

INTEGRATED RENAL-ELECTROLYTES

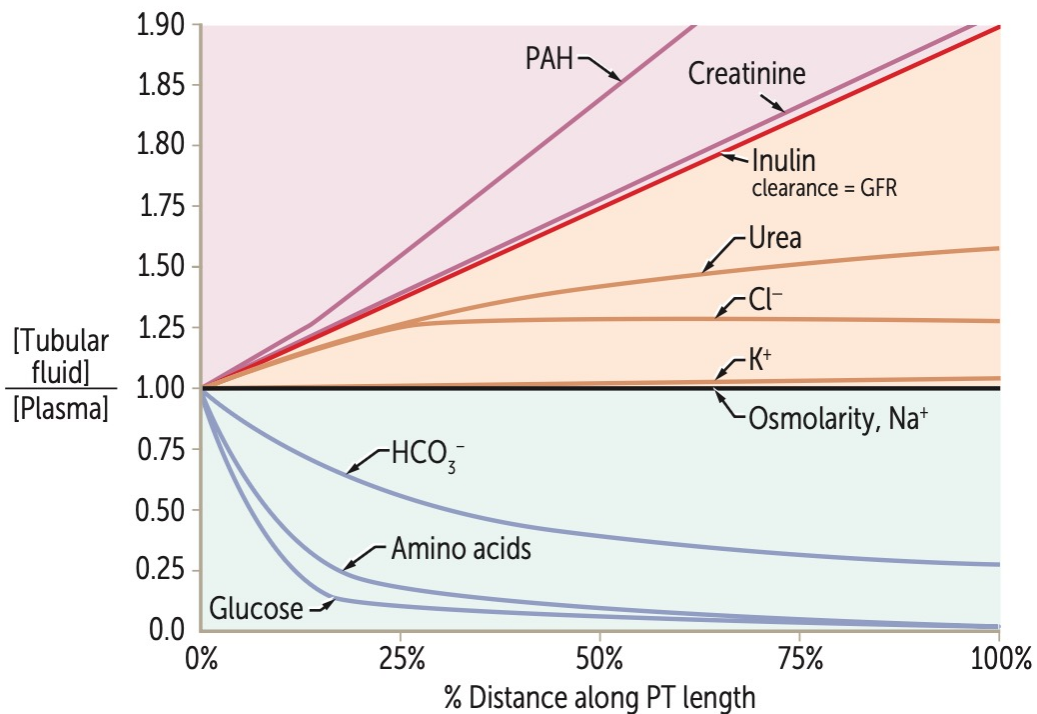
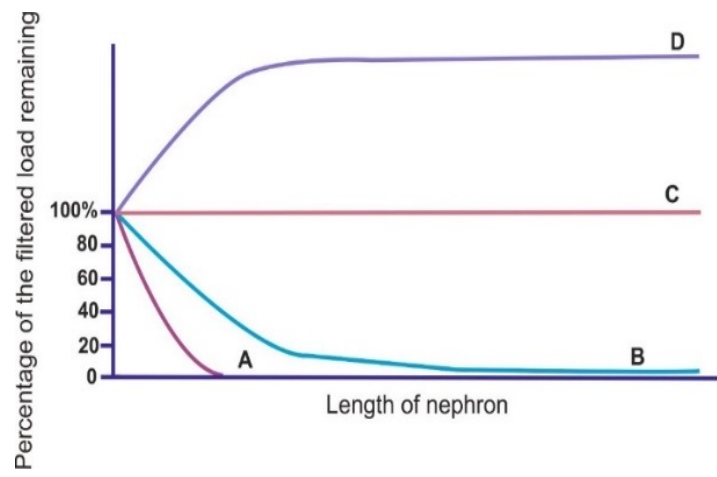
Renal Physiology

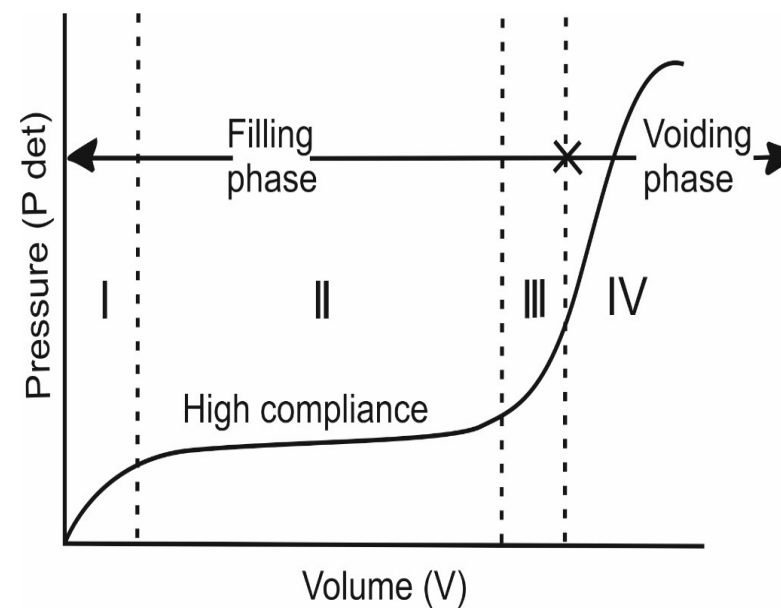
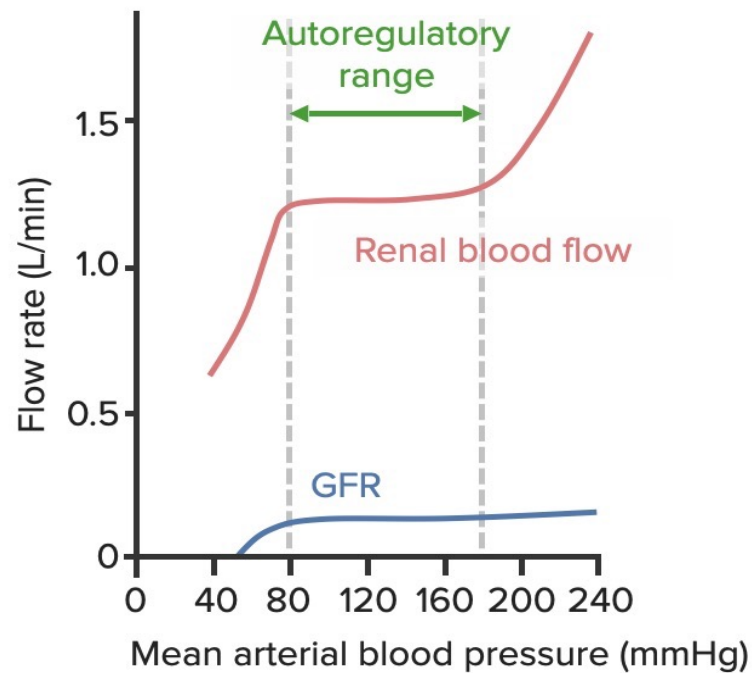
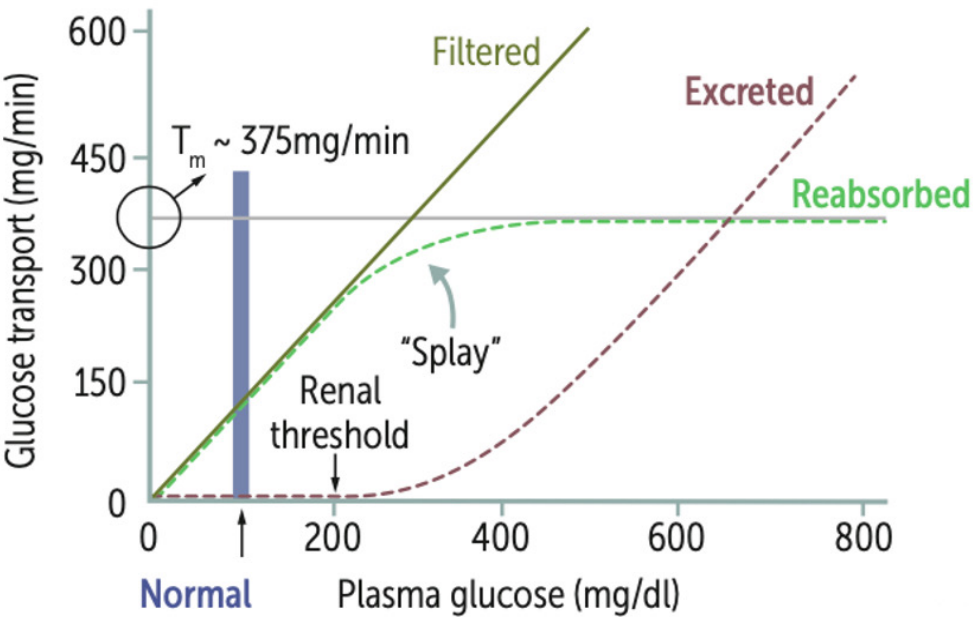
Clearance=
GFR=
RPF=
Filtration fraction=
Clearance: PAH > Creatinine > Inulin > Urea > Na > Glucose

