreted in view of what is the expected response for each particular clinical situation. Accurate interpretation of data regarding urine electrolytes concentration and osmolality requires updated and comprehensive knowledge of the different mechanisms of water and electrolyte homeostasis **(Kamel et al., 1990a)**.

The major electrolytes that are routinely measured include sodium, chloride and potassium. There are other electrolytes normally present in urine that are not routinely measured including ammonium, calcium, magnesium, bicarbonate, sulfate, phosphate and other organic anions.

The Aim of the Work

The purpose of this review is to provide a detailed account on the value of urinary electrolytes (sodium, potassium and chloride) and osmolality in the diagnosis of various disorders of fluid, electrolytes and acid-base metabolism. It will also define the different methods used for assessment of urinary electrolytes and osmolality. Furthermore, this review will focus on the basic physiology of fluid and electrolytes excretion by the kidney.

REVIEW OF LITERATURE



URINARY SODIUM

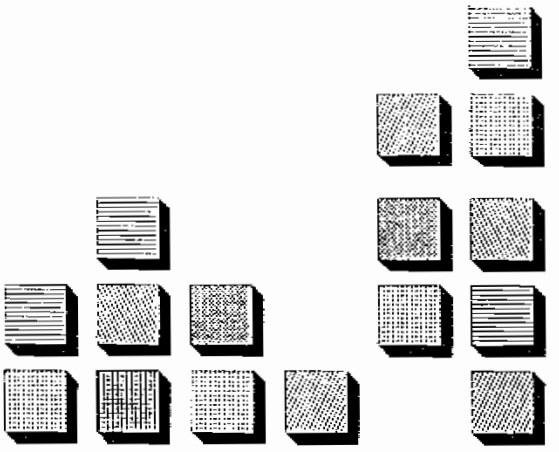
General features of sodium excretion by the kidney

Factors modulating sodium excretion

Clinical value of urinary sodium measurement

Tests used for assessment of urinary sodium

Methods for the determination of urinary Na and K



CHAPTER I
URINARY SODIUM

Sodium is the major cation of the extracellular fluid. It is the principal osmotically active solute responsible for the maintenance of intravascular and interstitial volumes. The quantity of sodium in the body approximates 48mmol/Kg, more than 30% of which is either exchangeable or only slowly exchangeable **(Halperin and Skorecki, 1986)**.

Urinary excretion of sodium varies with dietary intake but a typical range observed for persons on an average diet is 40-220mmol/day. the rate of sodium excretion shows large diurnal variation. During the night, it is only 20% of the peak during the day **(Tietz et al., 1986)**.

(A) General Features of Sodium Excretion by the Kidney

Regulation of sodium excretion depends on the balance between glomerular and tubular functions. Normally, the amount of sodium filtered daily by the kidneys is more than 100 times that ingested and more than five times the total amount of sodium in the body. However less than 1% of filtered sodium is excreted in the urine and the remaining 99% is reabsorbed along the length of the renal tubule **(Behrman et al., 1992)**.

Under normal conditions, changes in glomerular filteration rate (GFR) do not affect homeostasis, since changes in the filtered load of sodium produced by alterations in GFR are compensated for by appropriate changes in tubular reabsorption of sodium **(Klahr et al., 1973)**. Sodium reabsorption occurs along the entire nephron.

(1) Proximal Convoluted Tubule

Approximately two thirds (60%) of filtered sodium is reabsorbed by the proximal convoluted tubule. This fraction increases with contraction of extracellular fluid volume, and it decreases with volume expansion. The precentages of filtered sodium and water reabsorbed in the proximal tubule are proportional, so that the fluid remaining at

the end of proximal convoluted tubule has a sodium concentration compartable to that in the blood **(Behrman et al., 1992)**.

Net movement of soduim out of the proximal convoluted tubule represents the balance between sodium reabsorbed from the luminal fluid and that returned through intercellular space. Because such a high flux of soduim enters the epithelial cell across the luminal membranes, the sodium flux is unlikely to occur by purely passive mechanisms. Reabsorbed sodium is actively transported out of the cell across their basolateral membranes, producing an osmotic gradient that casues the movement of an equivalent amount of water **(De Wardener, 1978)**. The resulting hydrostatic force in the intercellular spaces and interstitial fliud, as well as the oncotic pressure exerted by the plasma protein in the peritubular capillary are responsible for returning the reabsorbed sodium and water into the vascular space. The balance between glomerular filteration rate and reabsorption of fluid from the proximal tubule (glomerulo-tubular balance) may be modulated through changes in the protein concentration in the blood at the level of the glomerular and peritubular capillaries **(Green et al., 1974)**.

(2) The Loop of Henle

Significant sodium reabsorption occurs in the loop of Henle and is central to the countercurrent multiplier system essential for water balance and the concentration of urine. Water reabsorption occurs in the descending limb of the loop of Henle, sodium reabsorption in the ascending limb. Sodium transport at this site may be secondary to active transport of the chloride rather than primary as it is at most other sites **(Kokko, 1970)**.

Although the loop of Henle is important in overall control of sodium reabsorption, no precise regulating mechanism has yet been delineated, nor has a maximal rate for sodium transport at this site been demonstrated. When the load of sodium delivered to the loop is increased, by changes either in GFR or in sodium reabsorption in the proximal tubule, most of the excess load is reabsorbed in the loop, providing a further protective mechanism and limiting the magnitude of changes of sodium delivery to the distal convoluted tubule **(Behrman et al., 1992)**.

(3) The Distal Nephron

The fine regulation of sodium balance probably occurs throughout the distal nephron in both the distal convoluted tubules and the collecting ducts. Sodium reabsorption at these sites is stimulated by aldosterone whose secretion is governed by the

renin-angiotensin system **(De Wardener, 1978)**, and by aspect of potassium balance **(Malnic et al., 1966)**.

Throughout the distal tubule and collecting duct, sodium is reabsorbed against a large concentration gradient from lumen to plasma. However, in comparison with the proximal convoluted tubule and the loop of Henle, the total capacity for sodium reabsorption is more limited. Thus, if the load of sodium reaching the distal tubule increases significantly, reabsorption does not increase proportionately and the added load is excreted in the urine **(Kokko, 1987)**.

Additional mechanisms may be responsible for renal regulation of sodium. Cortical nephrons, which have short loops of Henle, may be sodium-losing nephrons and the juxta medullary nephrons with long loops of Henle may be sodium retaining nephrons. Sodium balance could be accomplished by altering the proportion of renal blood flow directed to these two populations of nephron. Such a regulatory mechanism could be intrarenal and respond to local release of renin **(Behrman et al., 1992)**.