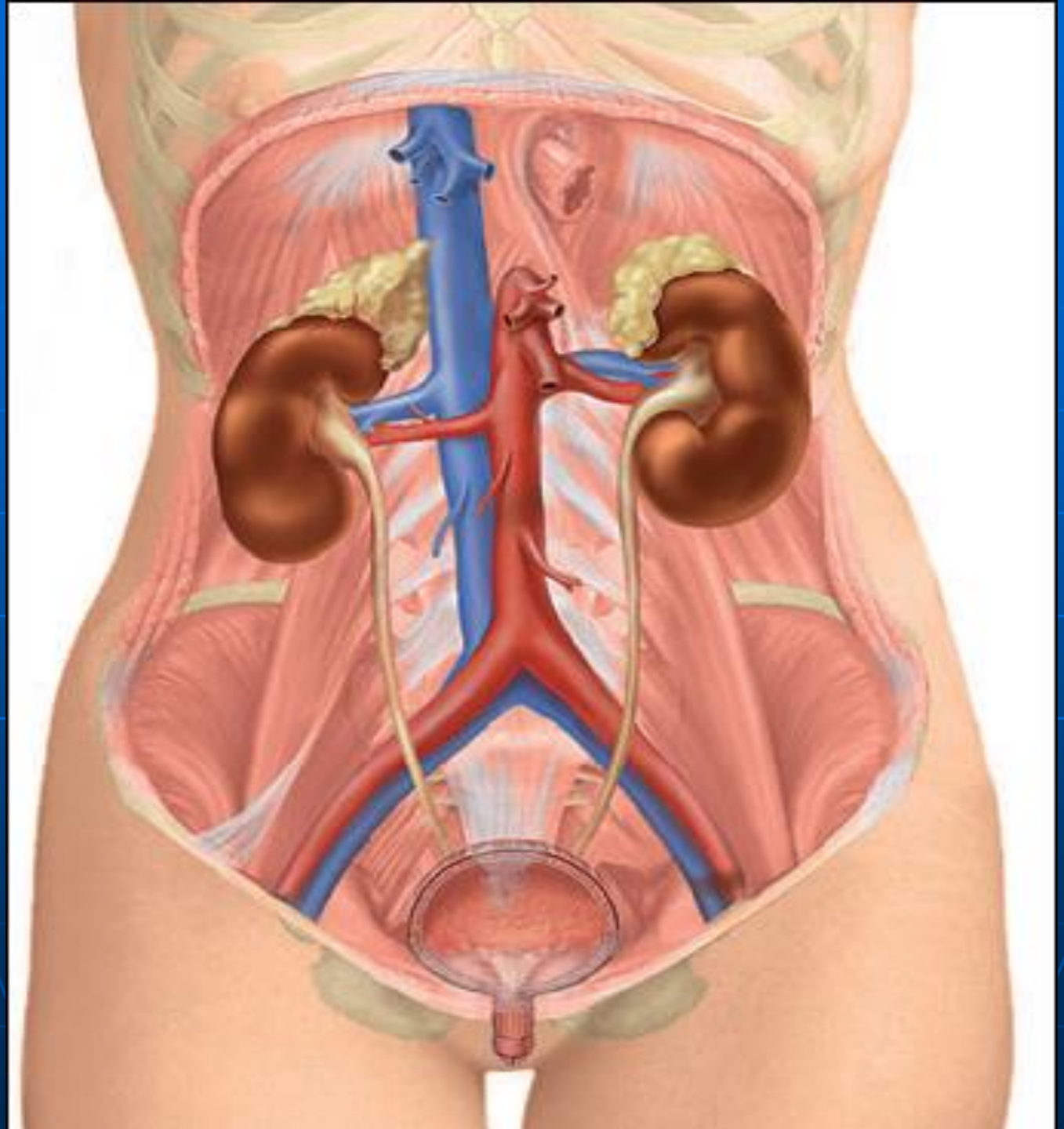
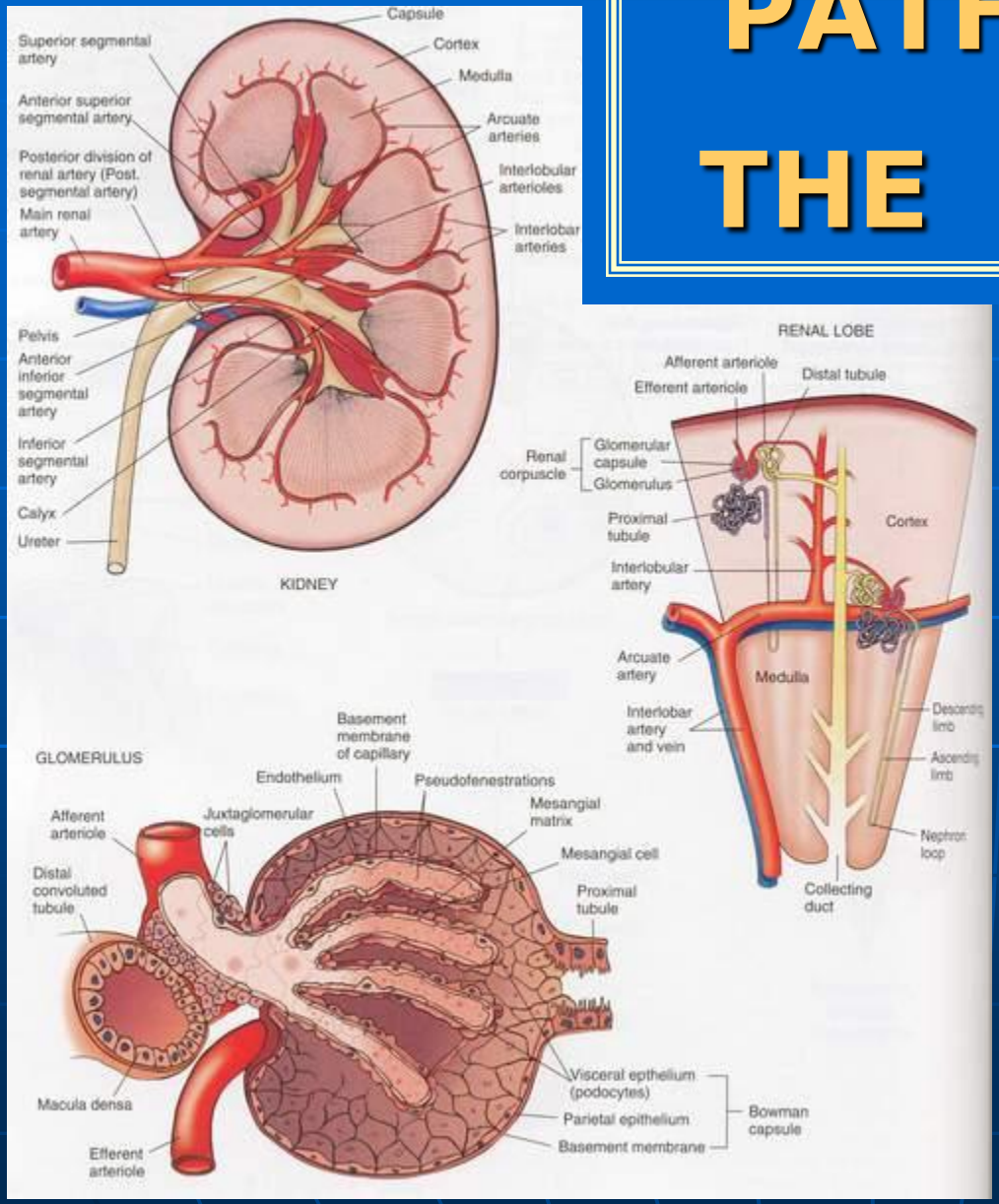
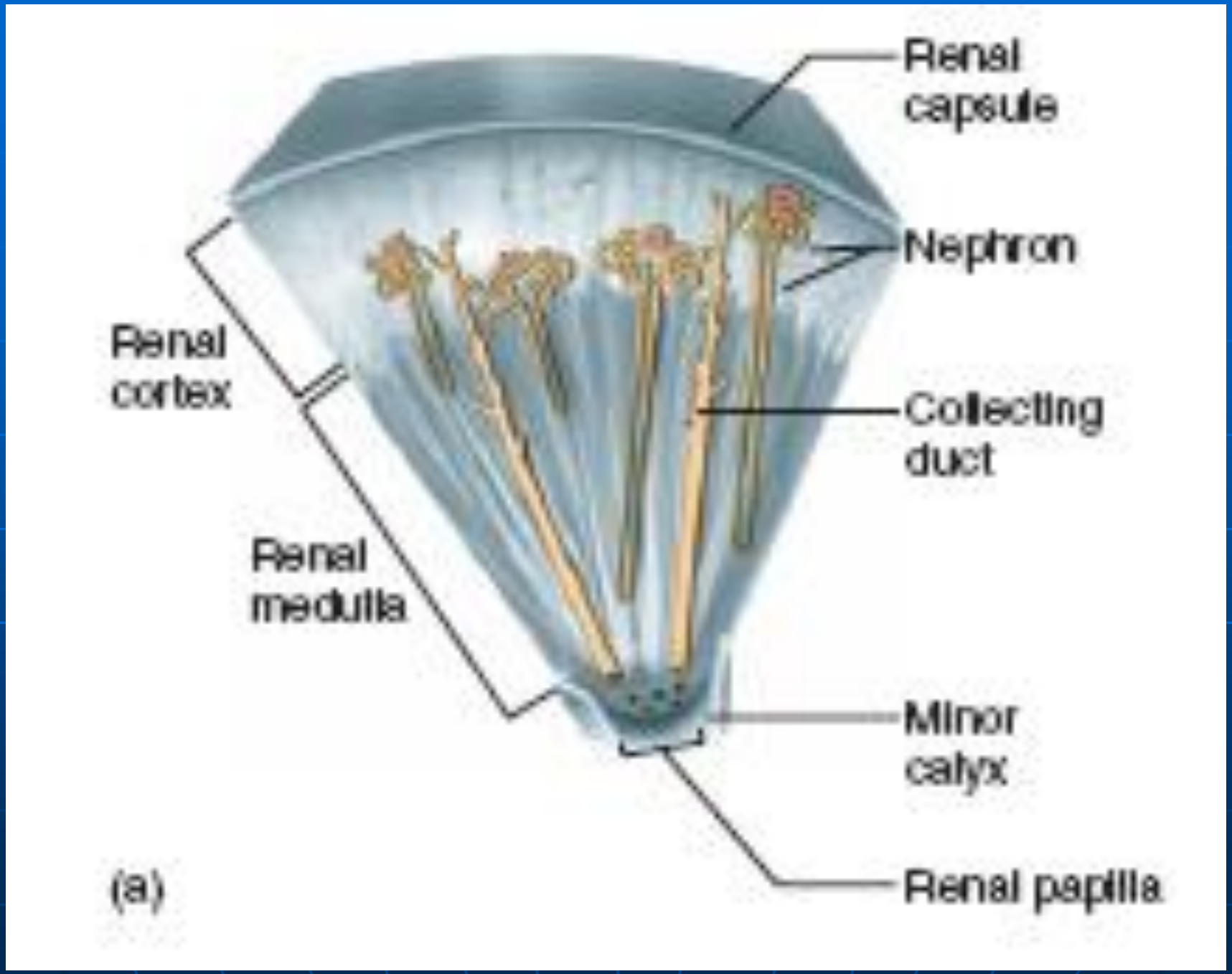
2. Pathology Anatomy of Urinary Tract