Counter-current :
CC multiplier
CC exchanger:

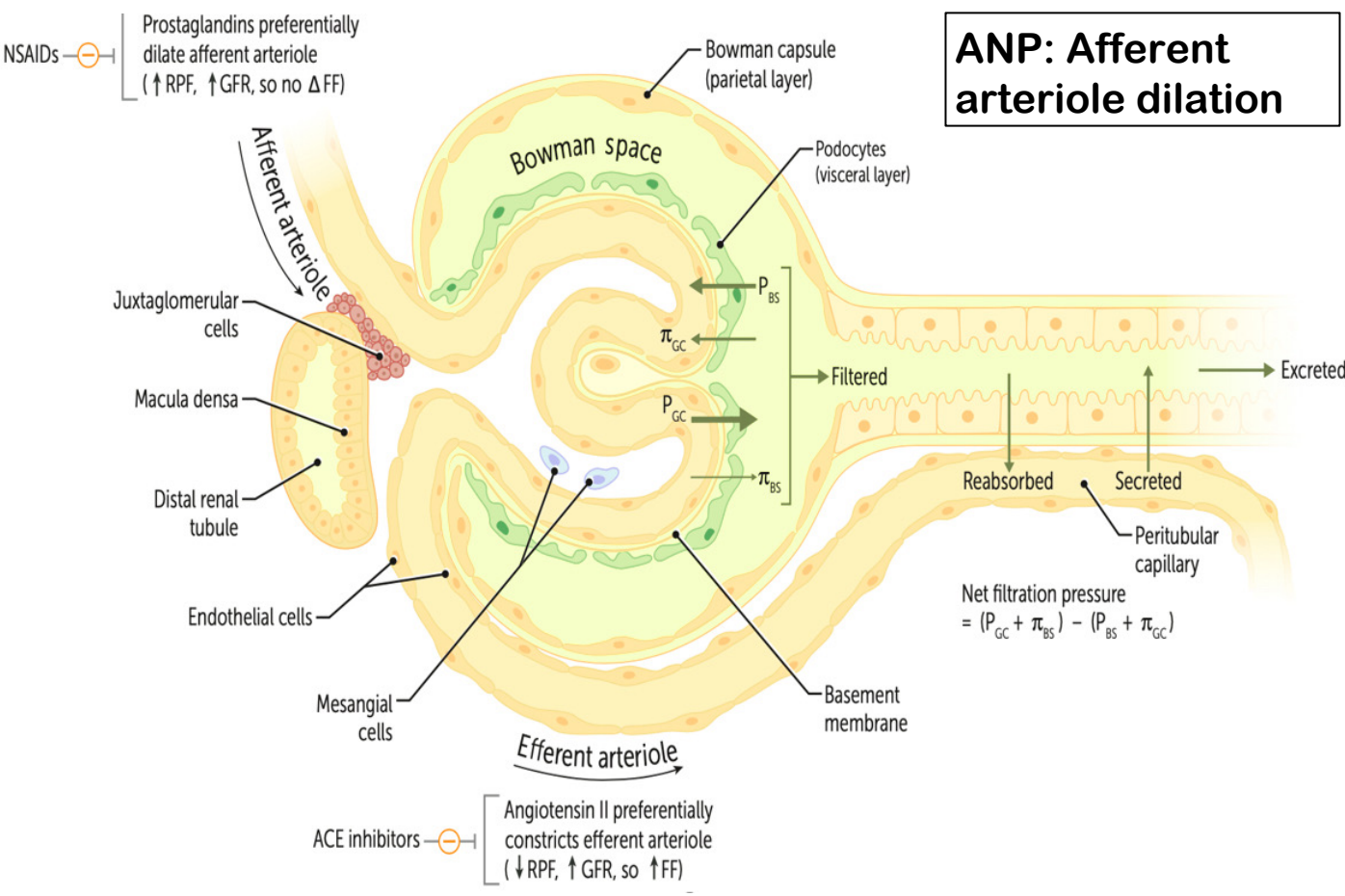
Macula Densa

Juxtaglomerular cells





Glomerular Filtration

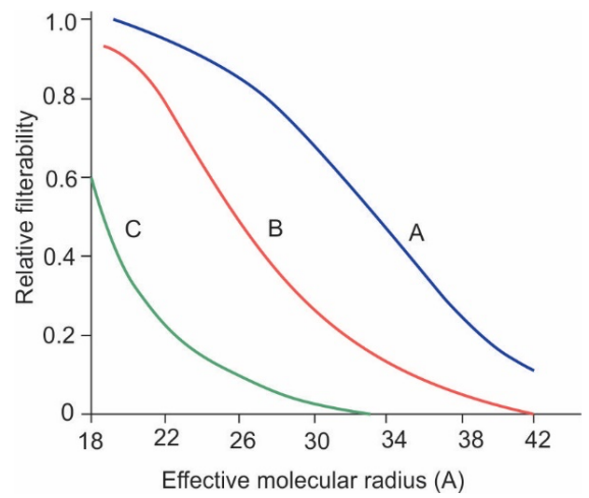


	<u>GFR</u>	<u>RPF</u>	<u>FF</u>
Afferent arteriole constriction			
Efferent arteriole constriction			
High plasma protein concentration			
Low plasma protein concentration			
Ureter constriction			
Dehydration			

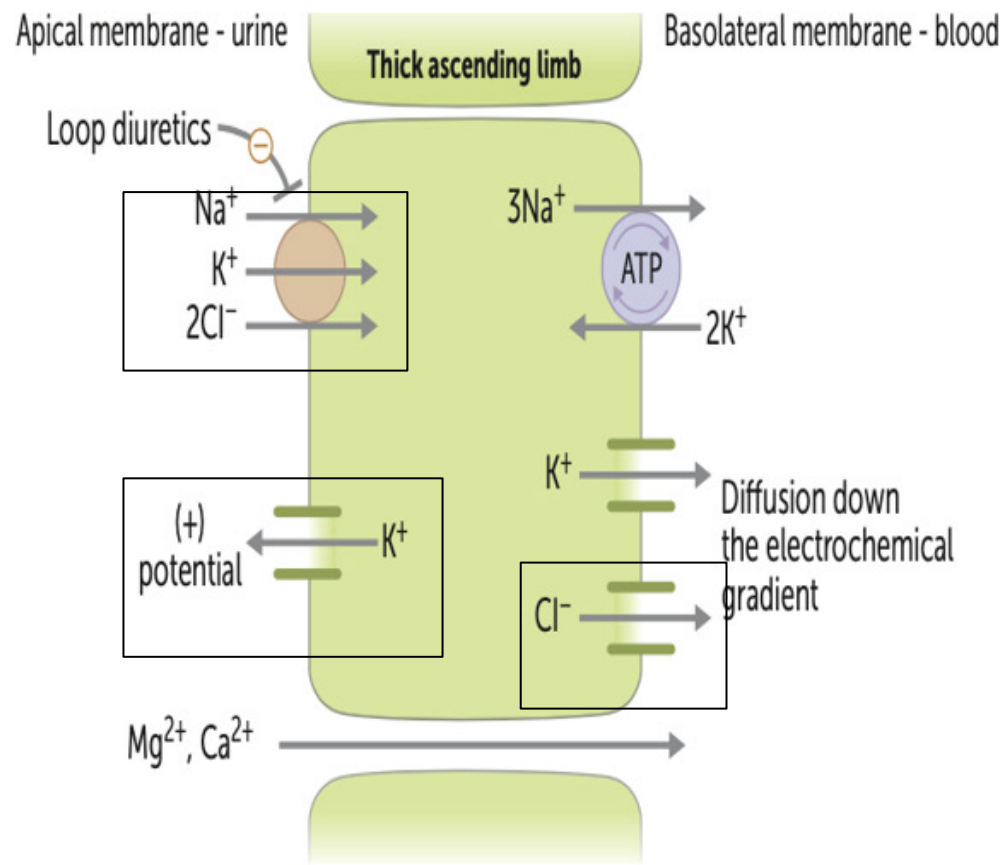
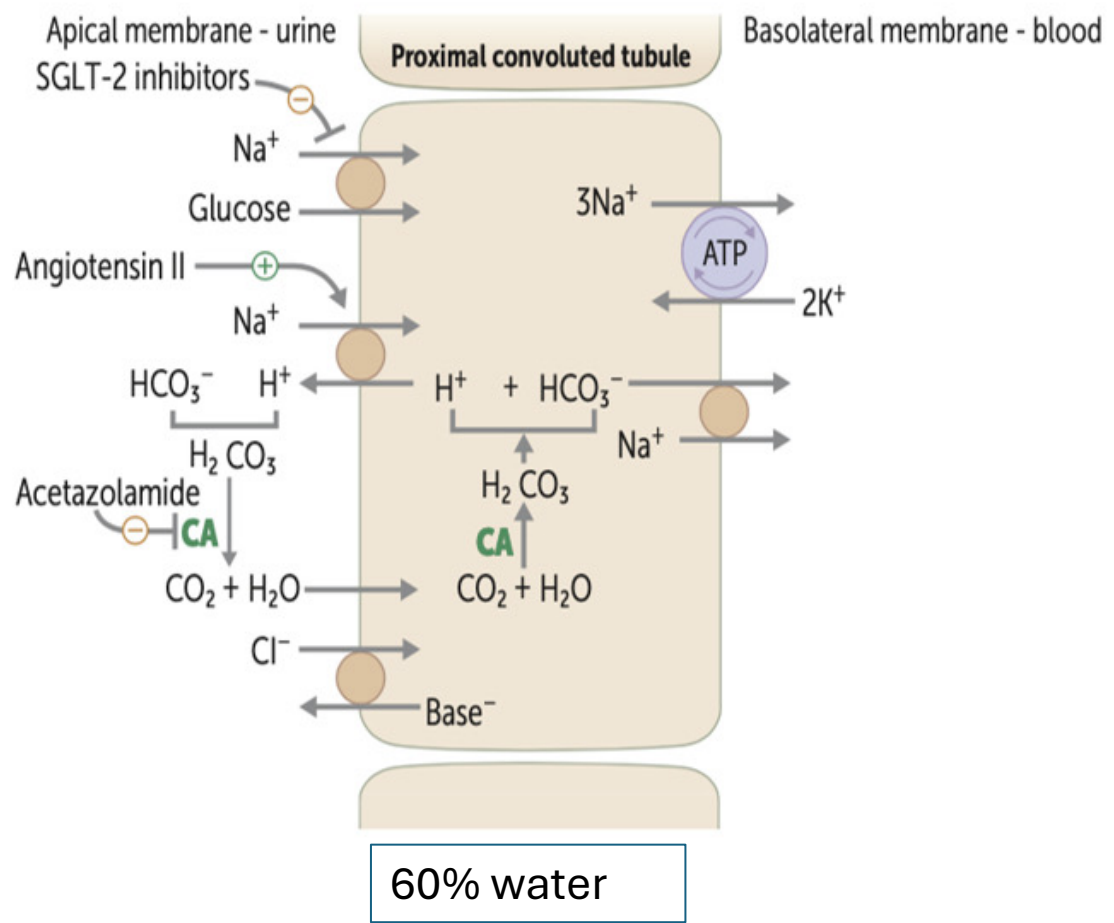
Glomerular filtration barrier:

