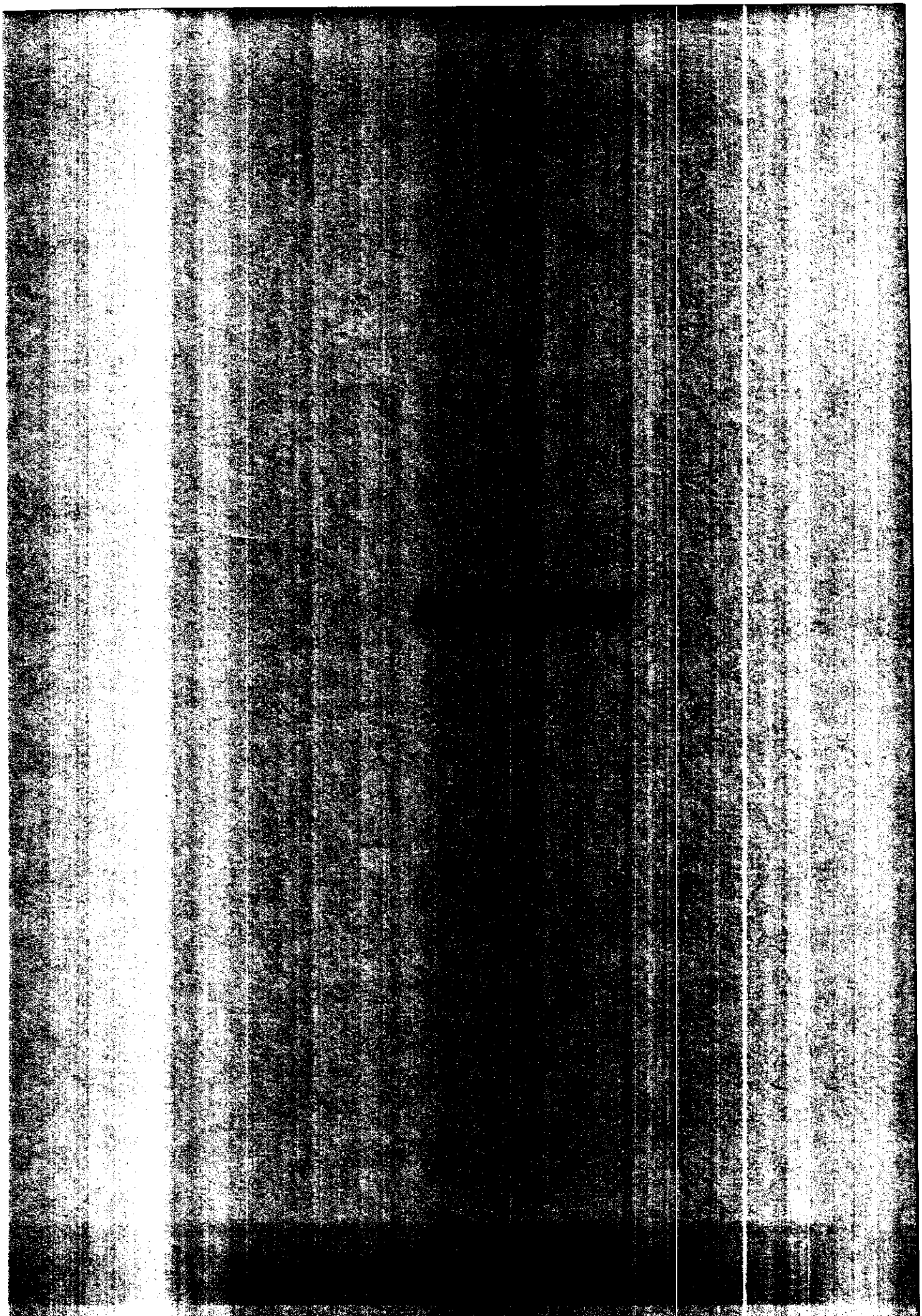

SUMMARY

At the time of birth, the kidney must assume the task of primary regulation of body fluid and electrolyte composition as well as responsibility for the exceretion of a variety of potentially toxic substance. The newborn infant in general and preterm infant in particular, have been considered to have significant limitation of renal function when adult standards are used as reference point.

Urine formation begins from the 8th - 11th week of the fetal life. The fetal kidney is able to dilute and acidify urine, to absorb phosphate and to transport organic materials.

The placenta is able to meet the excretory needs of the fetus, in the neonate with bilateral renal agensis, for example, the composition of the body tissue does 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 in utero. 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 systemic 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 neonate's renal function renders him intolerant of excessive fluid volume and electrolyte loads.

After the first 48 hours of life the urine excretion of 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 the diluting segment of the nephron.

Sodium balance: Sodium homeostasis in the term infant 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 distal tubular sodium resorption stimulated by high aldosterone levels.

During the first year of life, there is a more gradual increase in these functions, and by the first year of age, expressed in relation to weight or surface area, the value are comparable 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 void within 24 hours after birth and 99 % 48 hours. The mean value of maximal urine osmolality in the newborn period is 600 - 700 mOsm/kg H_2O . This low value does not reflect an inability of the immature kidney to concentrate urine but is an evidenc of the small amount of dietary protein that is metabolized and excreted as urea.

The kidney maintains water homestasis by changing urine volume and concentration. A healthy full term infant aged 3-4 days can adaptively increase their water excretion and produce a more dilute urine after an increase in fluid intake. The delayed response to water load exhibited in the premature infant and term neonate may be due to a relatively low GRF, which limits delivery of the nephron, especially in the immediate postnatal period.

Water conservation by the mature kidney requires a hypersomotic medullary interstitium and is established

by the loop of Henle countercurrent system. Water is passively reabsorbed from 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, overall function is quite adequate in both groups when fluid intake and environmental conditions are carefully controlled. When confronted with an infant with a pathologic decrease in urine output, the clinician must provide adequate fluid resuscitation for the infant with prerenal oliguria 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 requires careful assessment of physical findings and a few key laboratory determinations.