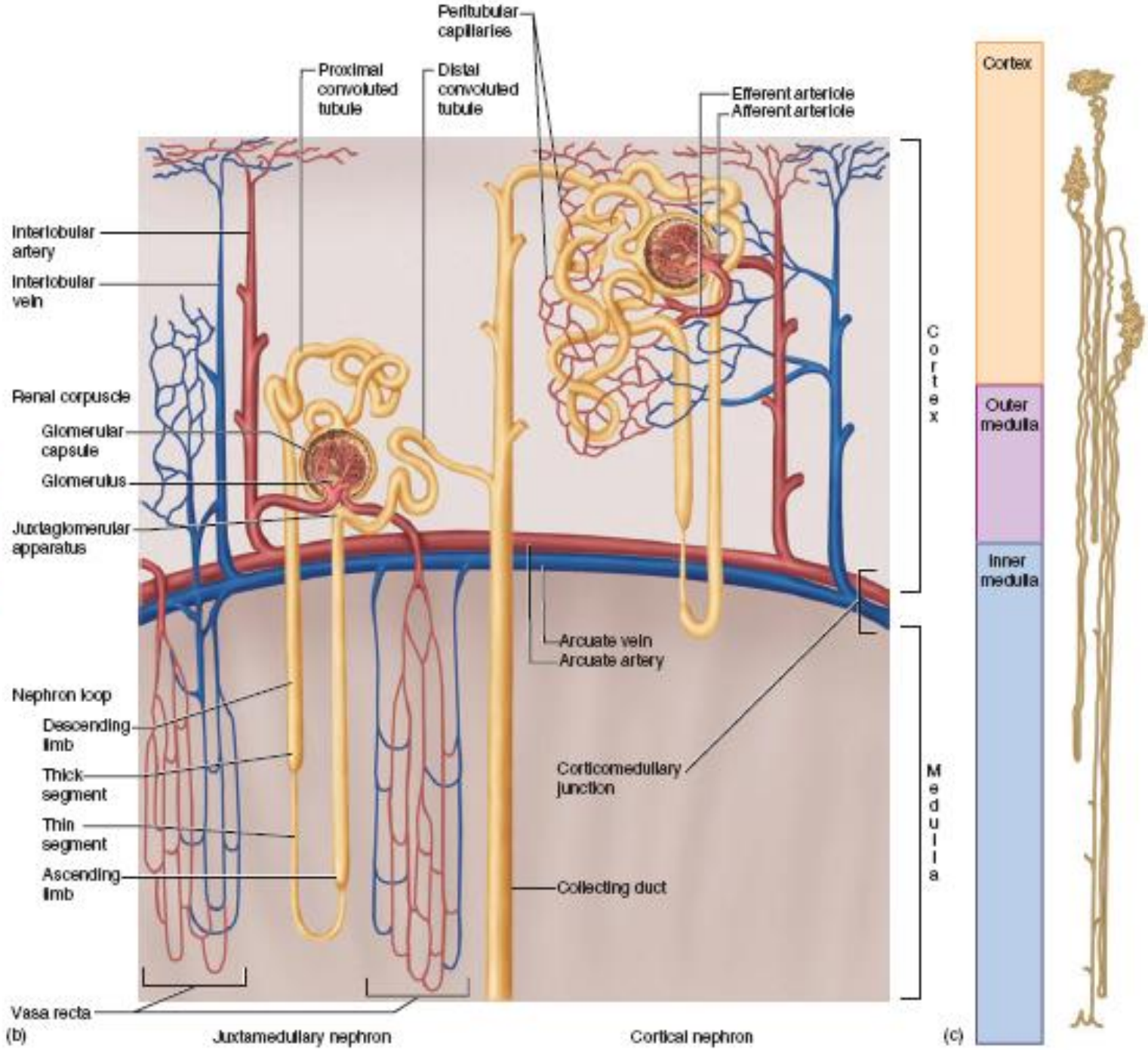
dr. Indrayanti, Sp.PA

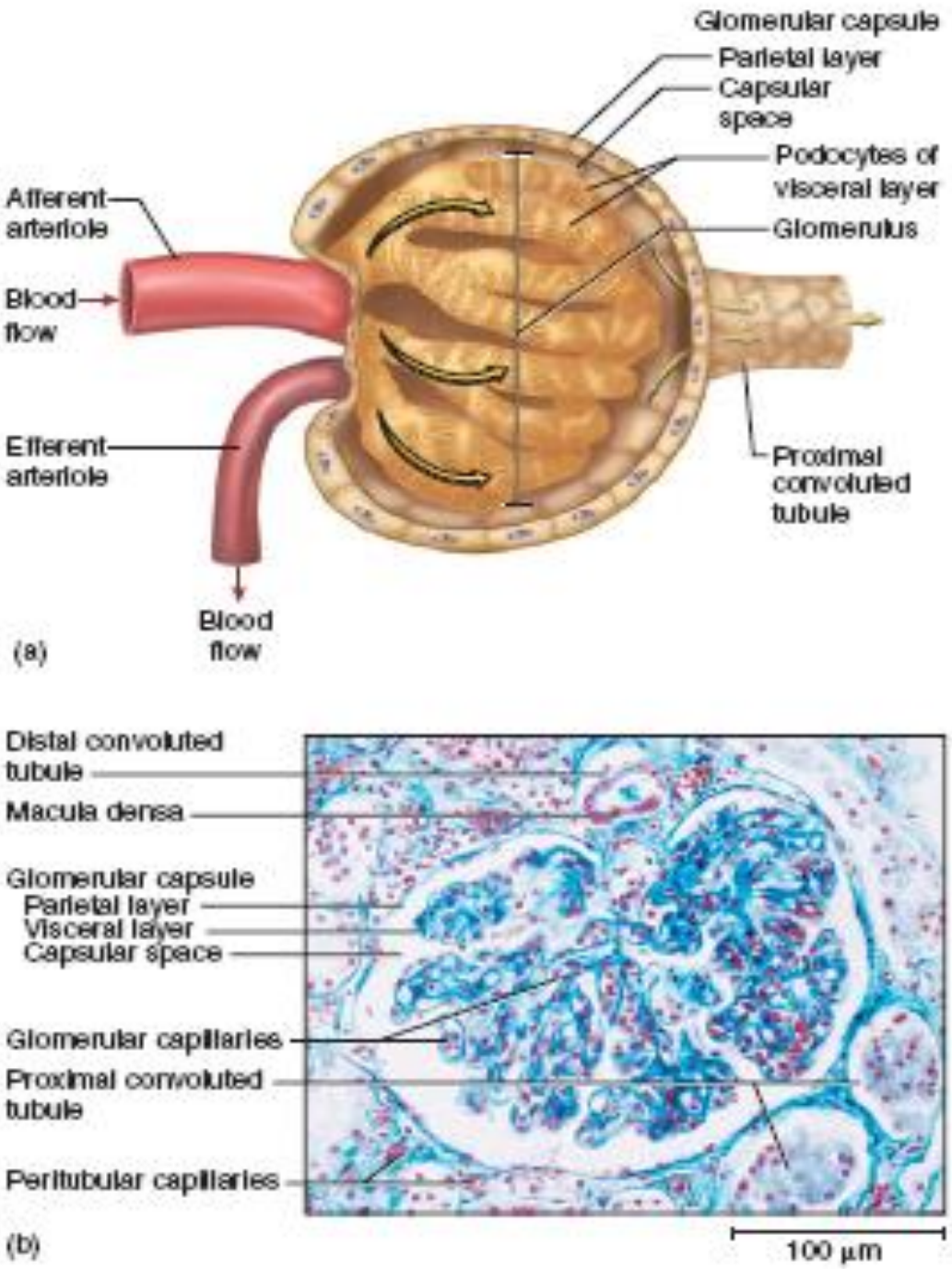


PATHOLOGY OF THE KIDNEY




Renal capsule
Nephron
Renal cortex
Collecting duct
Renal medulla
Minor calyx
Renal papilla
(a)


Peritubular capillaries
Proximal convoluted tubule
Distal convoluted tubule
Efferent arteriole
Afferent arteriole
Interlobular artery
Interlobular vein
Renal corpuscle
Glomerular capsule
Glomerulus
Juxtaglomerular apparatus
Arcuate vein
Arcuate artery
Nephron loop
Descending limb
Thick segment
Thin segment
Ascending limb
Corticomedullary junction
Collecting duct
Vasa recta
(b)
Juxtamedullary nephron
Cortical nephron
Cortex
Medulla
Cortex
Outer medulla
Inner medulla
(c)


Glomerular capsule
Parietal layer
Capsular space
Podocytes of visceral layer
Glomerulus
Afferent arteriole
Blood flow
Efferent arteriole
Proximal convoluted tubule
Blood flow
(a)
Distal convoluted tubule
Macula densa
Glomerular capsule
Parietal layer
Visceral layer
Capsular space
Glomerular capillaries
Proximal convoluted tubule
Peritubular capillaries
(b)
100 µm