- **Fenestrated** capillary endothelium: 50-100nm
- Basement membrane with type IV collagen and **heparan sulfate**
- Podocyte **foot processes** Slit diaphragm pore size: ~ 4 nm

Dopamine ANP NO cAMP PGE2

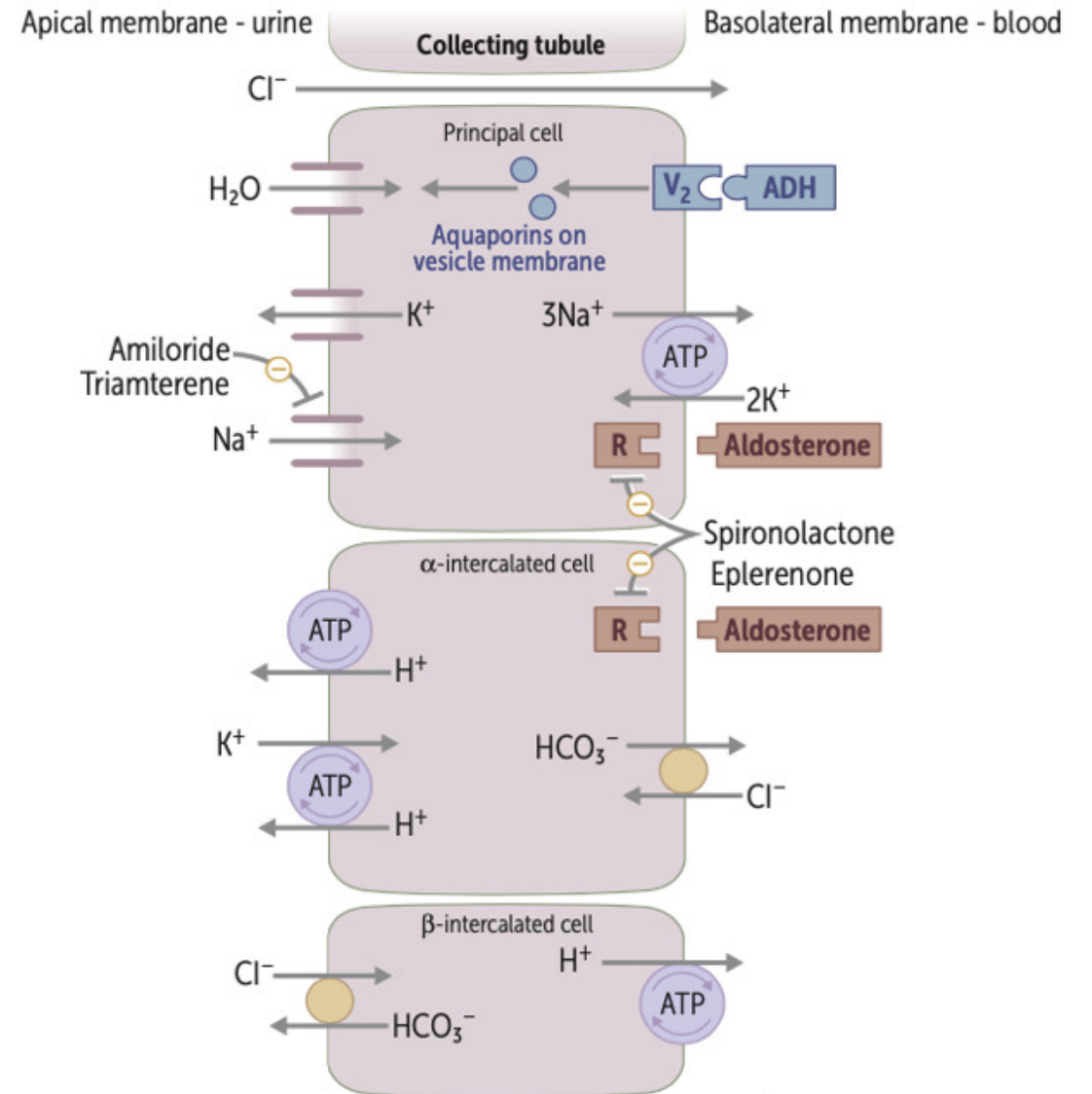
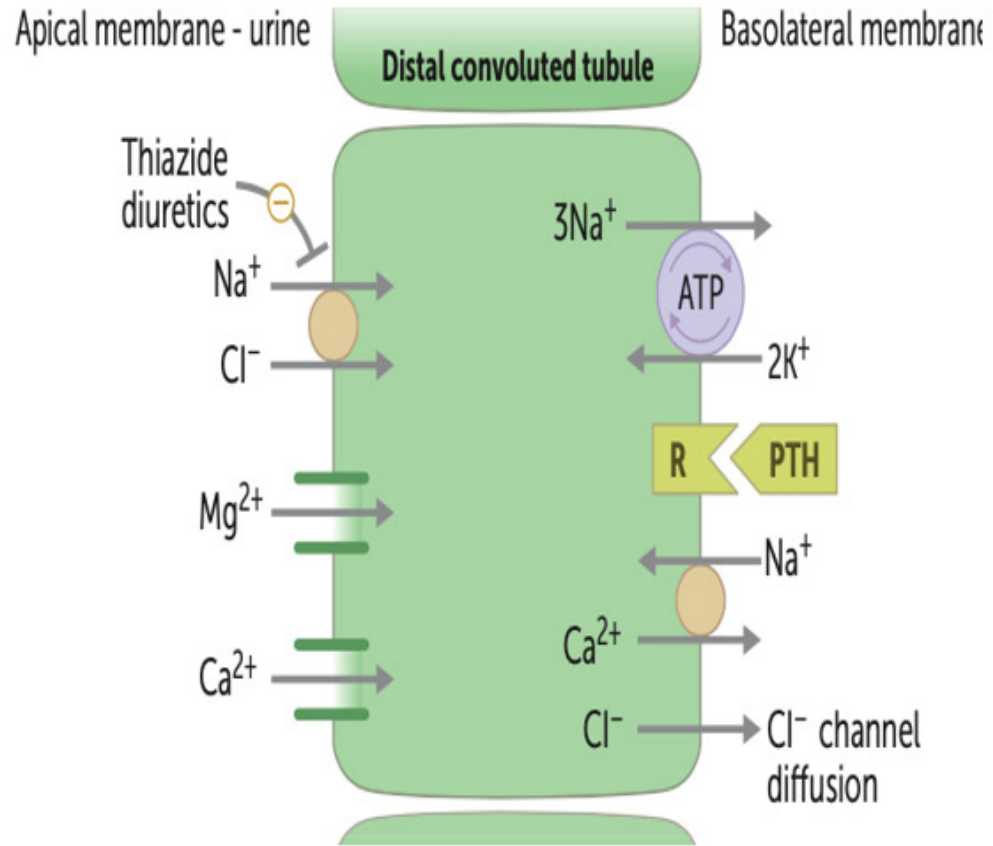


