Chapter 3

RENAL DEVELOPMENT

Kidney derives from metanephros from embryonic mesoderm. During 5th week of embryonic life ureteric diverticulum develops as outgrowth of mesonephric duct from point near to cloaca. It grows headwards into nephrogenic cord and becomes surrounded by mesodermal tissue which will give rise to metanephros. Primitive nephrons develop in close proximity to it.

Ureteric bud devides and subdivides while growing towards periphery of metanephros. First nephrons to be formed are those deeply situated in kidney. As each branch of bud is surrounded by nephrogenic tissue fetal kidney assumes lobulated appearance.

New nephrons continue to form upto 36 weeks of gestation only. Thereafter increase of nephron mass is by increase of tubular length and glomerular size.

Fetal sclerosis occurs in youngest glomeruli when their tubules do not communicate with ureteric bud as its growth ceases in later stages of fetal life. Other glomeruli show features of immaturity varying from unvascularized clumps of epithelial cells to occasional cuboidal epithelium and limited lobulation of tufts. Tubular immaturity is even more profound than that of glomeruli for ratio of glomerular surface area to tubular volume is much greater than in later childhood.

Ureteric bud by process of subdivision followed by coalescence gives rise to renal pelvis, calyces and collecting ducts. During this process oldest, deepest nephrons are lost. Occasionally glomeruli in aberrant positions beneath pelvic mucosa or within arterial walls will survive.

Urinary bladder is formed from ventral and cephalic portion of cloaca after this has been separated from rectum by urorectal septum. Into this are incorporated caudal ends of mesonephric tubes and ureteric buds. These form bladder and urethra.

Anatomical evidence of renal immaturity at birth is mirrored physiologically by glomerular filtration rate, PAH clearance, renal bicarbonate and glucose reabsorption which are markedly diminished.

Normally less urea is produced by rapidly growing baby. More dietary nitrogen is deposited as protein in tissues. Anabolism, growth and renal clearance

are responsible for maintenance of normal blood urea levels in small infants. Excessive dietary protein, growth retardation and tissue catabolism (eg in acute infections) can lead to urea synthesis in excess of renal excretory capacity. Thus raised blood urea in infant while abnormal does not indicate necessarily impairment of renal function.

Diminished urea excretion is responsible for poor concentrating ability of newborn kidney.

Fetus excretes large amount of sodium and water before birth. At birth newborn is deprived of limitless transplacental supply of water and sodium and must suddenly conserve both of these. This is achieved within few days after birth. ❑