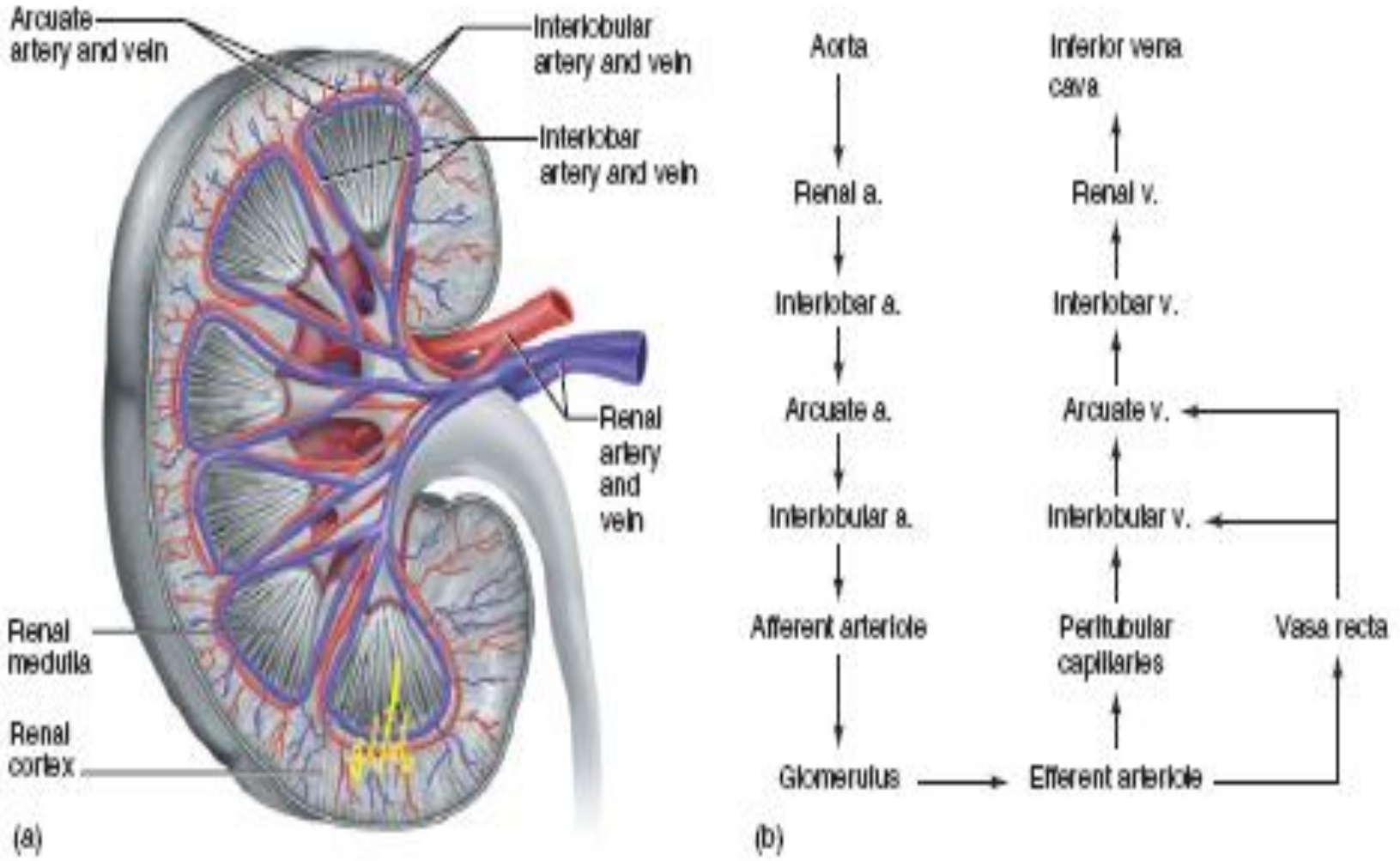
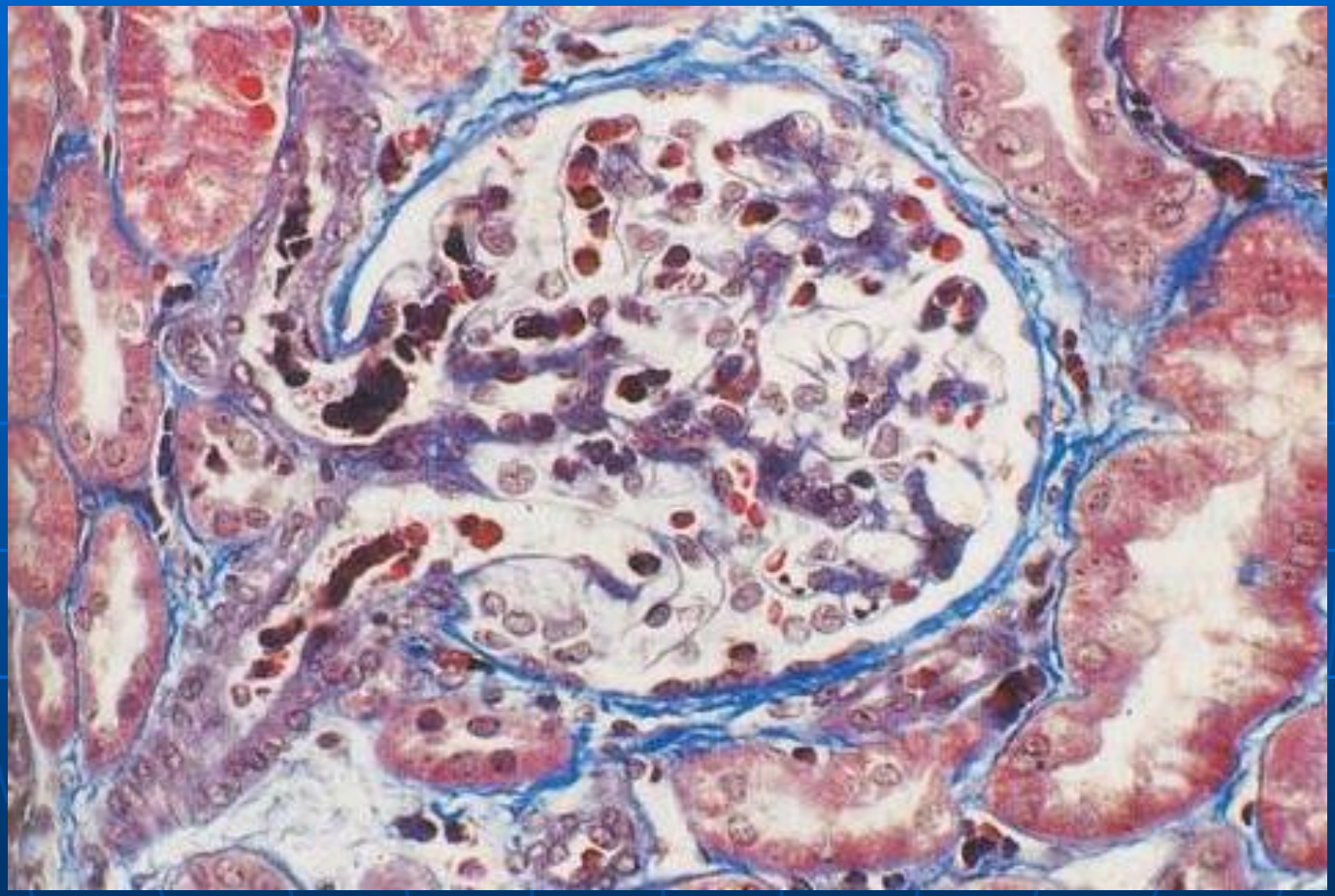
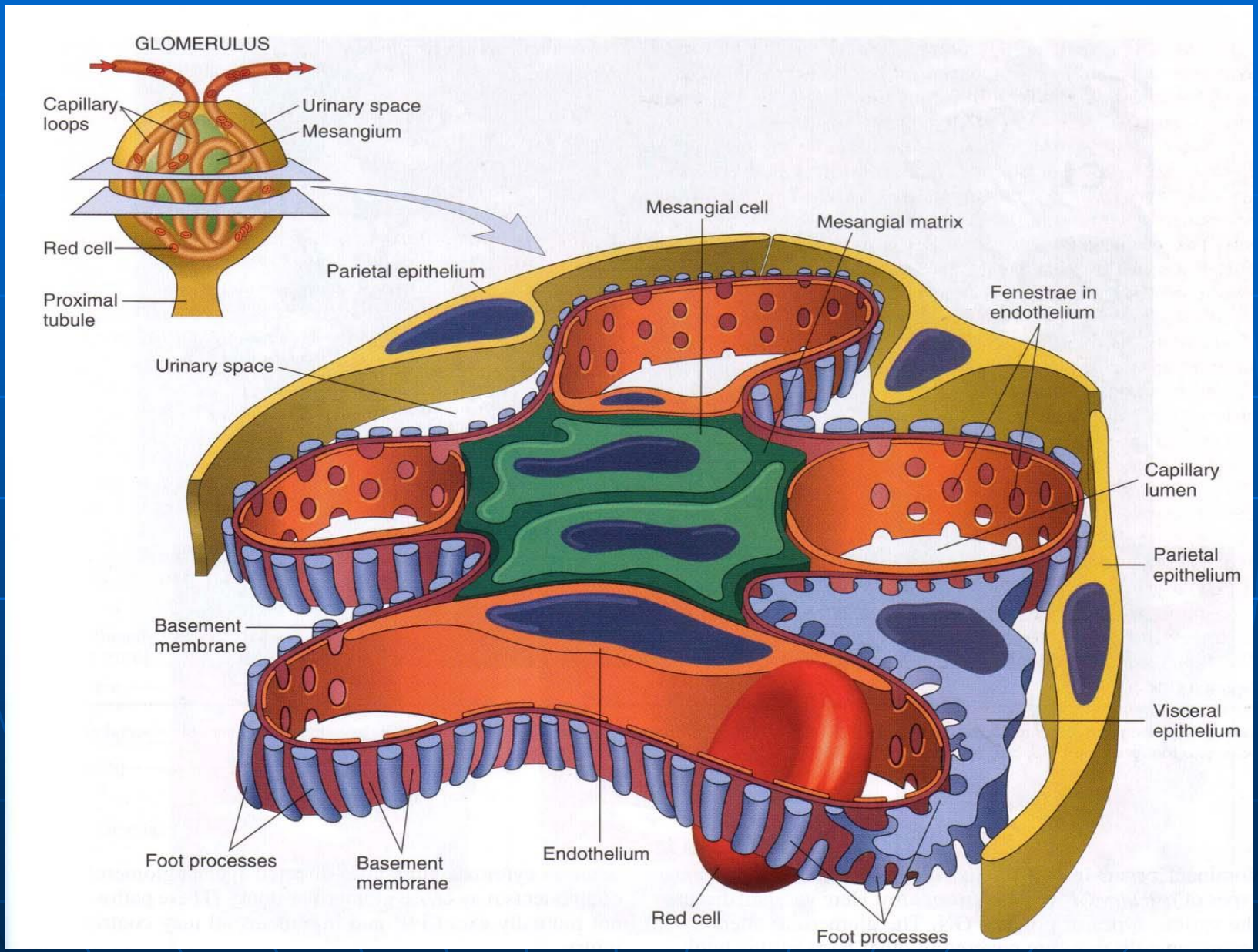


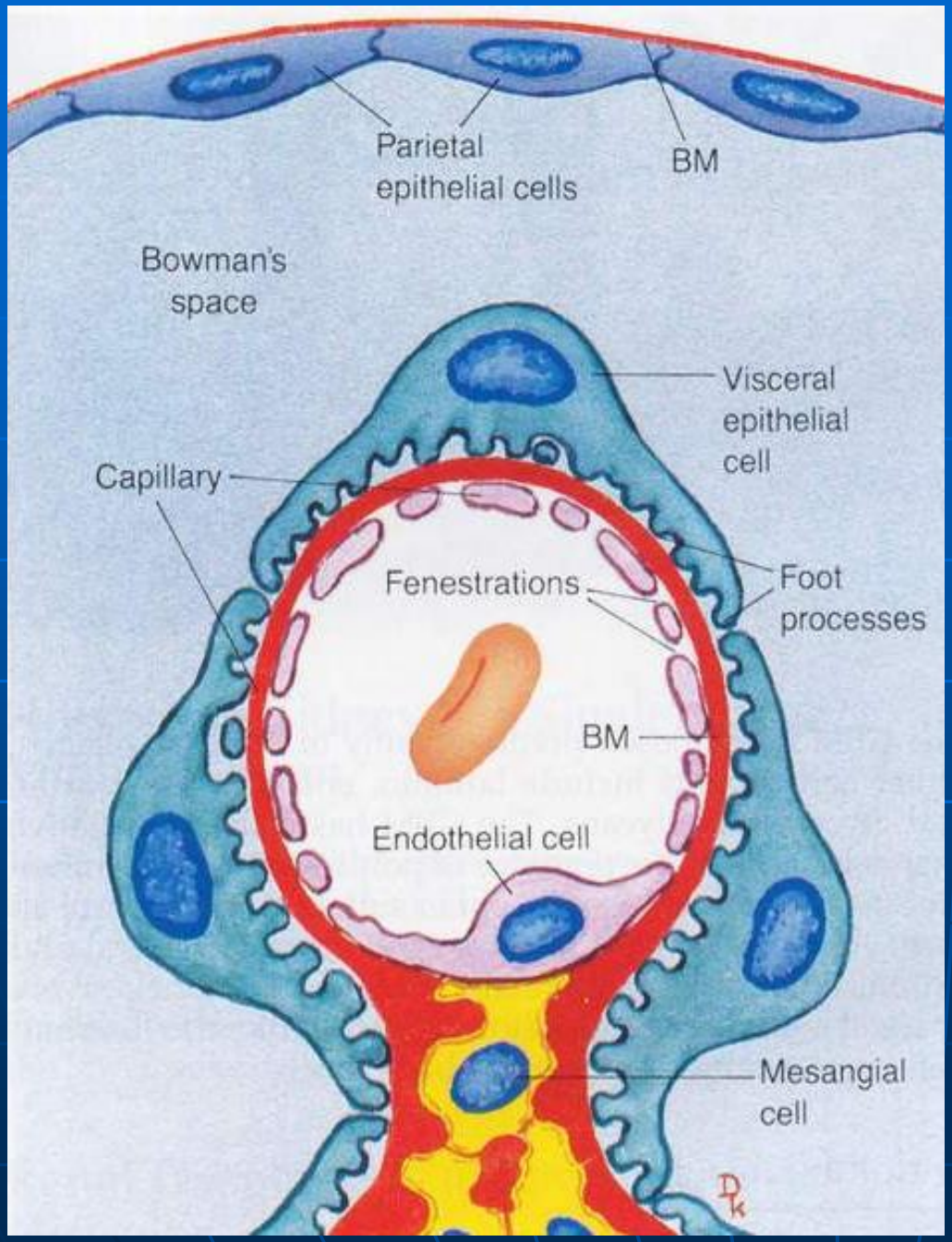
Figure 23.7 Renal Circulation. (*a*) The larger blood vessels of the kidney. (*b*) Flow chart of renal circulation. The pathway through the vasa recta (instead of peritubular capillaries) applies only to the juxtamedullary nephrons.

Normal Glomerulus



Schematic representation of a glomerular lobe





Normal Glomerulus

Single glomerular loop

The entire outer aspect of the GBM (peripheral loop and stalk) is covered by the podocyte (visceral epithelial cells) foot processes

The outer portion of the fenestrated endothelial cells are in contact with the inner surface of the GBM

The central part is in contact with the mesangial cells and adjacent mesangial matrix