Renal tubules



PTH:
PCT:
DCT:

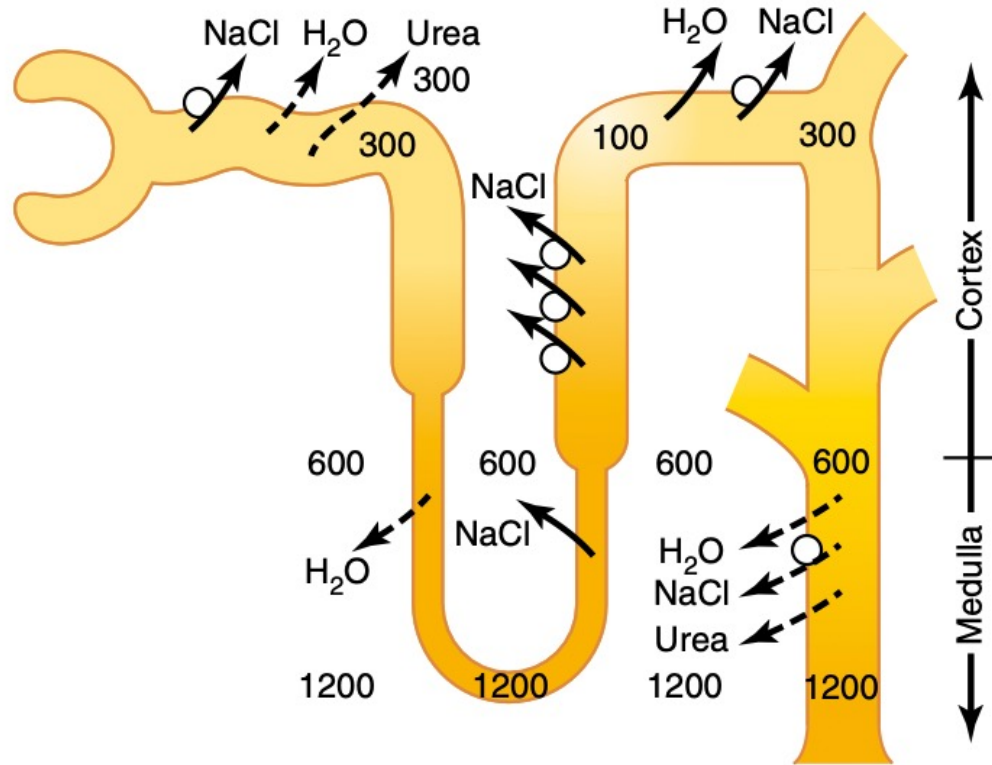
THIN DESCENDING LOH:



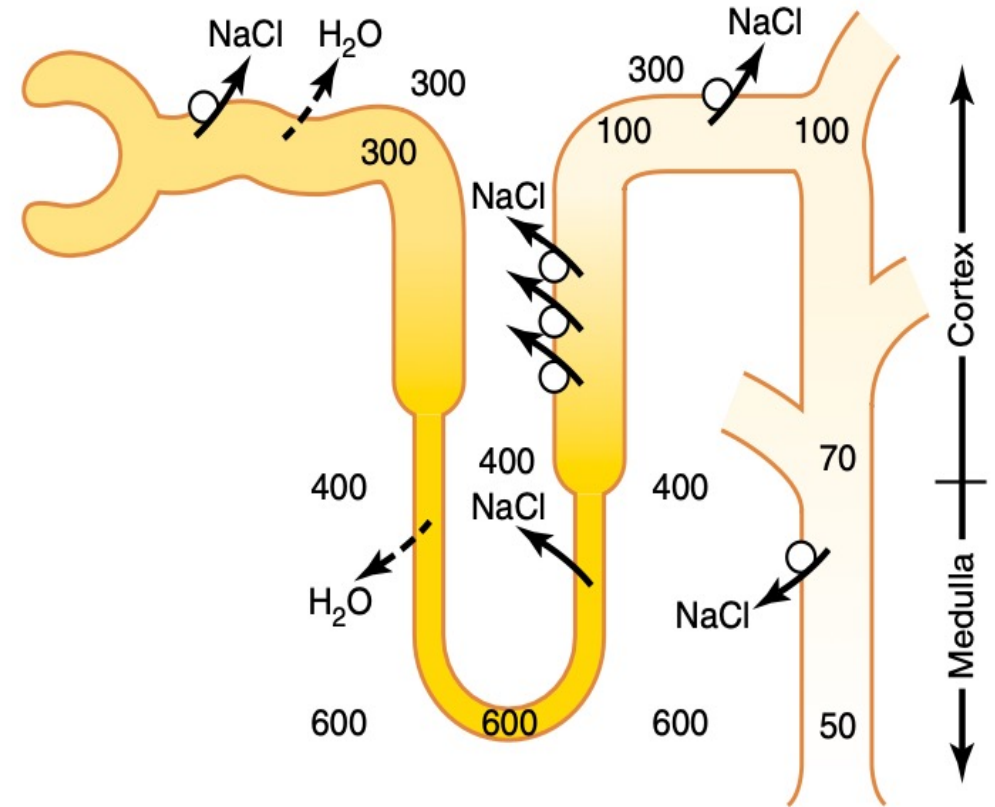
1-2% water absorption: no ADH
10% water absorption: ADH

The story of ADH

ADH normal



Absent ADH



Diuretics Pharmacology

Acetazolamide
S/E:

- Type 2 RTA
- Paresthesias
- NH3 toxicity
- Sulfa allergy
- Hypokalemia
- Calcium phosphate stones (insoluble at high urine pH)

DOC

- mountain sickness
- Familial hypokalemic PP

Thiazide
S/E:

- Metabolic alkalosis
- Hyponatremia
- Hyperglycemia
- Hyperlipidemia
- Hyperuricemia
- Hypercalcemia
- Sulfa allergy

Chlorthalidone
Chlorthiazide
Indapamide
Metolazone

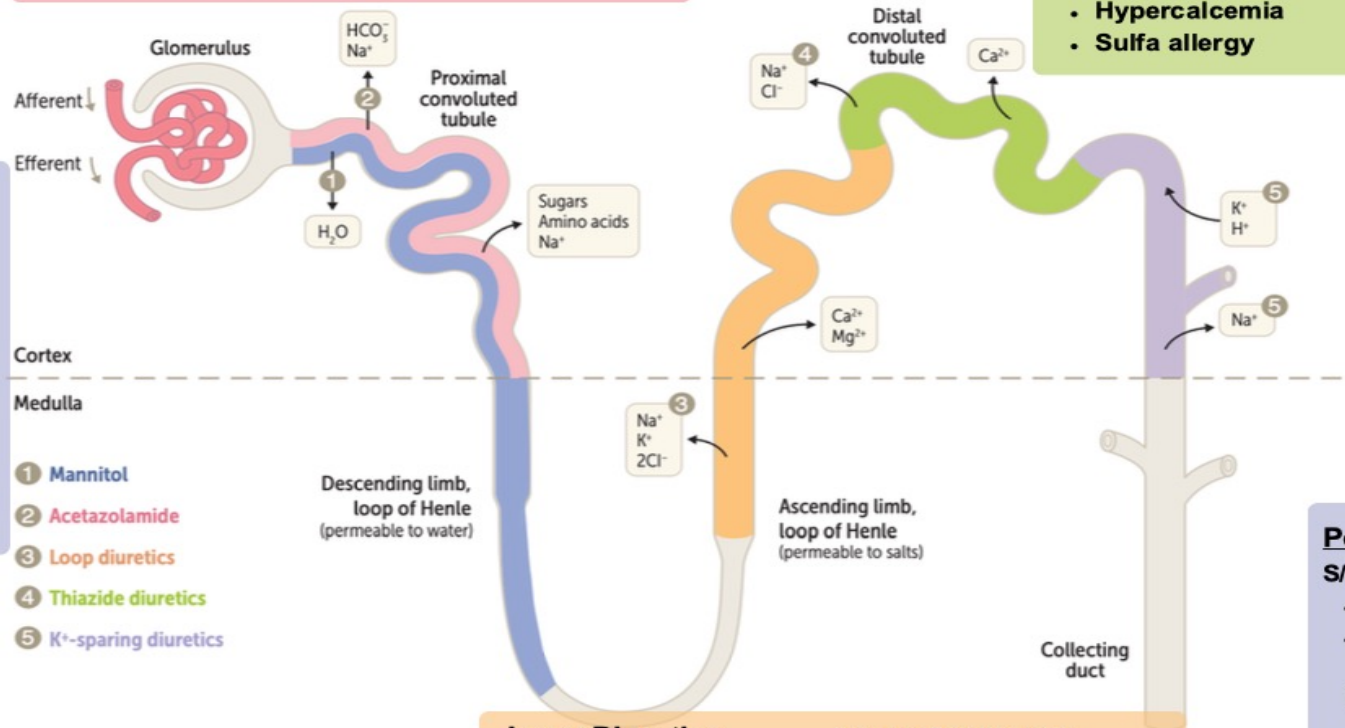
DOC:
 ACG, DDS
 Cerebral edema

Mannitol

S/E:

