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Renal adaptive changes and sodium handling in the fetal-to-newborn transition

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A B S T R A C T

Appropriate fluid and electrolyte management is critical for optimal care of very low birth weight or sick infants. Delivery of such care requires an understanding of developmental changes in renal water and salt handling that occur with advancing gestational age as well as postnatal age. This review focuses on the principles of sodium homeostasis during fetal and postnatal life. The physiology of renal tubular transport mechanisms, as well as neurohumoral factors impacting renal tubular transport are highlighted. Clinical implications and guidelines to the provision of sodium to this vulnerable population are also discussed.

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1. Introduction

The placenta plays a primary role in the regulation fluid and electrolyte homeostasis in the fetus. Though not essential for regulation of extracellular fluid volume before birth, the fetal kidneys become increasingly functional during development. Following birth, the newborn is entirely dependent upon the kidney to maintain sodium and water balance. Preterm birth poses a unique situation is which the immature kidney, lacking fully functional regulatory systems, is forced to assume the role that the placenta would otherwise take. The developmental changes in the mechanisms by which the maturing kidney attempts to meet these needs are discussed.

2. Renal regulation of sodium homeostasis during development

In utero, the major regulator of fluid and electrolyte balance is the placenta and innate renal function is not necessary to maintain fluid and electrolyte homeostasis. The fetus experiences an intake (net transplacental transfer) of water and sodium that greatly exceeds that of the newborn. In late gestation fetal sheep, an animal model which has provided a large amount of the experimental data to help us understand developmental renal function and physiology, the net transplacental fluid transfer has been estimated at 0.4 mL/kg/min (24 mL/kg/h), net transfer of sodium being 0.76 mEq/kg/h (18.2 mEq/kg/day) [1,2]. Using a sodium tracer method, sodium transfer across the human placenta has been estimated to be about 130 mEq/d at term [3]. Whereas a certain amount of water and sodium is retained by the fetus for growth, the healthy fetus is required to excrete a large amount of water and sodium, primarily via urine and to a lesser extent lung liquid. The fetal kidney accomplishes this need even though it receives a comparatively small fraction of the cardiac output (3% vs 15% in the newborn and 25% in the adult) because mechanisms to retain sodium are relatively immature [4–6]. Fetal urinary excretion of sodium, (urine sodium concentration (U_{Na}) × volume/unit time) $U_{Na}V$ (expressed as mEq/min), and $U_{Na}V$ normalized to glomerular filtration rate (GFR), and thus expressed as fractional excretion of sodium (FE_{Na}), is greater in the fetus than in the newborn. In fact, FE_{Na} is greater in the fetus at 0.75 gestation (14–15%) than 0.9 gestation (11%) or near term (5%, 145 days), suggesting that developmental changes in renal tubular sodium transport occur in utero [7].

Data regarding human fetal renal function are limited, but available information provides important insight into maturational changes in renal sodium metabolism. Human fetal urine production occurs as early as 12 weeks after conception [8]. Analysis of fetal urine obtained from infants diagnosed prenatally with obstructive uropathy though showing no clinical evidence of renal dysfunction at 1–2 years of age demonstrated a reduction in urine Na concentration with a concomitant increase in (urinary?) creatinine as gestational age advanced between 20 and 38 weeks [8]. By contrast, urine protein and phosphorus concentrations are low and

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remain unchanged over this period of time. Taken together, these data suggest that glomerular function and tubular reabsorption of phosphorus reach maturity earlier in development than tubular sodium reabsorption. Similar developmental changes in urine sodium concentration were reported from fetuses whose urine was obtained prior to elective termination or red blood cell transfusion for Rh isoimmunization [9]. Along with a decreasing urine sodium concentration, fractional excretion of sodium similarly decreases during the second half of gestation [10].

Marked changes in renal sodium handling occur with and following birth, the extent of which is dependent upon gestation. In term fetal sheep delivered by cesarean section, $U_{Na}V$ and FE_{Na} are initially maintained at fetal levels, though rapidly decrease during the first few hours of postnatal life (Fig. 1) [11]. These findings, which were associated with increased glomerular filtration rate (GFR) but not aldosterone levels, suggest a rapid increase in renal tubular sodium reabsorption. In term infants, FE_{Na} decreases from 3.4% to 1.5% in the first few hours of life and continues to decrease to adult values (<1%) over subsequent days [12]. Several studies have shown that in preterm infants FE_{Na} and $U_{Na}V$ are inversely associated with gestational age at birth and postnatal age (Figs. 2 and 3) [13,14]. Gabhaju et al. reported longitudinal data on 129 preterm infants stratified into four groups according to gestational age [15]. FE_{Na} exceeded 6% in infants <28 weeks of gestation on day of life 3, decreasing to about 4% by the end of the first week of life and to 2% at a month of age. Infants with less extreme prematurity (29–36 weeks of gestation), had lower FE_{Na} at 3 d of age and similarly demonstrated a maturational decrease in FE_{Na} over the first month of life. By 28 days of life, there was no statistically significant difference in FE_{Na} among the gestation age groups, though infants born at <28 weeks of gestation appeared to have FE_{Na} twice that of older infants. These findings suggest marked capacity for postnatal tubular maturation of sodium reabsorption