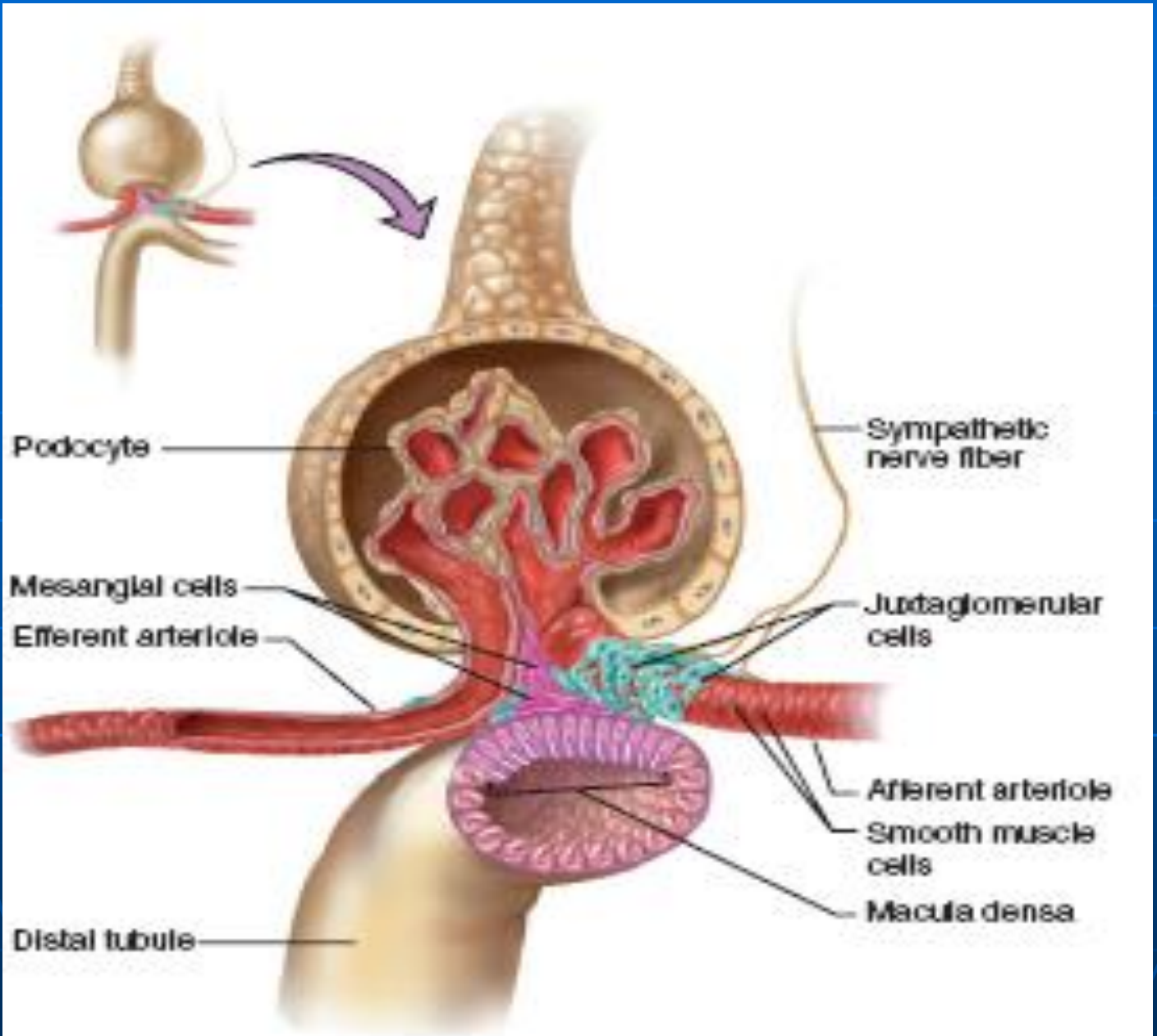
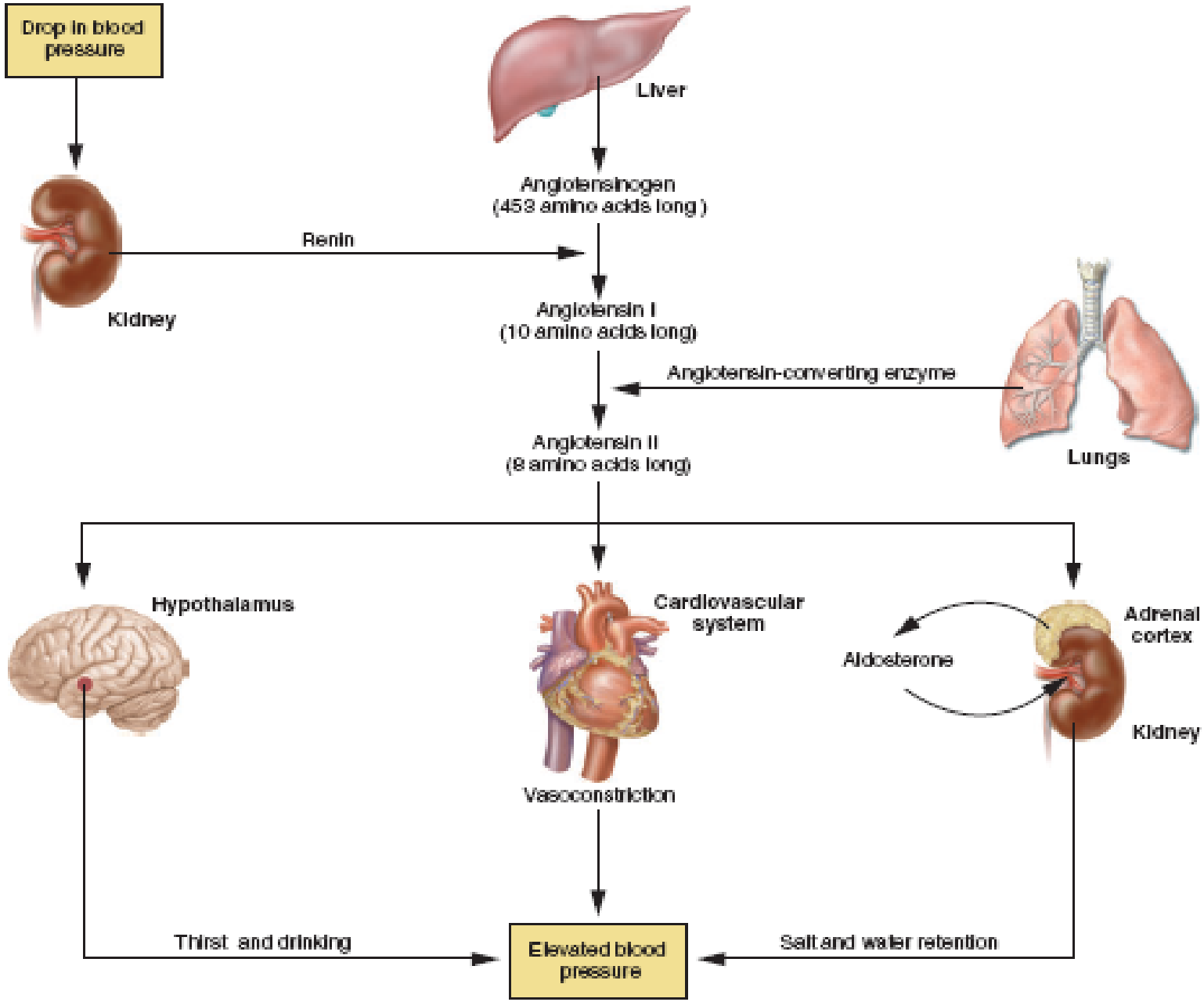
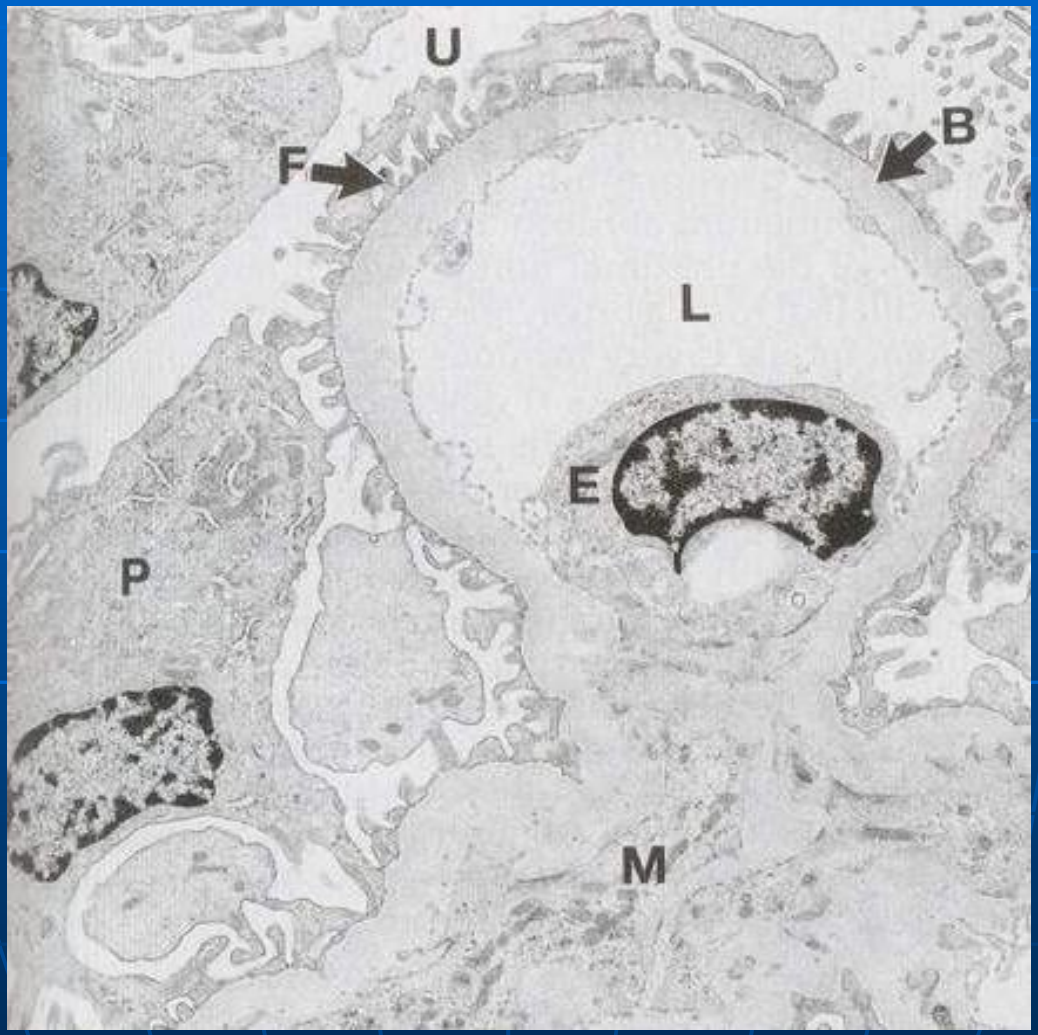


Figure 23.11 The Juxtaglomerular Apparatus.

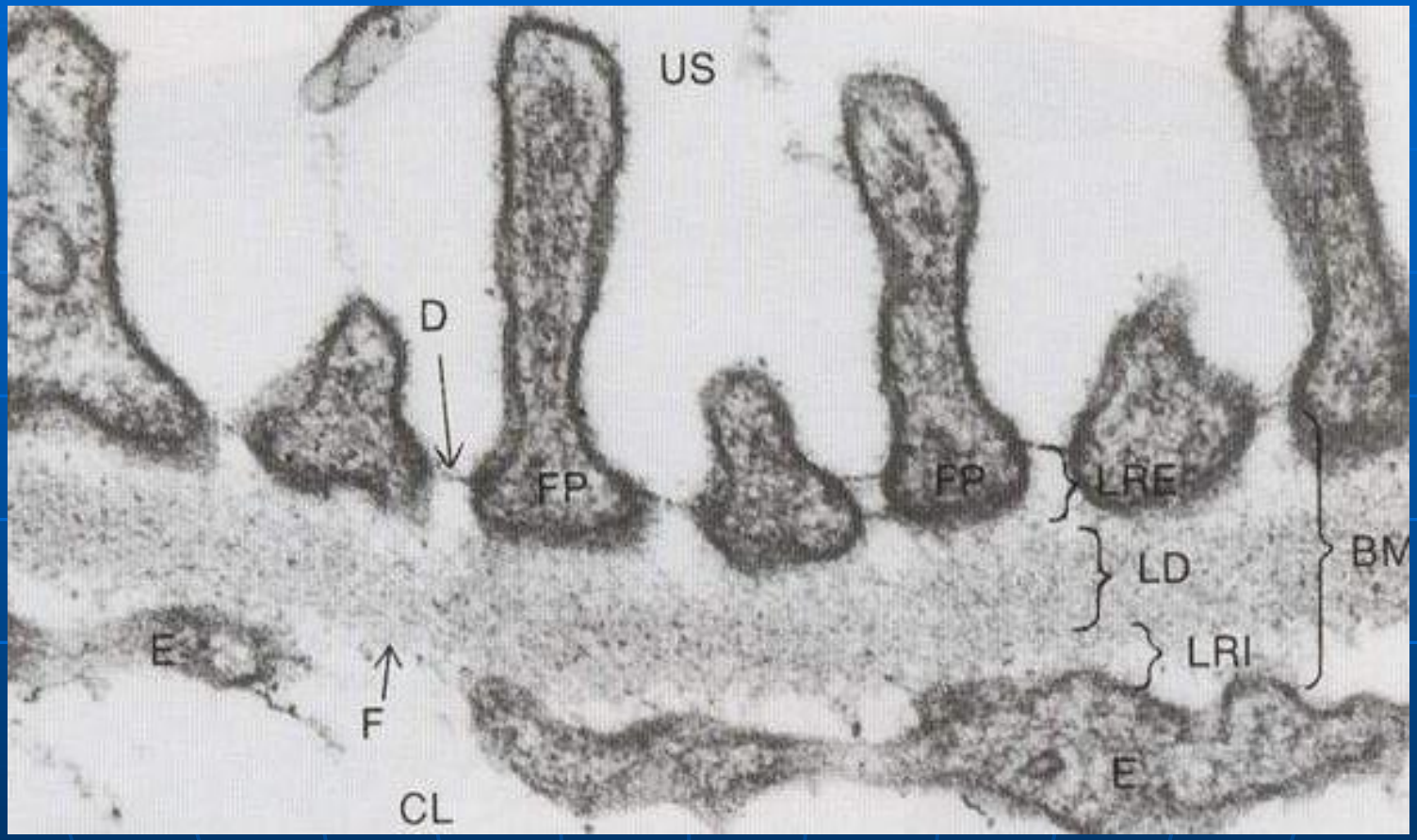


23.13 The Renin-Angiotensin-Aldosterone Mechanism. This chain of events is activated by a drop in blood pressure and acts to raise