- Dehydration
- Hypo- or hypernatremia
- Pulmonary edema

C/I: Anuria, HF



Loop Diuretics
S/E:

- Ototoxicity
- Dehydration
- Allergy
- Sulfa allergy
- Metabolic Alkalosis
- Interstitial Nephritis
- Gout

Loops: Cardiogenic p.edema

Torsemide:
Bumetanide:
Ethacrynic acid:

Potassium Sparing
S/E:

- Hyperkalemia
- Metabolic acidosis

DOC ascites, resistant HtN

AMILORIDE:
 DOC Li induced NDI,
 Liddle, cystic fibrosis

Tubular Disorders

TAL
Na-K-2CL



DCT
Na-cl



CD
ENac



Dx

BP

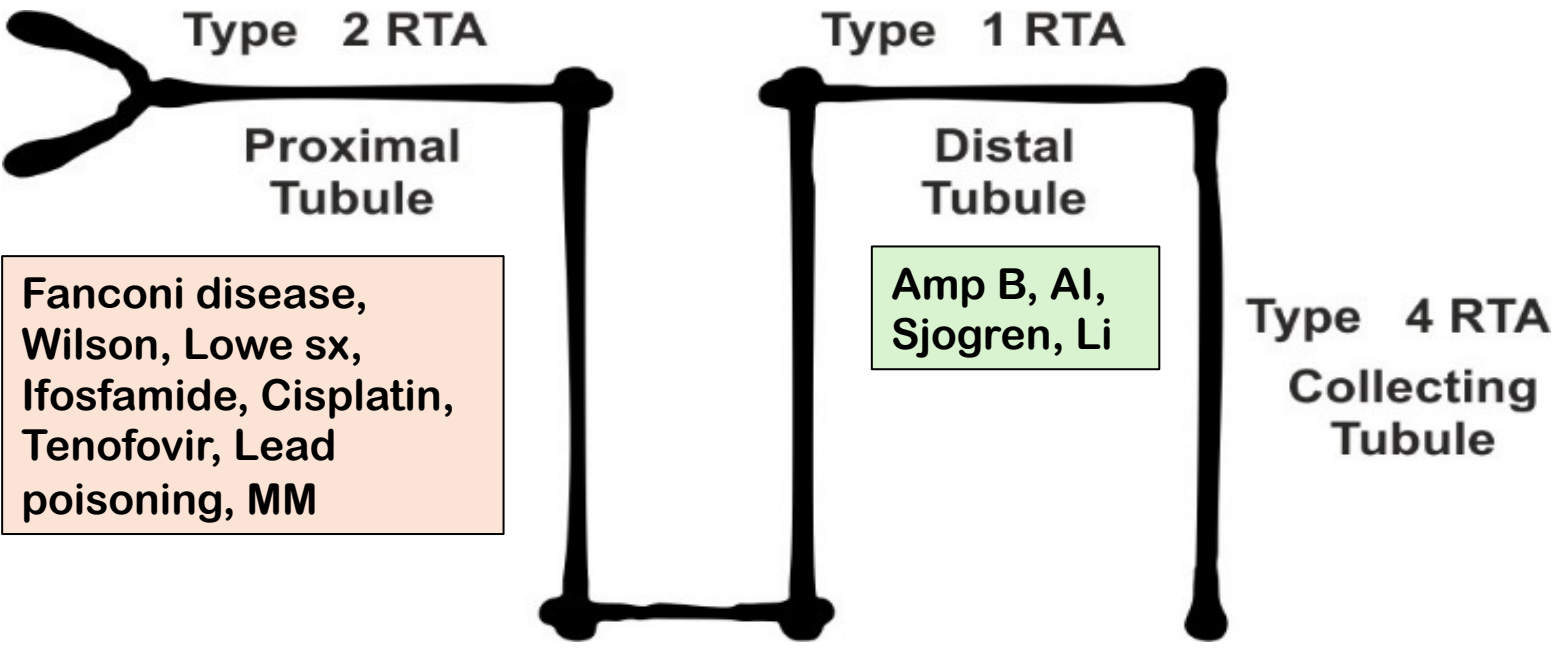
K⁺

P^H

Ca²⁺

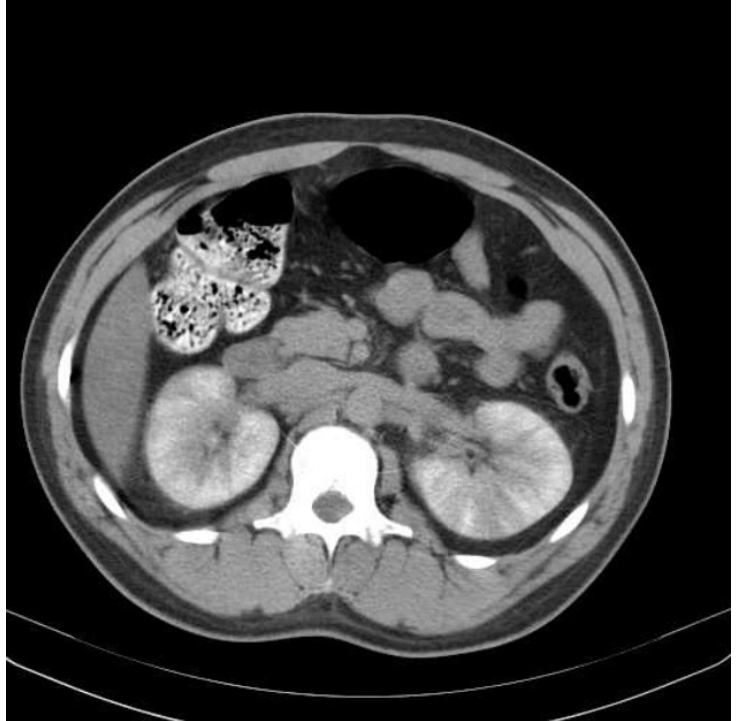
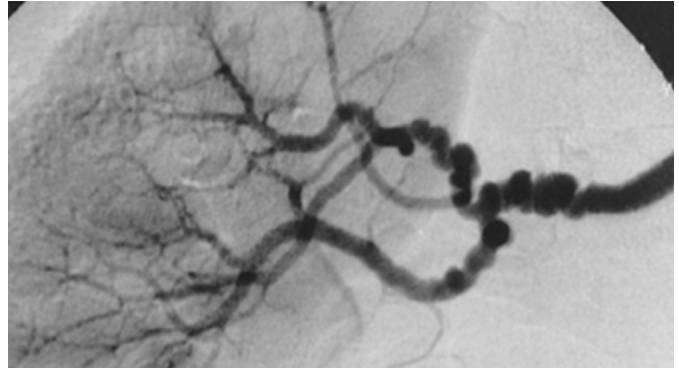
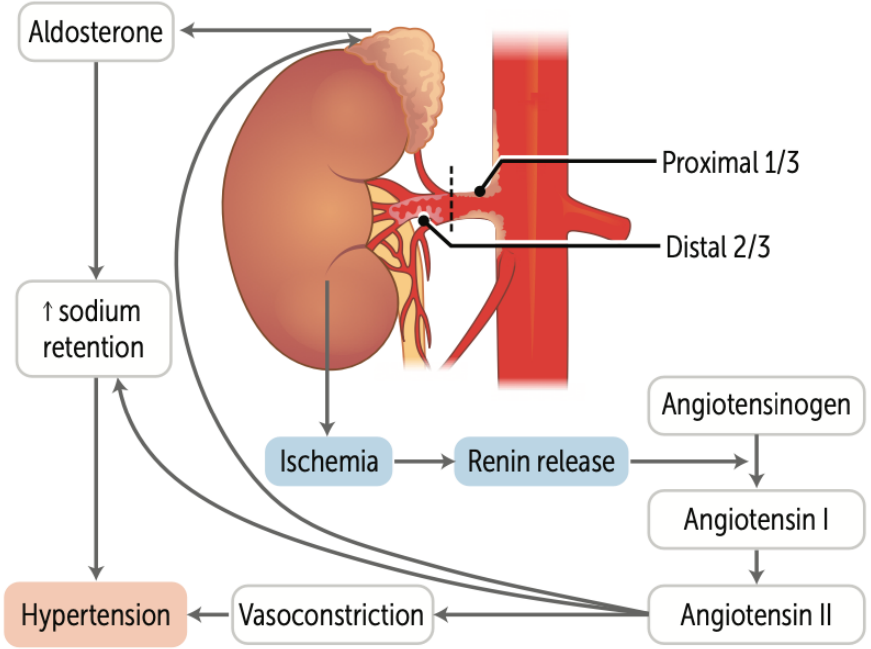
Dx					
BP					
K ⁺					
P ^H					
Ca ²⁺					

