A Study Of Renal Fnction in The New Born Infants

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At the time of birth. the kidney must 8asume the task of primary regulation of body fluid and electrolyte composition as well 8S responsibility for the exceretion of a v8riety of potentially toxic substance. The newborn infant in general and preterm infant in particular, have been considered to havesignificant limitation of renal function when adult standards are used as reference point. Urine formation begina from the 8th-llth weekof the fetal life. The fetal kidney is able to dilute and acidify urine. to absorb phosphate and to transportorganic materials. The placenta is able to meet the excretory needsof the fetus. in the neonate with bilateral ranal agensis, for example, the composition of the body tissuedoes not differ from normal. Renal blood flow and glomerular filtration rate increase linearly with gestational age. Vascular resistance is high in the fetal kidney and restricts renal blood flow and glomerular filtration rate inutero. At parturation, a dramatic rise in glomerular filtration rate occurs in infants greater than 34 weeks gestational age, which principally due to decrease in vascular resistance and a rise in syetemic blood pressure. In contrast, the premature neonate born before 34 weeks does not demonstrate this rapid rise in glomerular filtration that is noted in more mature babies. The low glomerular filtration rate that characterized the premature neonateh renal function renders him intolerant of excessive fluid volume and electrolyte loads. After the first 48 hours of life the urine excertionof a normal infant is 3 - 4 ml/kg/hr.The capacity of the infant’s to dilute urine is qualitatively the same as the adult’s and indicates adequate ability to deliver sodium and chloride to thediluting segment of the nephron. Sodium balance: Sodium homeostasis in the terminfant is characterized by a positive sodium balance over a wide range of sodium intakes. Conversely, the same infant will exhibit a limited ability to increase sodium excertion when given a saline load. Both of these phenomena are thought to be due to enhanced distaltUbular sodium resorption stimulated by high aldosterone levels. During the first year of life, there is a moregradual increase in these functions, and by the first year of age, expressed in r elation to weight or surface area, the value are comparsble to those of adults. Glomerular function is relatively more mature than tubular function. It results in a lower fractinal reabsorption of many filtered solutes in the proximal tubule than in the case in later life and probably accounts for the fact that infants excerete a higher percentage of glucose, phosphate and amino acids than do older children and adults. Ninety three percent of normal neonates voidwithin 24 hours after birth and 99 % 48 hours. The mean value of maximal urine osmolality in the newborn period is 600 - 700 mOsm/kg H20. This low value doesnot reflect an inability of the immature kidney to

concentrate urine but is an evidenc of the small amountof dietary protein that is metabolized and excreted as urea. The kidney maintains water homestasis by changingurine volume and concentration. A healthy full term infant aged 3-4 days can adaptively increase their water excretion and produce a more dilute Ufine after an increase in fluid intake. The delayed response towater load exhibited in the premature infant and term neonate may be due to a relatively low GRF. which limitedelivery of the nephron. especially in the immediate postnatal period. Water conservation by the mature kidney requires a hypereomotic medullary interstitium and is established by the loop of Henle countercurrent system. Water is passively reabsorbed fro. the distal tubule and collecting ducts under the influence of arginine vasopressin. The diagnosis of renal dysfunction in the neonate can be a challenging problem for the practicing pediatrician. Although there are real differences in renal function between term and preterm infants, over-all function is quite adequate in both groupe when fluid intake andenvironmental conditions ara carefully controlled. When confronted with an infant with a pethologic decrease inurine output, the clinician must provide adequste fluid resuscitation for the infant with prerenal aliguria without inducing fluid overload in the infant with established, intrinsic renal failure. In addition, the infant’with obstruction to urine flow must be distinguished. This require careful assessment of physioal findings and a few key laboratory determinations.