Normal Glomerulus



The structure of Glomerular filter

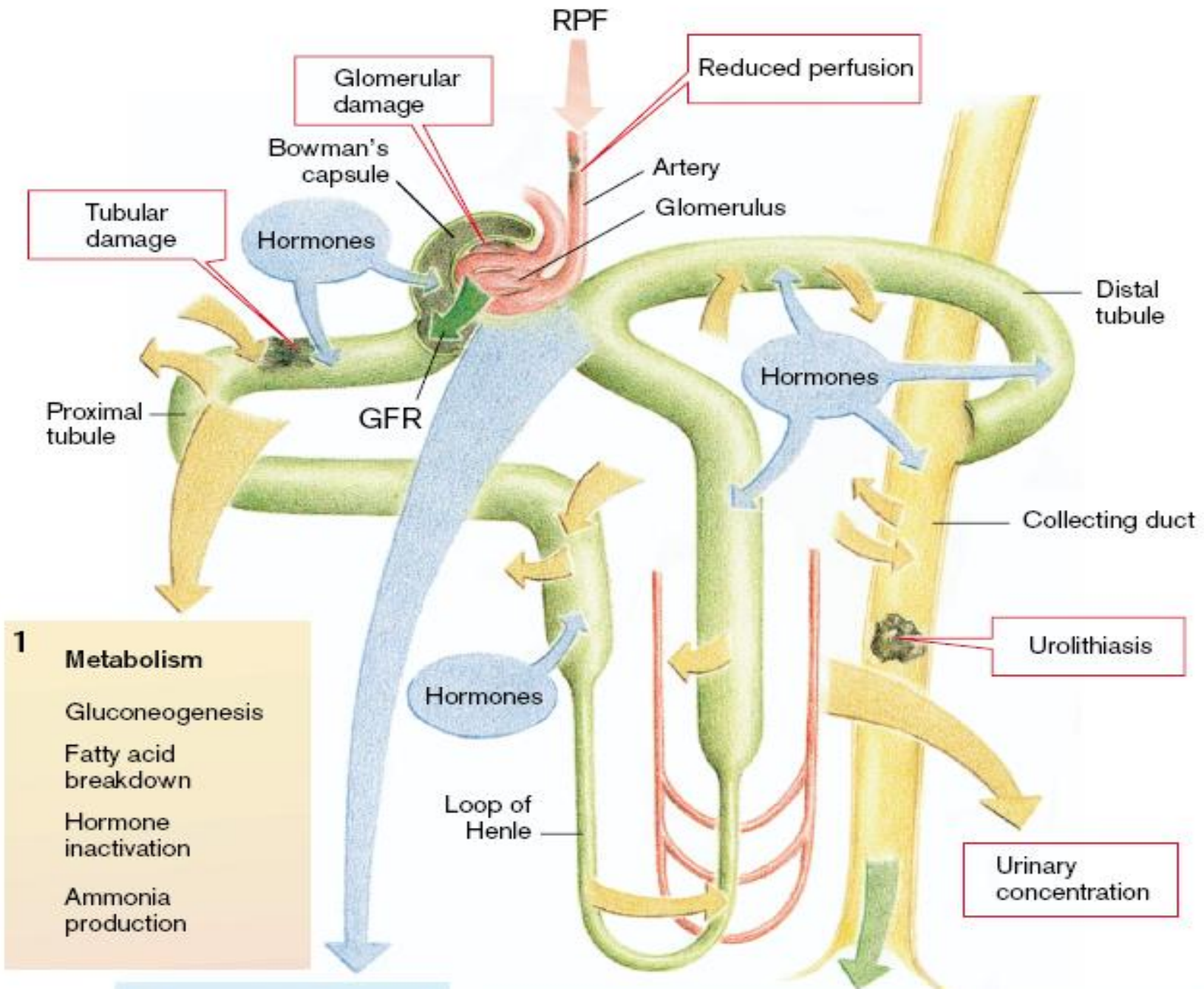


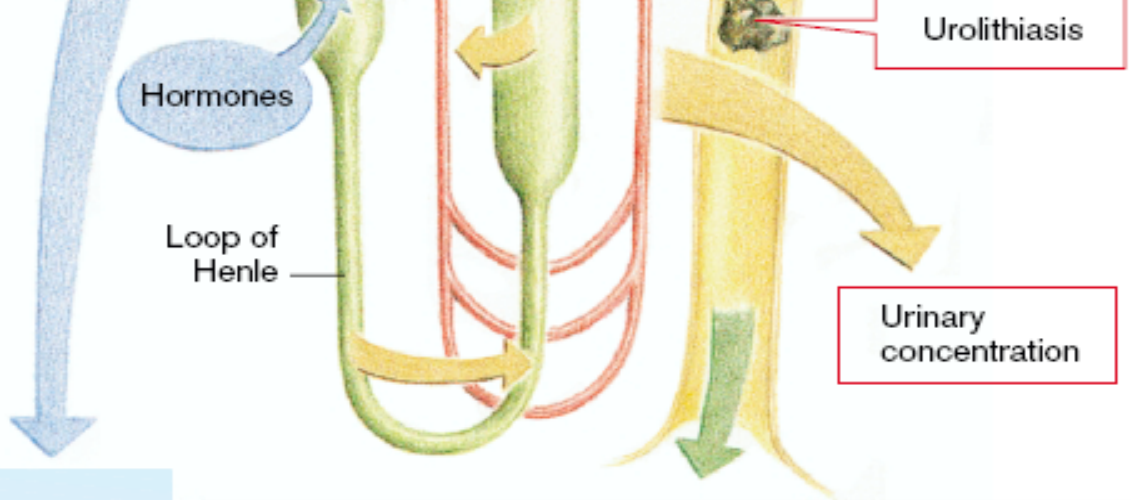
CL: cpillary lumen, **US:** urinary space, **F:** fenestration of the endothelial cells **(E),**
BM: basement membrane, **LRI:** lamina rara interna, **LD:** lamina densa,
D: the slit pore diaphragm, **FP:** podocyte foot processes

KIDNEY

I. Congenital / developmental disorders
II. Renal calculi
III. Urinary obstruction
IV. Glomerular disease
V. Pyelonephritis (tubulointerstitial nephritis)
VI. Hypertension
VII. Acute renal failure (ARF)
VIII. Chronic renal failure (CRF)
IX. Neoplasms
X. Renal manifestations of systemic disease

A. Pathophysiology of the Kidney (Overview)





1 Metabolism

- Gluconeogenesis
- Fatty acid breakdown
- Hormone inactivation
- Ammonia production

2 Hormone release

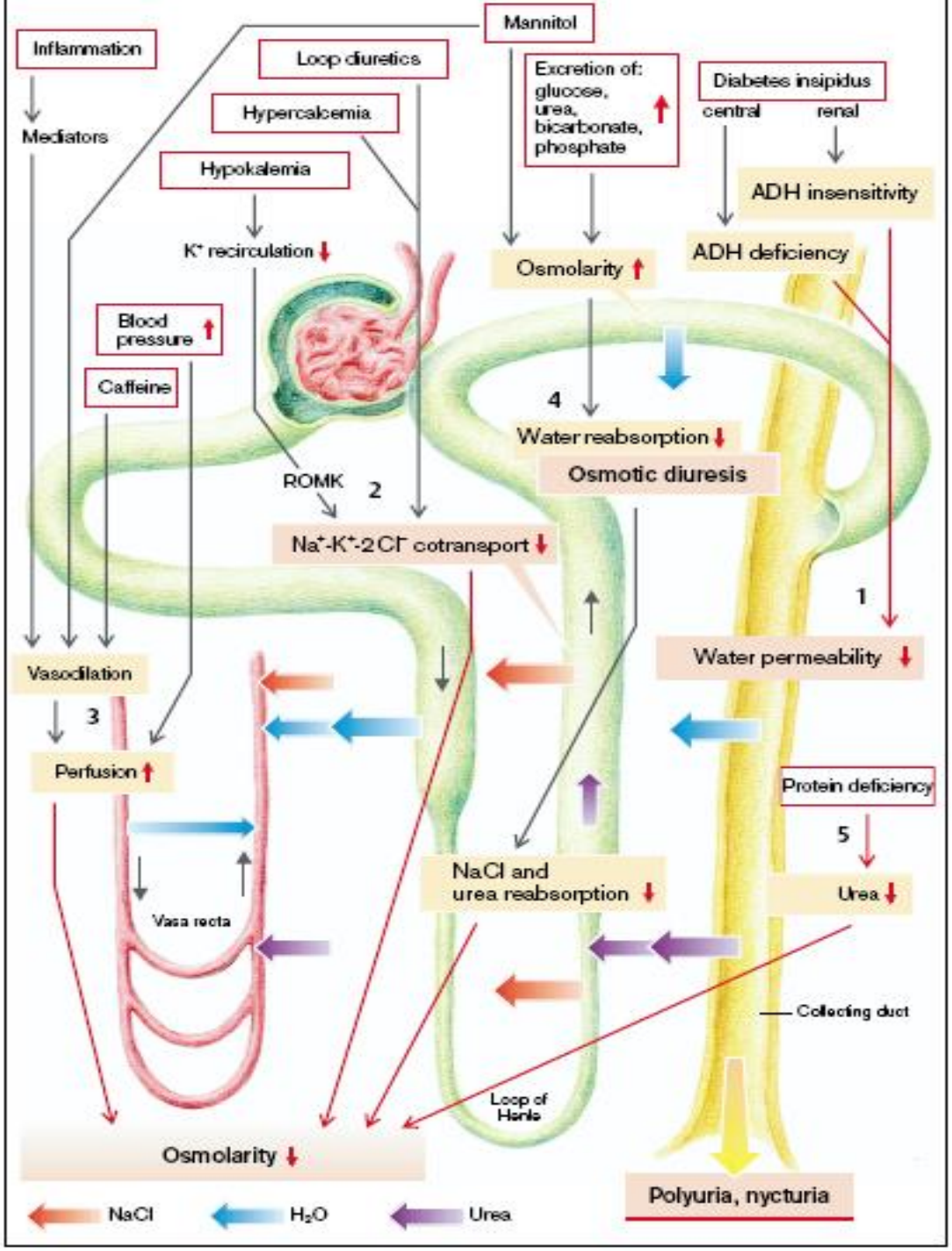
- Erythropoietin
- Calcitriol
- Renin, angiotensin
- Kinins
- Prostaglandins

3 Excretion

Regulation	Elimination: Loss of useful substances:	Elimination: Retention of useless or harmful substances:
H_2O	Glucose	Uric acid
K^+	Amino acids	Urea
Na^+/Cl^-	Proteins	Creatinine
H^+/HCO_3^-		VnO_4
Ca^{2+}/HPO_4^{2-}		Xenobiotics
Mg^{2+}		Uremia toxins