Renal Tubular Acidosis



	K	Ca	Urinary pH	Nephrolithiasis

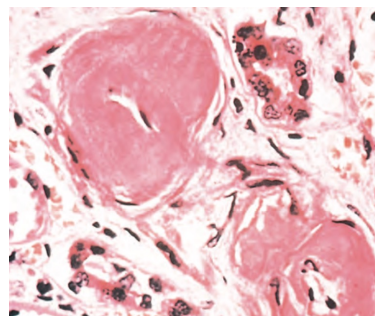
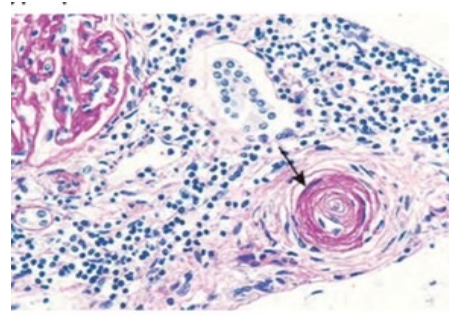
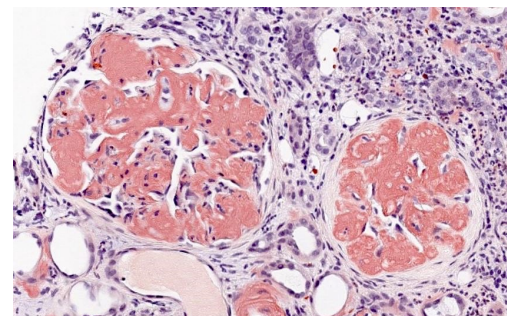
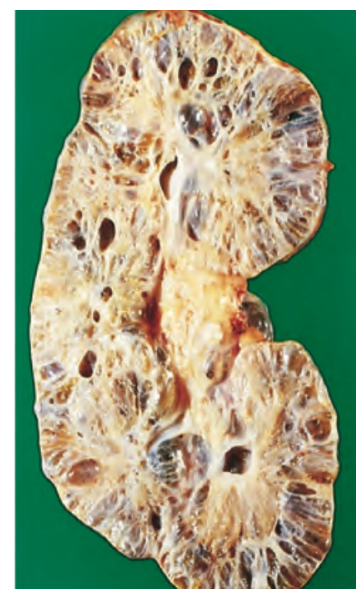
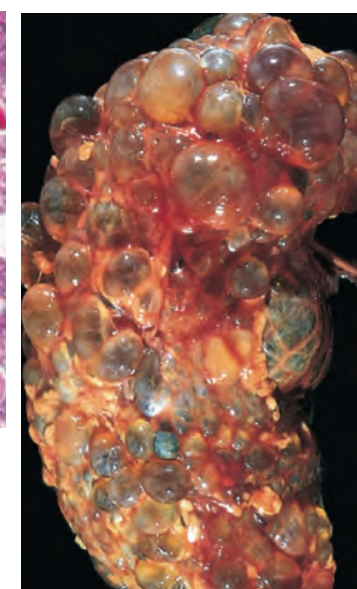
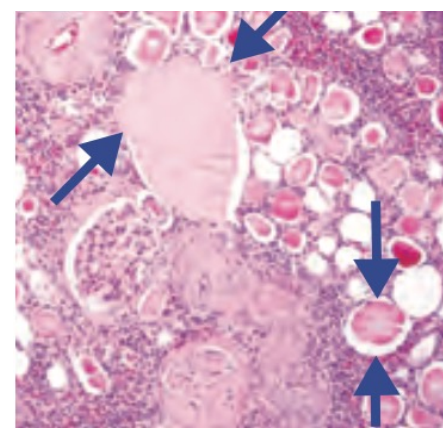
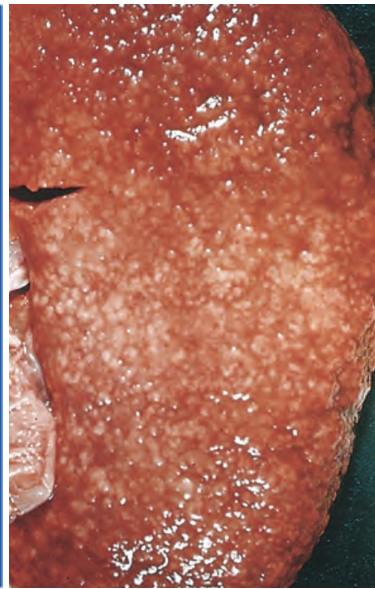
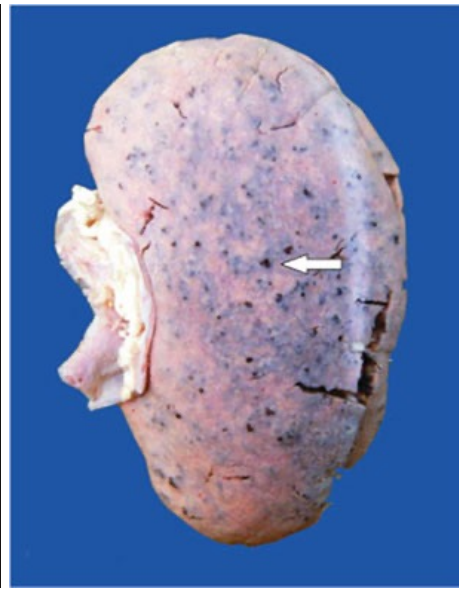
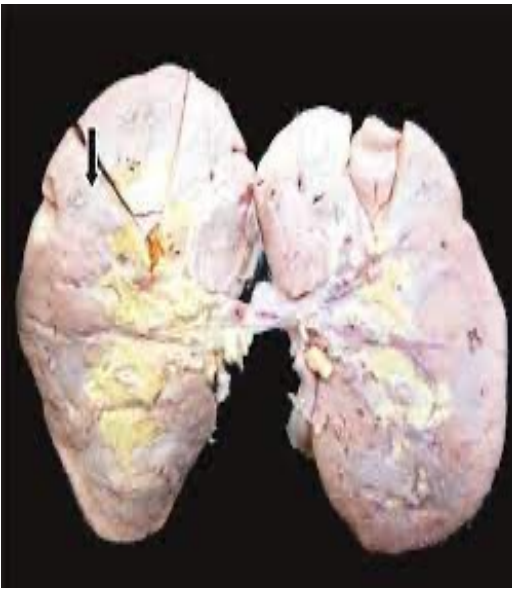
Reno-Vascular Diseases



Renal - Segmental - Interlobar - Arcuate - Interlobular artery - Afferent arteriole - Glomerulus - Efferent arteriole - Peritubular capillaries