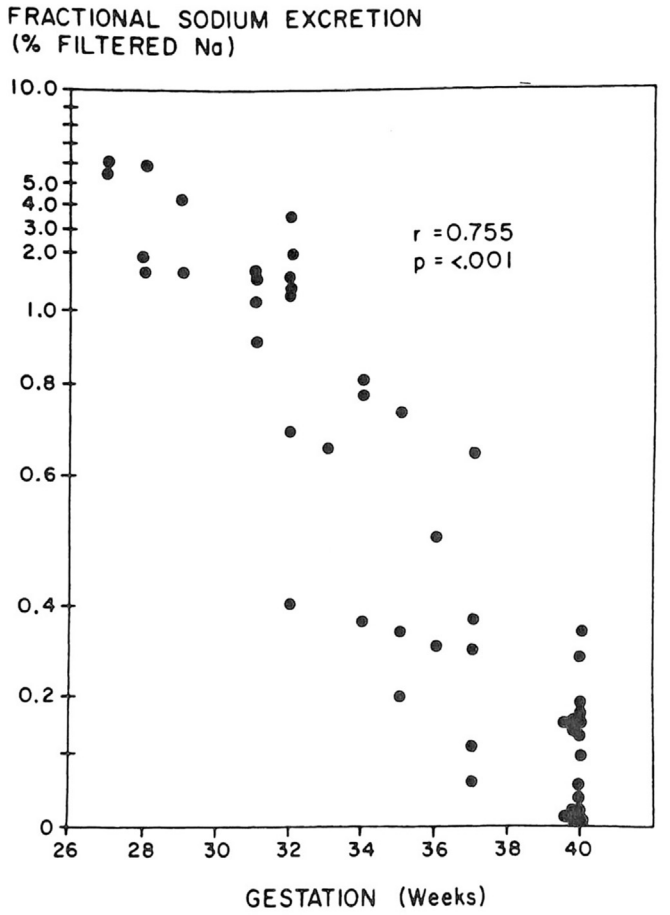

Fig. 2. Scattergram representing inverse correlation between calculated fractional excretion of sodium and gestational age in 60 infants between 24 and 40 h of age. (Reproduced with permission from Bueva and Guignard [13].)

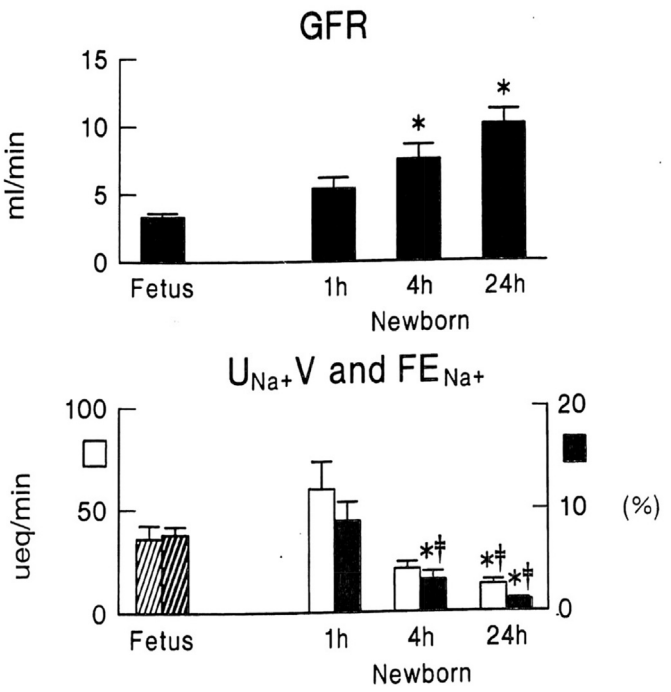

Fig. 1. Glomerular filtration rate (GFR), urinary sodium excretion ($U_{Na+}V$, μEq/min)) and fractional excretion of sodium (FE_{Na+}, %) before and after birth in near-term sheep delivered by cesarean section. $^{*}P < 0.05$ when compared with fetal values. $^{‡}P < 0.05$ when compared with newborn values at 1 h. Values are means ± SE. (Reproduced with permission from Nakamura et al. [11].)

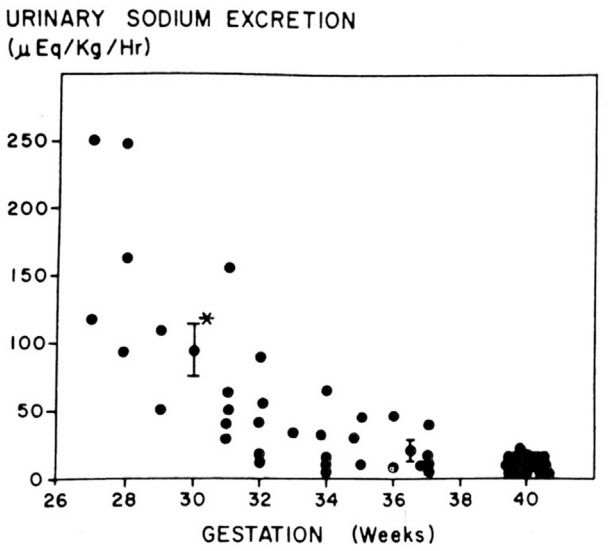

Fig. 3. Scattergram representing inverse correlation between urinary sodium excretion and gestational age in 60 infants between 24 and 40 h of age. Circles with bars plotted at 30 and 37 weeks of gestation represent mean ± SEM for values of 27–32 and 33–40 weeks of gestation, respectively. $^{*}P < 0.05$ compared with 37 week gestation value. (Reproduced with permission from Bueva and Guignard [13].)

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