Erythropoiesis | **Water, electrolyte and mineral balance** | **Blood pressure**

Abnormalities of Urinary Concentration



I. Congenital/developmental disorders

A. Renal agenesis

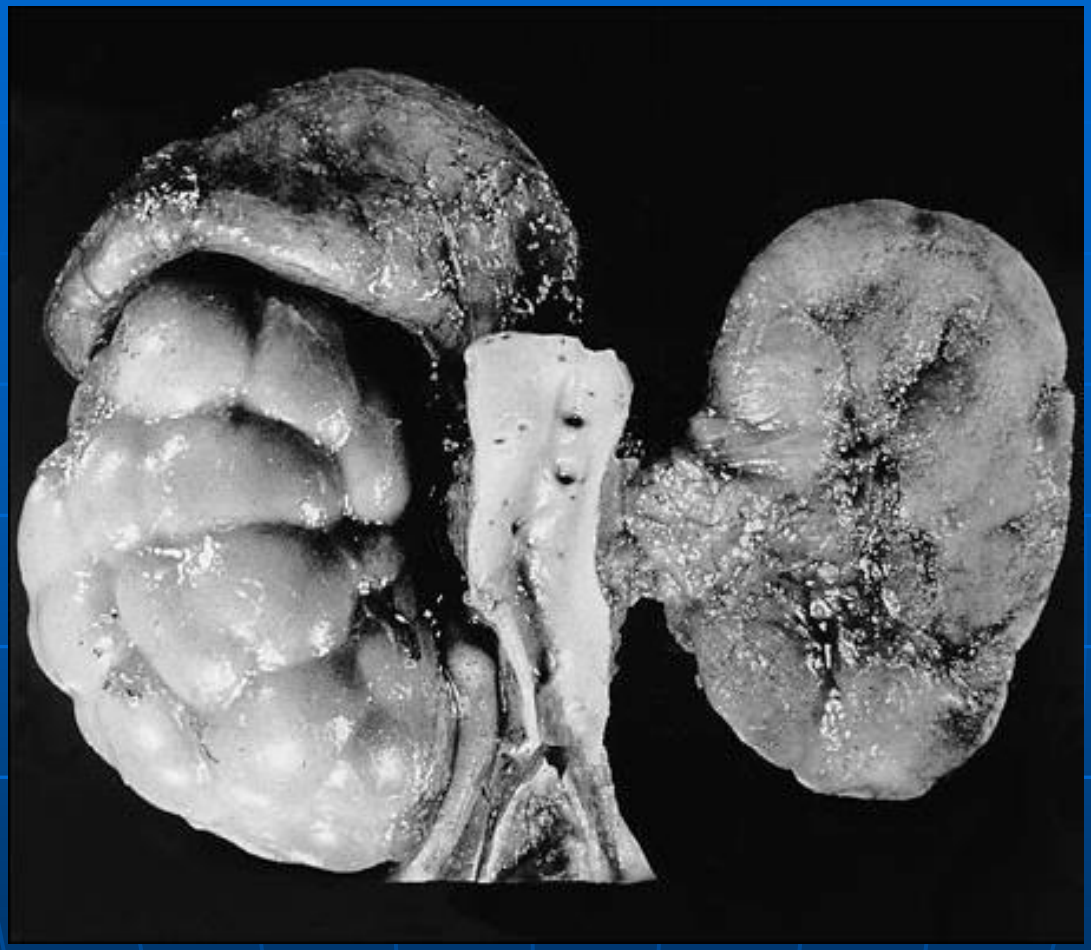
B. Pelvic kidney

C. Horseshoe kidney

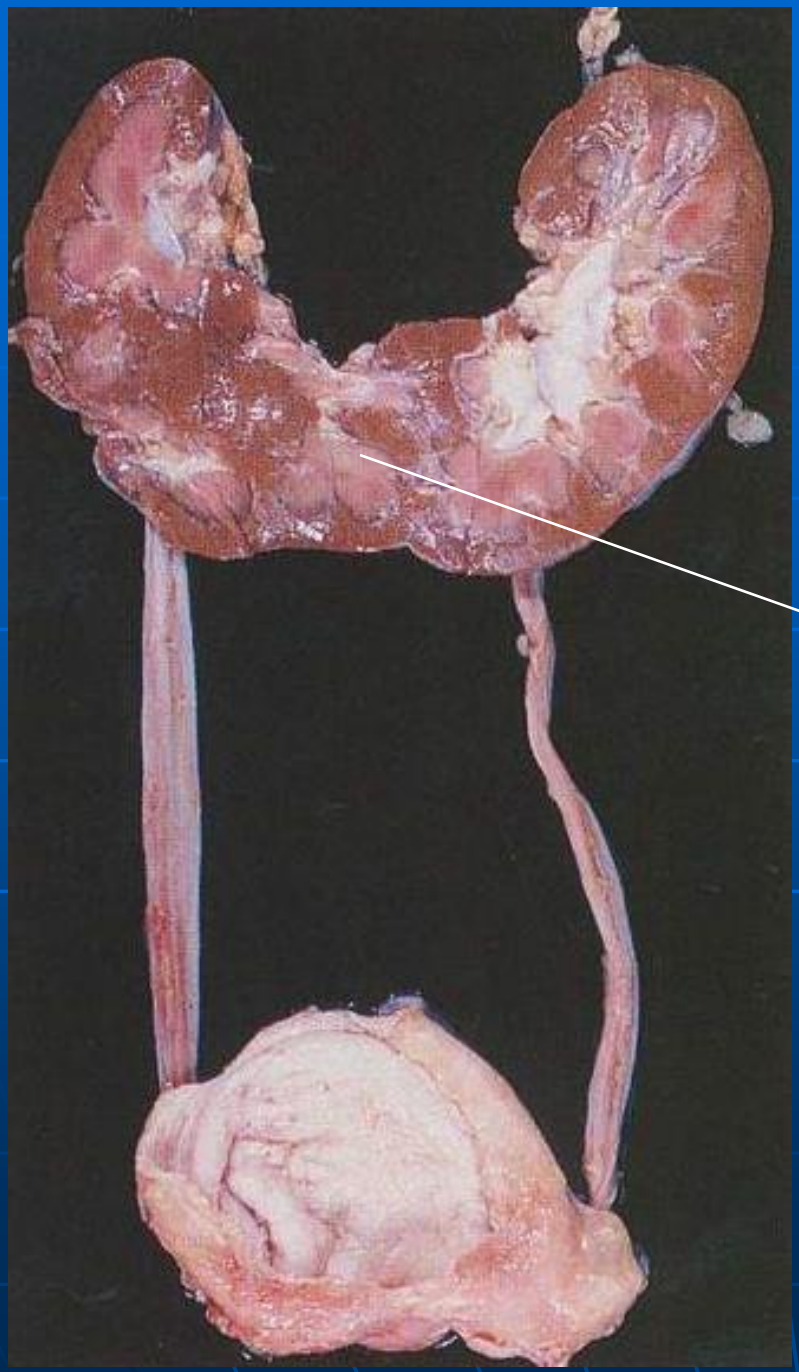
D. Cystic disease

1. Childhood polycystic kidney (Potter's type I)
2. Dysplastic kidney (Potter's type II)
3. Adult polycystic kidney (Potter's type III)
4. Medullary sponge kidney
5. Uremic-medullary cystic disease

Renal agenesis



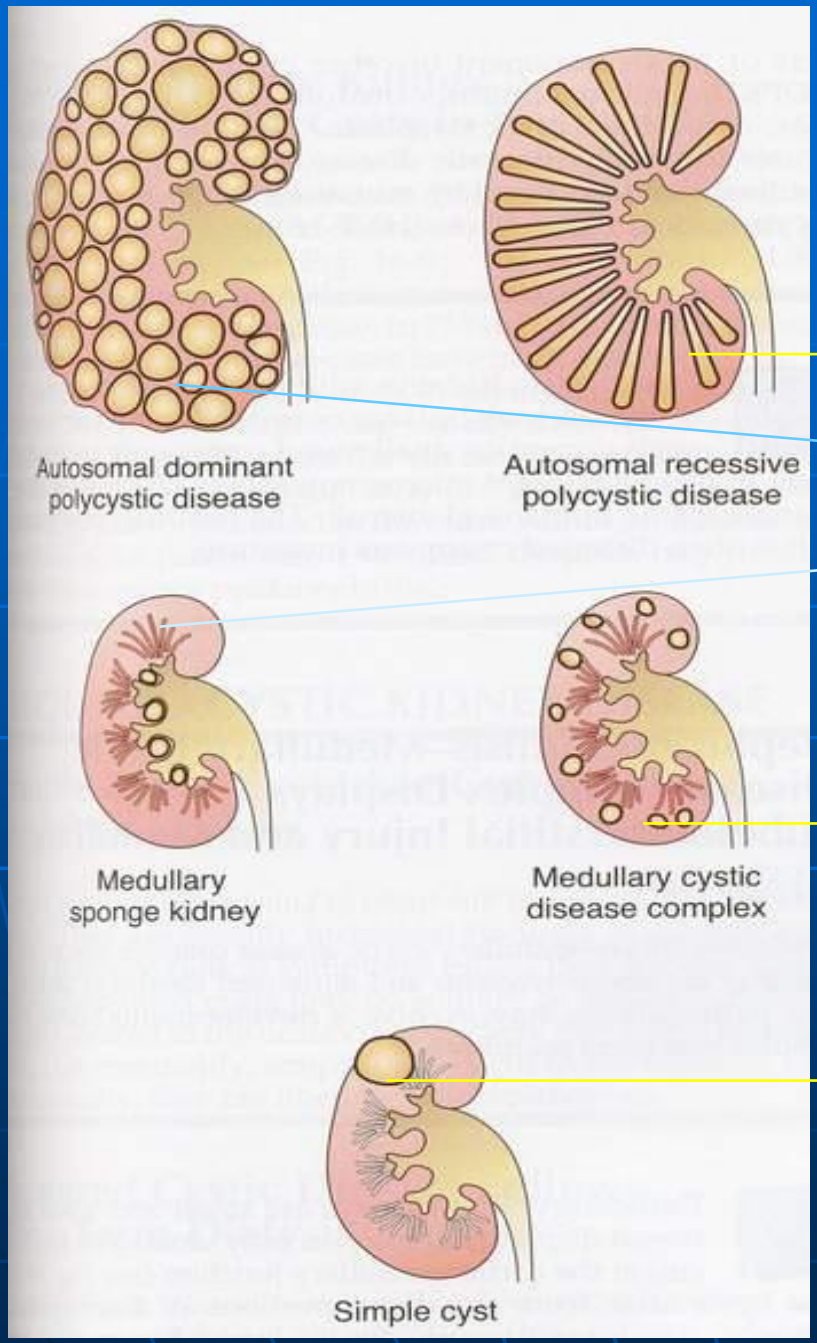
Agenesis is the absence of an organ or part of the body due to absence of its primordium. Example: unilateral left renal agenesis, in which the left adrenal gland assumes a coronal position



Horse-shoe Kidney

The kidneys are fused at the lower pole

Cystic diseases of the kidney



Infantile polycystic disease

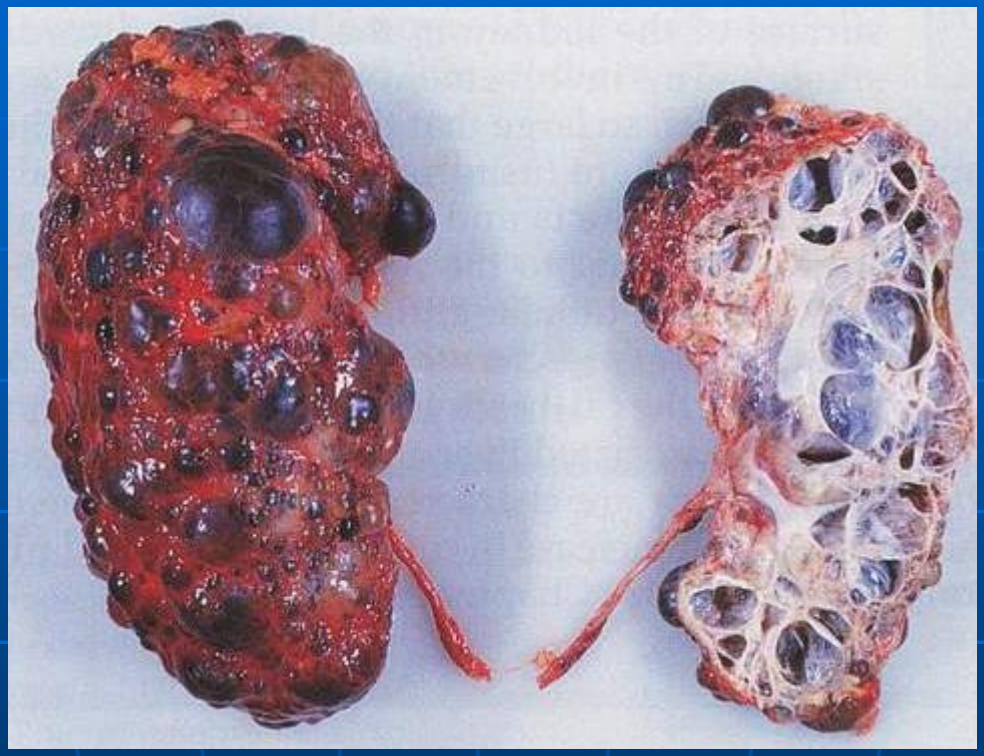
Adult polycystic disease

Is distinguishe by multiple small cyst (less than 5 mm in diameter) in the papillae
Asymptomatic until ages 30-60 y

Multiple cortical and medullary cyst
Formed in patient with end-stage renal disease who are maintained on dialysis (aftr 5 years)

Found incidentally in half persons over 50 years
Usually in the medulla
Lined by flat eipthelium