Acute ureteric obstruction/
pyelonephritis/ renal vein
thrombosis

Renal Pathology



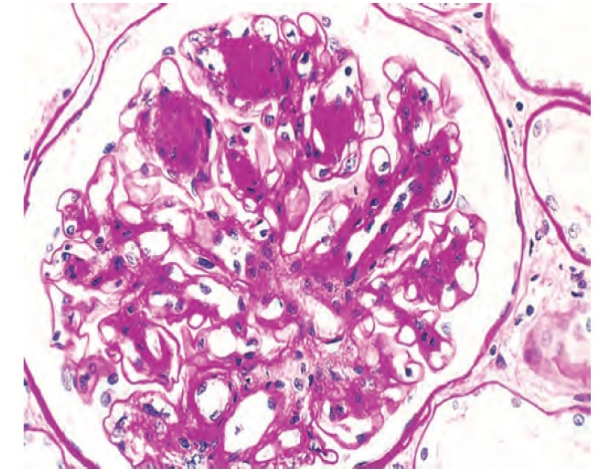
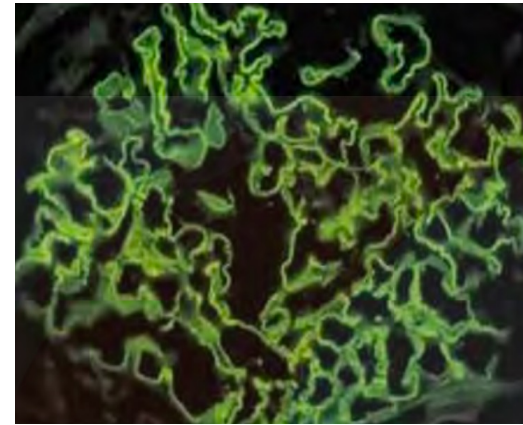
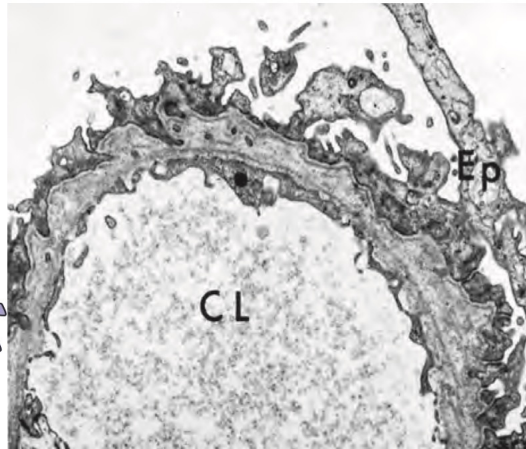
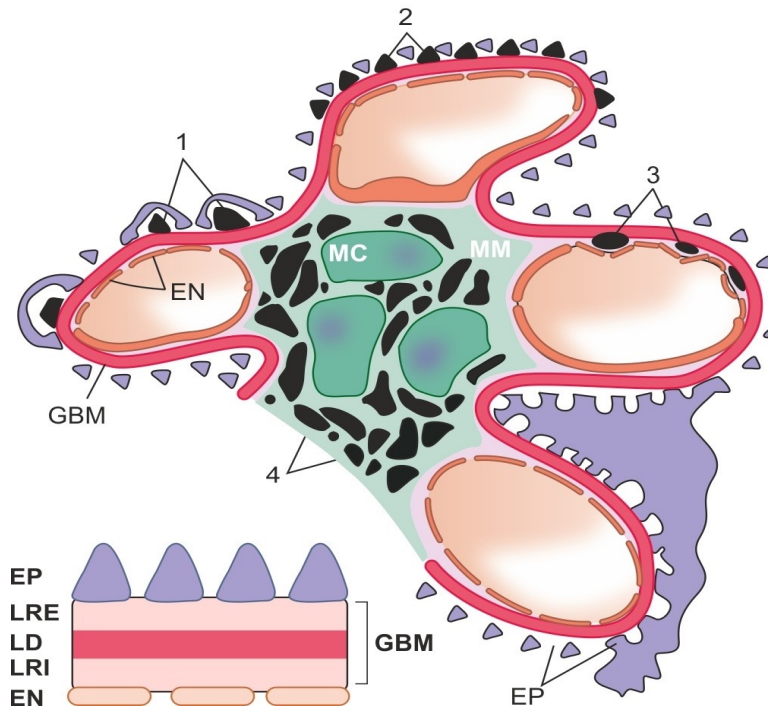
Chr 16,4: PKD1 & 2
Polycystin

- Aortic dissection
- Berry aneurysm
- Diverticulosis

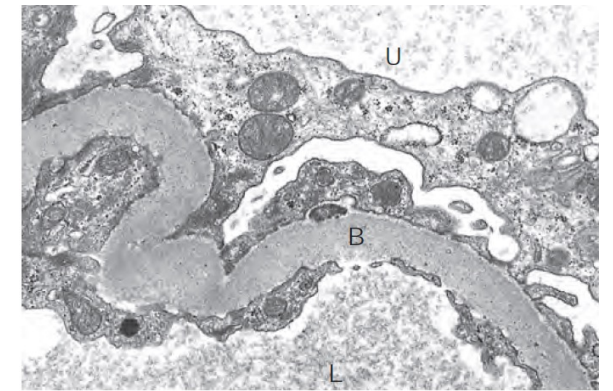
Chr 6: PKHD
Fibrocystin

Congenital
hepatic fibrosis

Glomerular Diseases



TYPE IV COLLAGEN



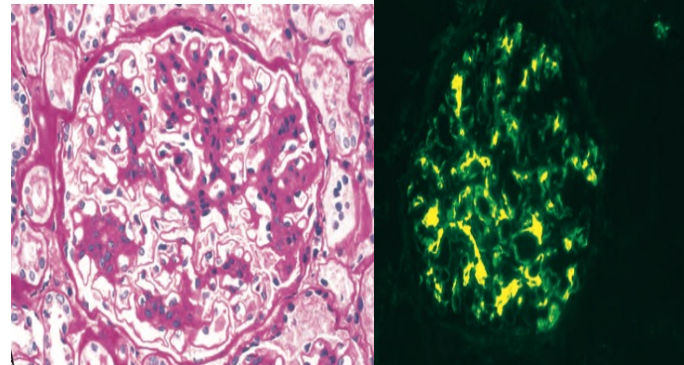
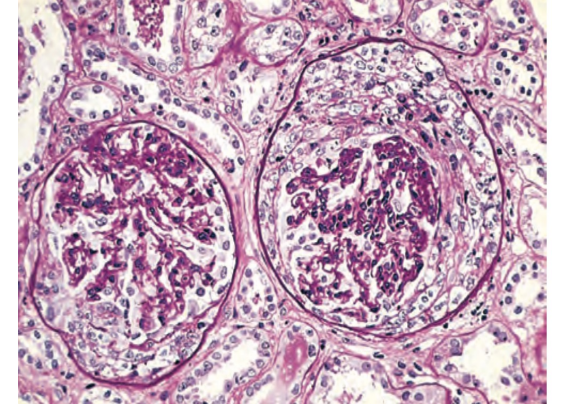
Nephritic Syndrome

Hematuria, Oliguria, Hypertension

Child
Hematuria 10-21d after
pharyngitis/impetigo
(strain 12,4,1)
Type III hysn
C3 transient low
70% Anti-DNase+
30% ASLO +

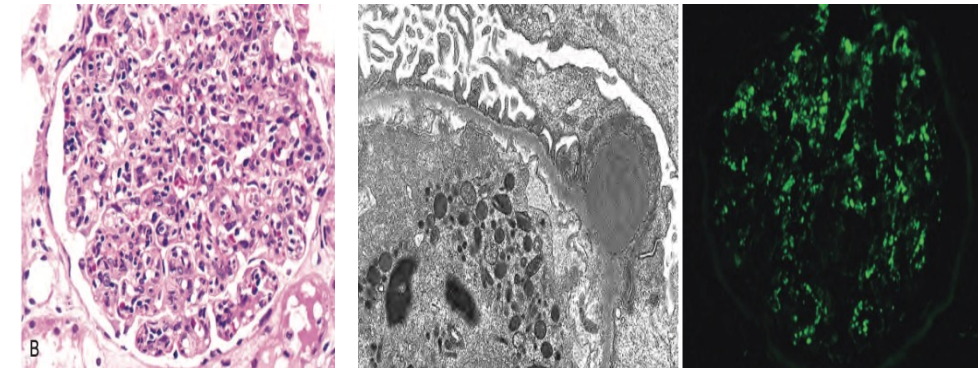
Adult
Hematuria 3d after pharyngitis
Recurrent gross hematuria
C3 normal

Rapid progressive



Linear:
Granular:
Pauci-immune:

Sparsentan: ARB + Endothelin -



Nephrotic Syndrome

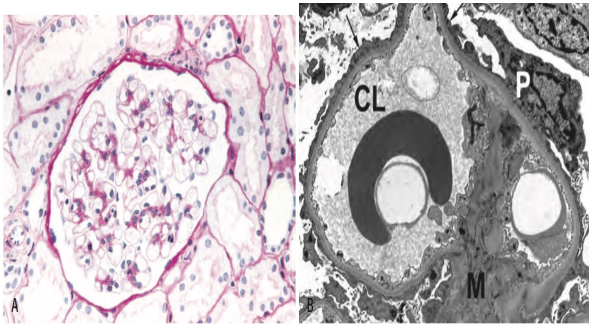
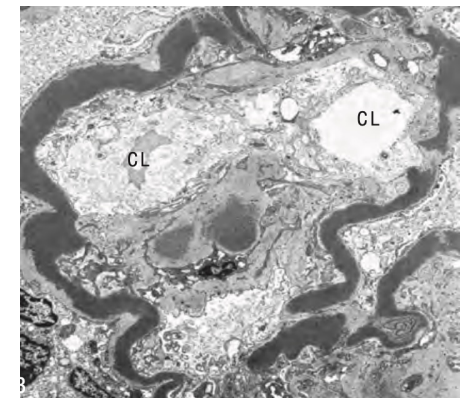
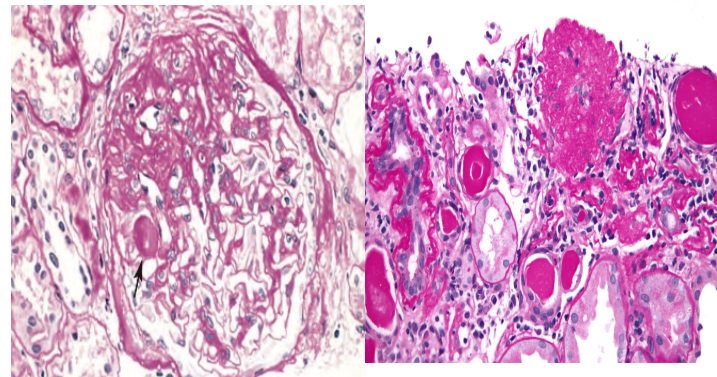
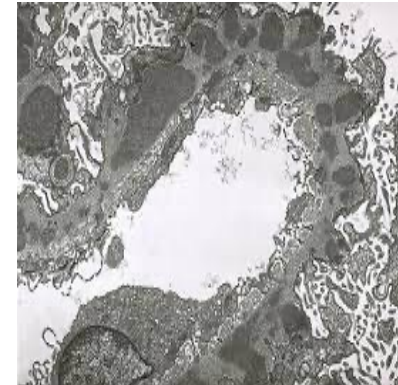
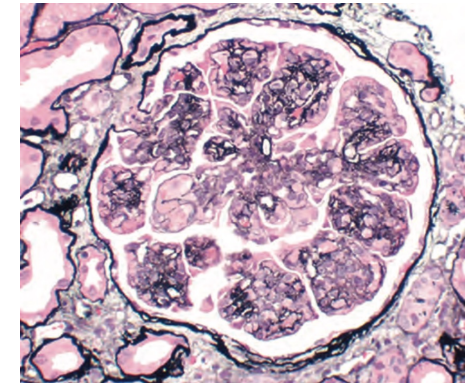
Proteinuria >3.5g/d, Edema, frothy urine