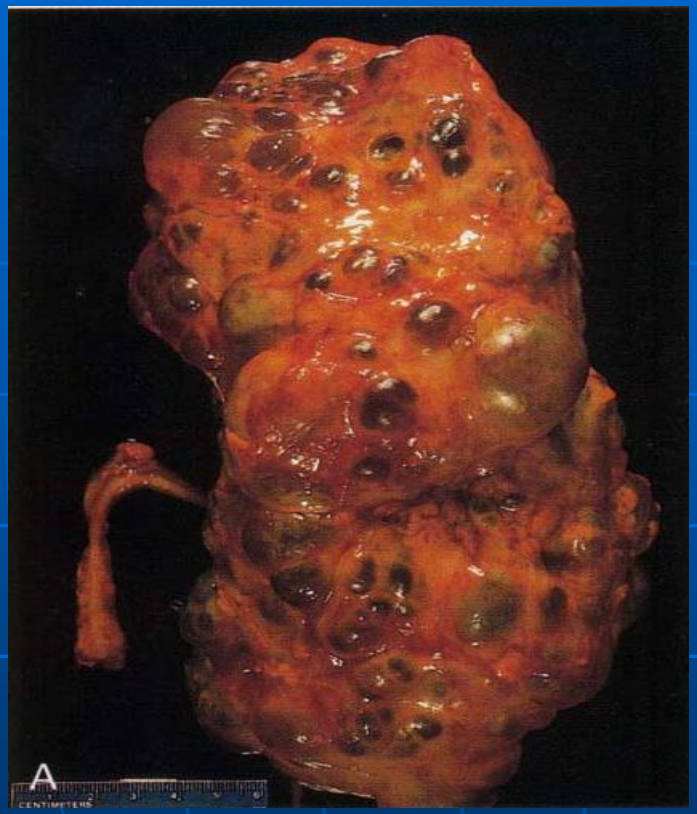
Adult Polycystic Kidney (Potter's type III)



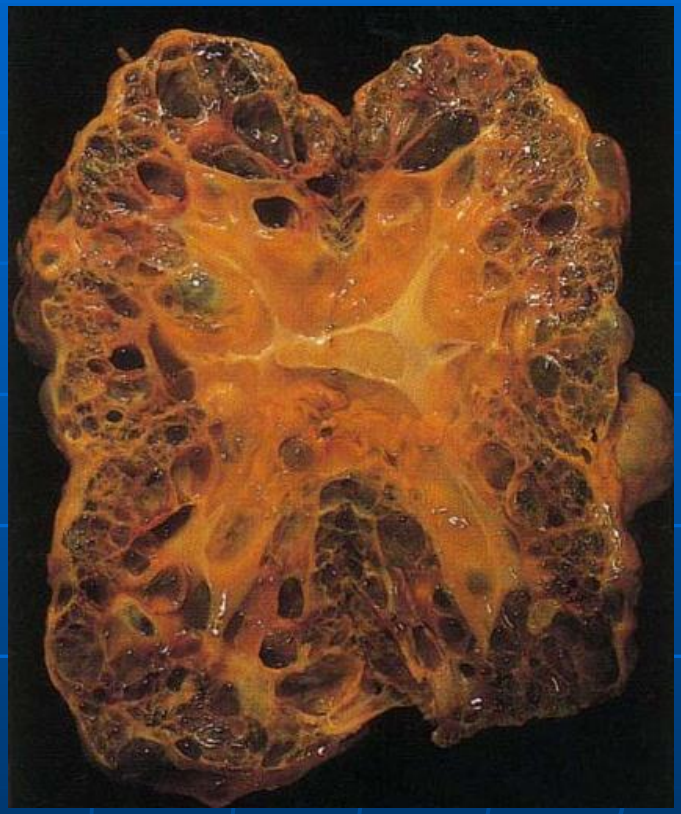
- Most common of a group of congenital diseases, weighing 4500 g
- Autosomal dominant
- Most patient do not develop clinical manifestation until 4th decade
- Half of patient develop end-stage renal failure

Cystic kidney (adult type)

Adult Polycystic Kidney (Potter's type III)



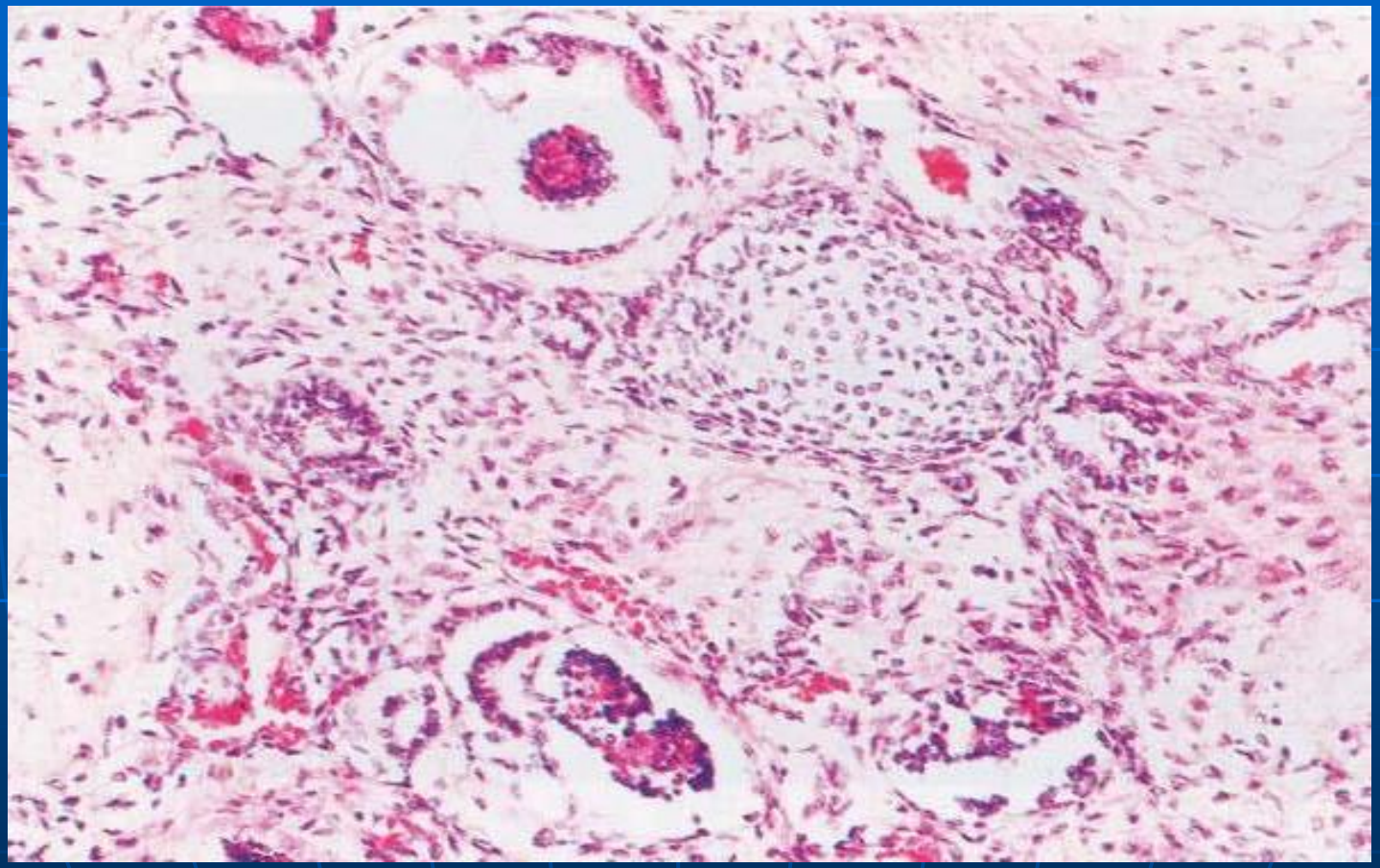
External surface



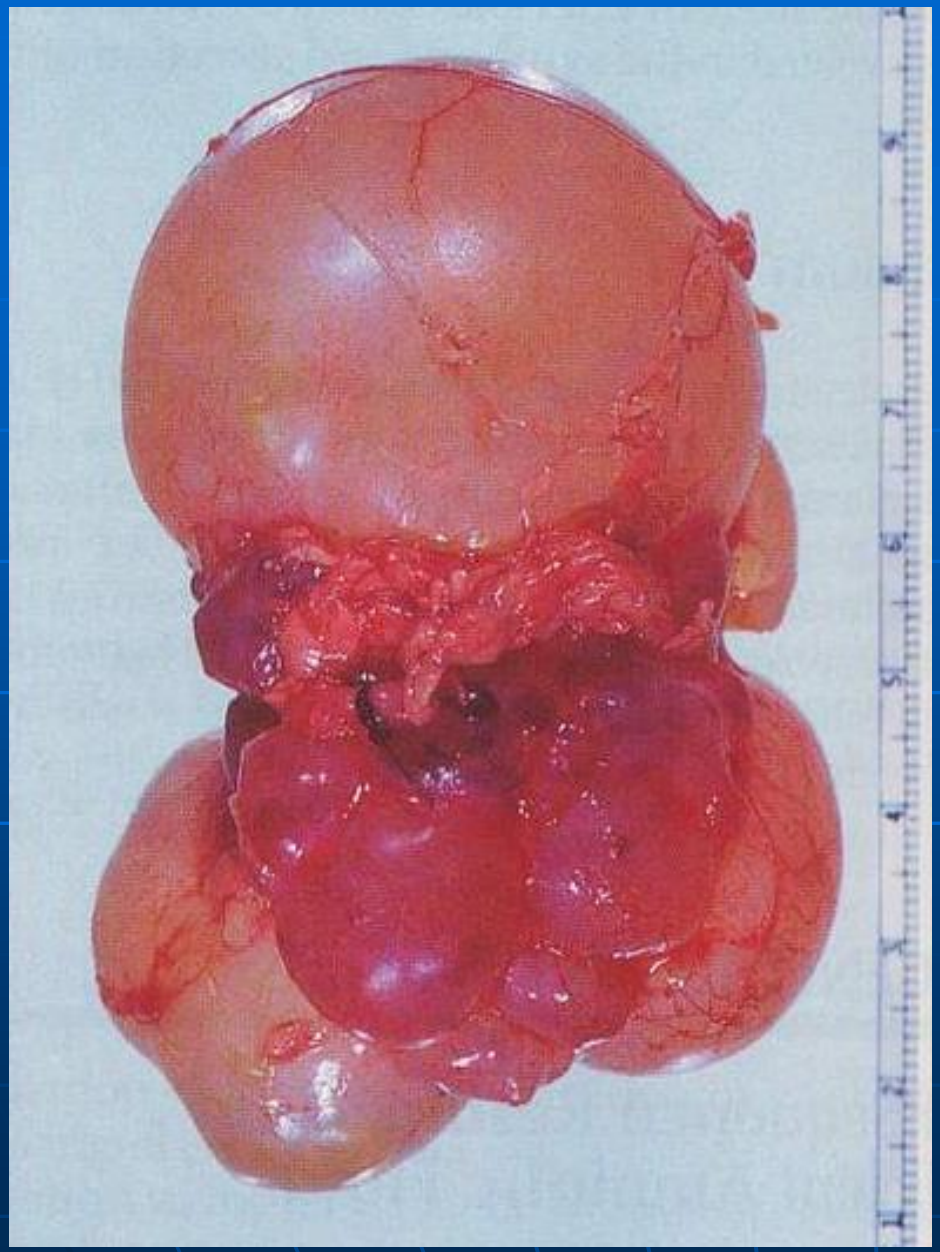
Bisected
The kidney is markedly enlarged with numerous dilated cyst in the parenchyma (note the centimeter rule)

Dysplastic Kidney / Renal Dysplasia

(Potters type II)



Immature glomeruli, tubules, and cartilage are surrounded by loose, undifferentiated mesenchymal tissue



Multicystic Renal Dysplasia
(Potters type II)

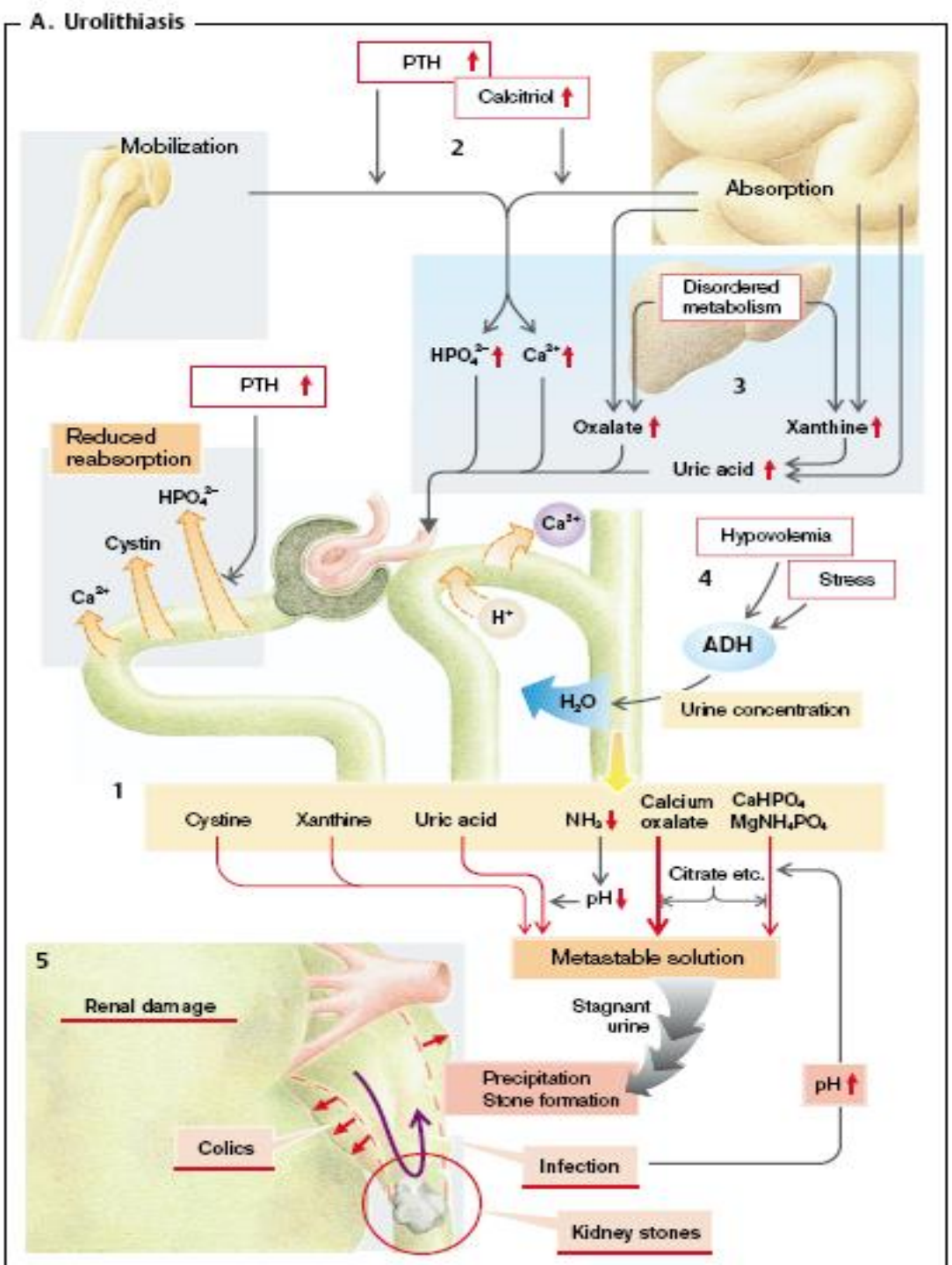
An irregular mass of variably sized cysts does not have a reniform shape

Infantile/childhood polycystic disease

- Autosomal recessive
- Characterized by cystic transformation of collecting duct.
- Rare, and 75 % died in the perinatal period (often becase of pulmonary hypoplasia caused by oligohydramnion → Potter sequence.
- Caused by mutation of PKHD1 gene

II. Renal calculi

- Stone constituent: Ca, oxalates, uric acid, cystine, etc
- Acidic urine facilitate precipitation of uric acid & cystine
- Alkaline → phosphate, may combine with Mg and NH4 (converted from urea by Proteus & other urea-splitting bacteria) → staghorn calculi → calices & pelvis renis
- The majority of stones contain Ca → oxalate, phosphate and hydoxyapatite
- Large stone → asymptomatic, hematuria
- Small stone → ureter colick
- Bacteria & urine stasis → predisposing factor

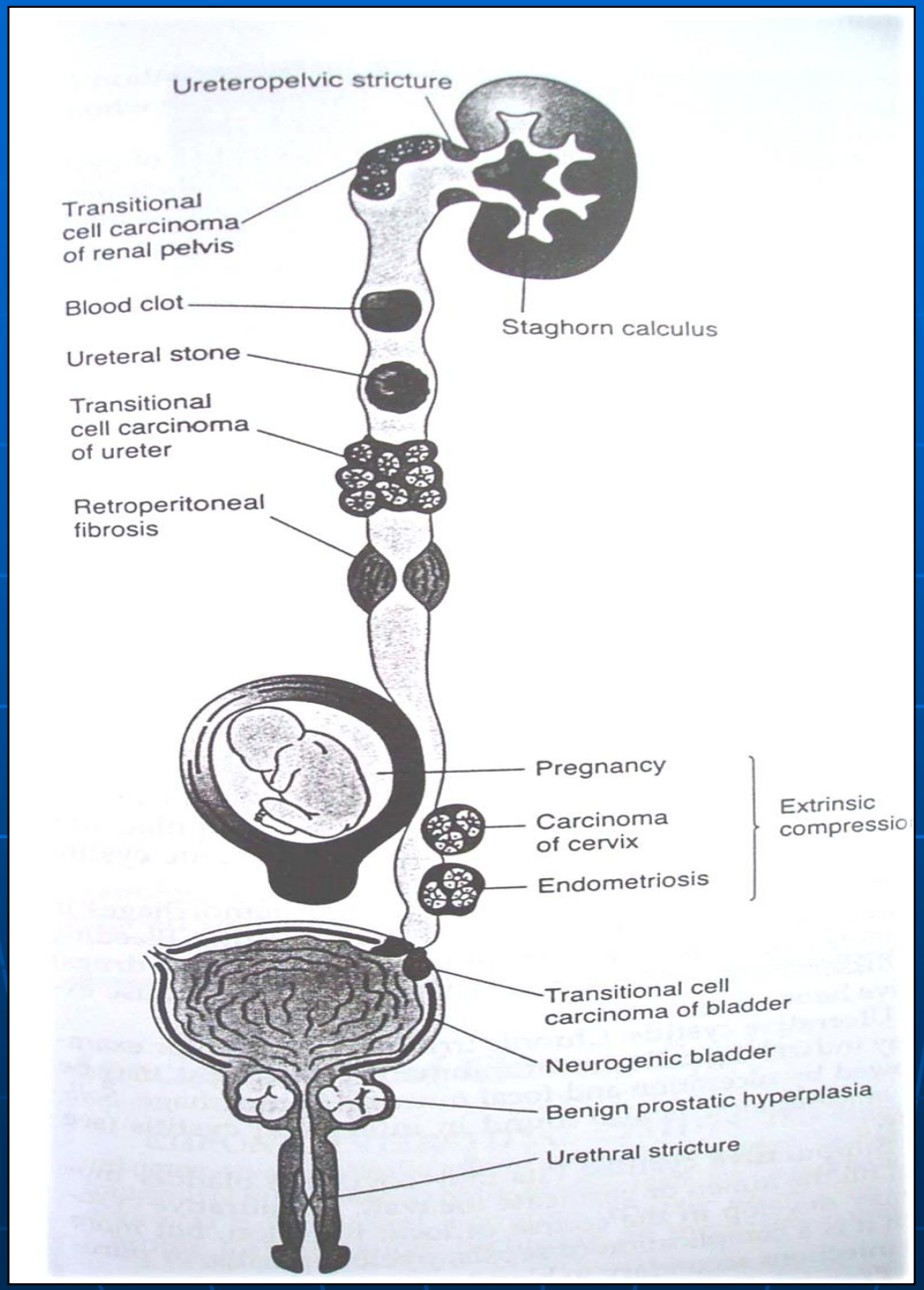

A. Urolithiasis
PTH
Calcitriol
2
Mobilization
Absorption
Disordered metabolism
HPO_4^{2-}
Ca^{2+}
3
PTH
Oxalate
Xanthine
Reduced reabsorption
Uric acid
HPO_4^{2-}
Cystin
Ca^{2+}
Ca^{2+}
H^+
Hypovolemia
4
Stress
ADH
H_2O
Urine concentration
1
Cystine
Xanthine
Uric acid
NH_3
Calcium oxalate
$CaHPO_4$
$MgNH_4PO_4$
Citrate etc.
pH
Metastable solution
5
Renal damage
Stagnant urine
Precipitation
Stone formation
pH
Colics
Infection
Kidney stones

Prevalence of various types of renal stones

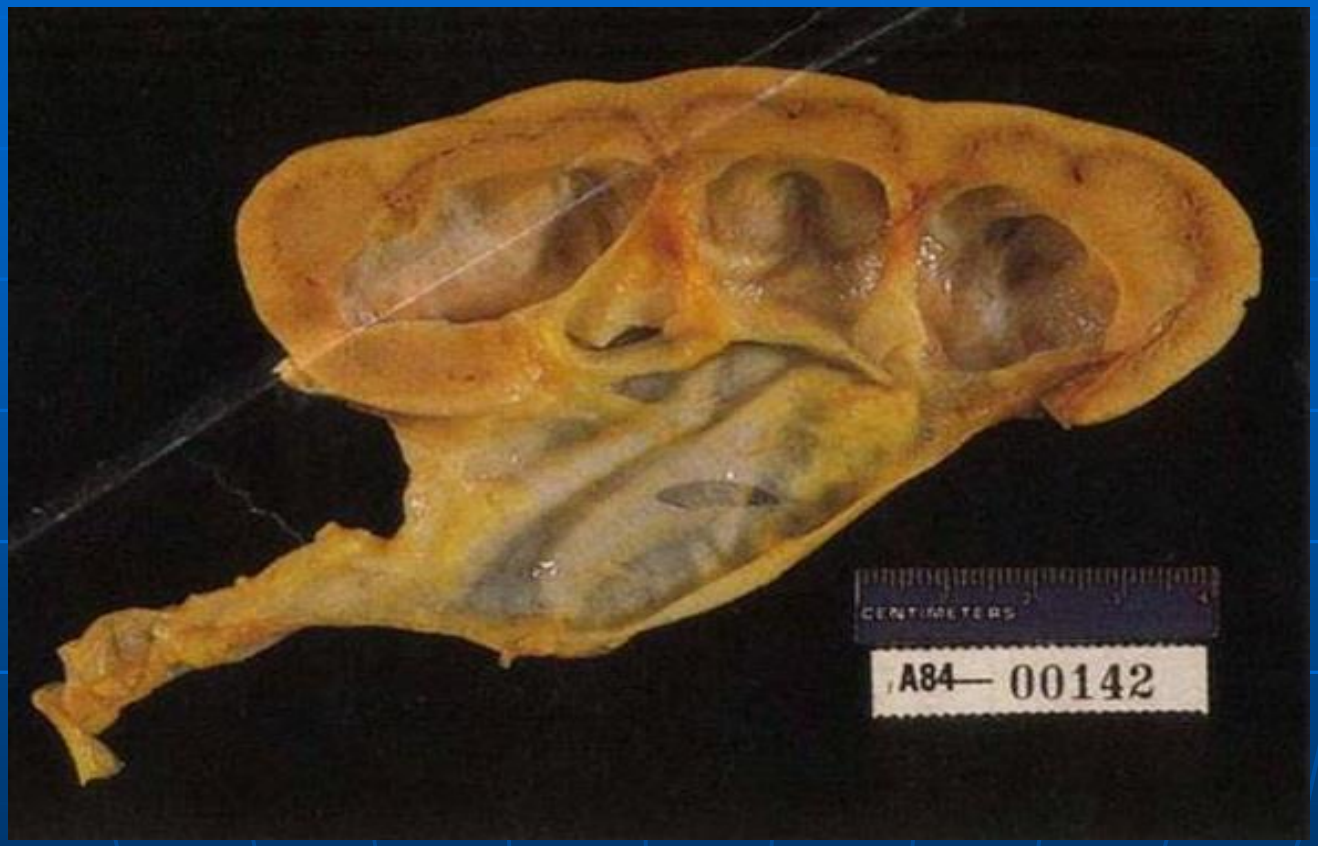
Stone	Percentage
Calcium oxalate / phosphate	75%
Idopathic hypercalciuria (50%)	
Hypercalcemia & hypercalciuria (10%)	
Hyperoxaluria (5%)	
Enteric (4.5%)	
Primary (0.5%)	
Hyperuricosuria (20%)	
No known metabolic abnormality (15-20%)	
Struvite (Mg, Nh_3, Ca, PO_4)	10 - 15%
Renal infection	
Uric acid	6%
Associated with hyperuricemia	
Associated with hyperuricosuria	
Idopathic (50% of uric acid stones)	
Cystine	1-2%
Others or unknown	+/-10%

III. Urinary obstruction

- Etiology: stones, BPH, congenital defects, tumors, functional disorders, pregnancy → predisposing to infection & stone formation → hydronephrosis → progressive atrophy of the kidney
- It may be unilateral or bilateral (→ anuria)
- Renal changes become irreversible after about 3 weeks (several months of partial obstruction)
- Acute obstruction → acute dilatation & stretching of renal capsule → pain
- Gradual obstruction → may be asymptomatic
- Hypertension may ensue


Ureteropelvic stricture
Transitional cell carcinoma of renal pelvis
Blood clot
Staghorn calculus
Ureteral stone
Transitional cell carcinoma of ureter
Retroperitoneal fibrosis
Pregnancy
Carcinoma of cervix
Endometriosis
Extrinsic compressio
Transitional cell carcinoma of bladder
Neurogenic bladder
Benign prostatic hyperplasia
Urethral stricture

Hydronephrosis



- Marked dilatation of the pelvis and calyces and thinning of renal parenchyma