Child
Prior URTI
NSAIDS, Hodgkin's
lymphoma
Urine loss of:

Adults
Heroin
HIVAN: APOL1 polymorphism
Reflux nephropathy
Obesity
Sickle cell anemia
NPHS2: Podocin-AR FSGS
Actinin 4: AD FSGS
TRPC6: Adult FSGS

MC in elderly
Adenoca / melanoma
NSAID, penicillamine, gold
Hep B/
PLAR2
Thrombospondin, CD10

Adult
HCV / malaria
Cryoglobulinemia



TOC:
SRNS:
DOC:
Steroid dependent NS:

**MMF/ Cyclophosphamide/
Levamisole**
FRNS:

Nephrin NPHS1-Finnish type: Congenital NS

AKI vs CKD

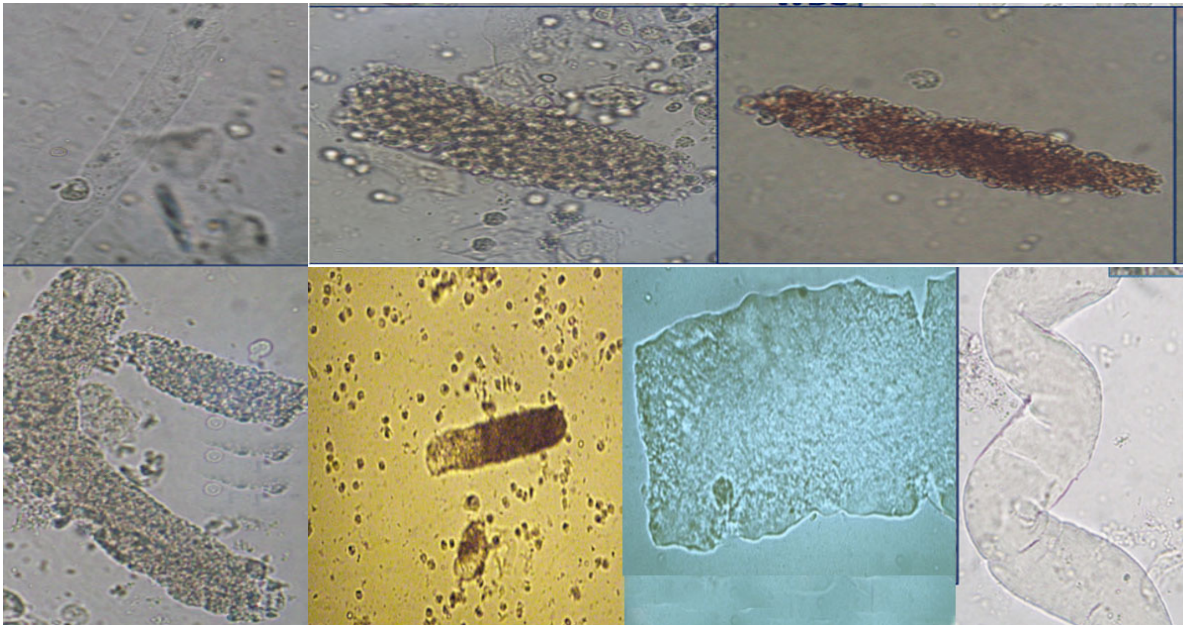
	<u>AKI</u>	<u>CKD</u>
CMD		
Size		
Urine Osmolarity		
Anemia		
MBD		
Casts		

Biomarkers of AKI:

Cystatin C
KIM-1
NGAL
TIMP2
NABG

Renal papillary necrosis:

- NSAIDS
- Sickle cell disease or trait
- Acute pyelonephritis
- Infections
- Diabetes mellitus

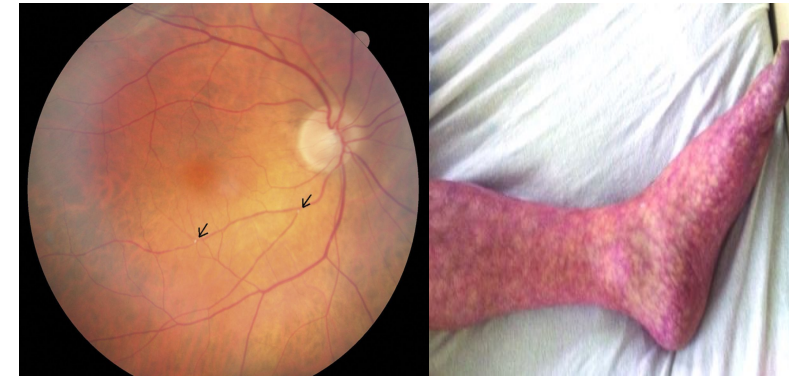


Hematuria:
Glomerular:

Non-Glomerular:

AKI

	<u>Pre renal</u>	<u>Acute Tubular necrosis</u>
Cause	Hypovolemia, CHF, NSAID / ACE-	Sepsis, Ischemia, Nephrotoxins: Rhabdomyolysis, IVH, Tumor lysis, MM, Drugs, Iodinated contrast
FeNa		
Urine Na		
Urine Osmolality		
BUN/Creatinine		
Urine casts		



Eosinophilia + Low complement

Fever + Rash + Eosinophilia + Drugs

Vancomycin, Aminoglycosides, Tenofovir, Cisplatin, Cidofovir, Foscarnet, AmpB, Crystals: Acyclovir, Indinavir

Acute Kidney Injury – Phases
 Initiation (damage)
 Maintenance (oligo/anuric)
 Resolution: polyuria → risk of

Risk KDIGO Stage 1	EGFR Decrease by 25%/ Creatinine Increase by 1.5x	UO <0.5 mL/kg/hour for 6 hours
Injury KDIGO Stage 2	EGFR Decrease by 50%/ Creatinine Increase by 2x	UO <0.5 mL/kg/hour for 12 hours
Failure KDIGO Stage 3	EGFR Decrease by 75% Creatinine Increase by 3x	UO <0.3 mL/kg/hour for 24 hours or anuric for 12 hour
Loss	Persistent renal failure > 4 weeks	-
End stage	Persistent renal failure >3 months	-

CKD

MCC of CKD:

MCC of death:

Anemia:

Indication: Hb <10 g/dl

Target:

Bone disease:

Rx:

Calciphylaxis:

Acid-base:

Uremia:

Pericarditis

P. Edema

pH <7.2

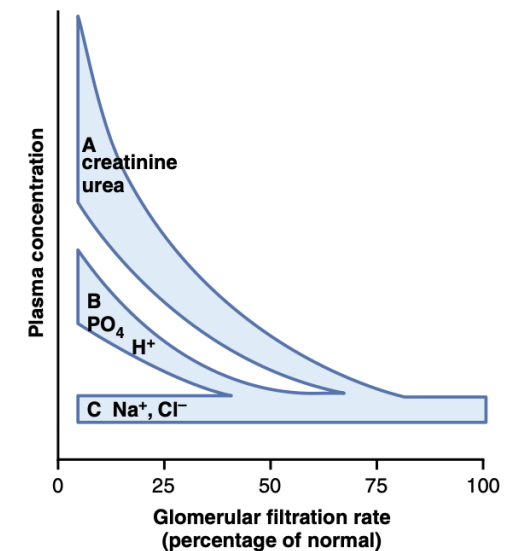
Potassium >6.5

Encephalopathy

Bleeding:

Dialysis disequilibrium syndrome DOC:

Stage of CKD	eGFR
Stage 1	≥ 90
Stage 2	60–89
Stage 3a	45–59
Stage 3b	30–44
Stage 4	15–29
Stage 5	<15

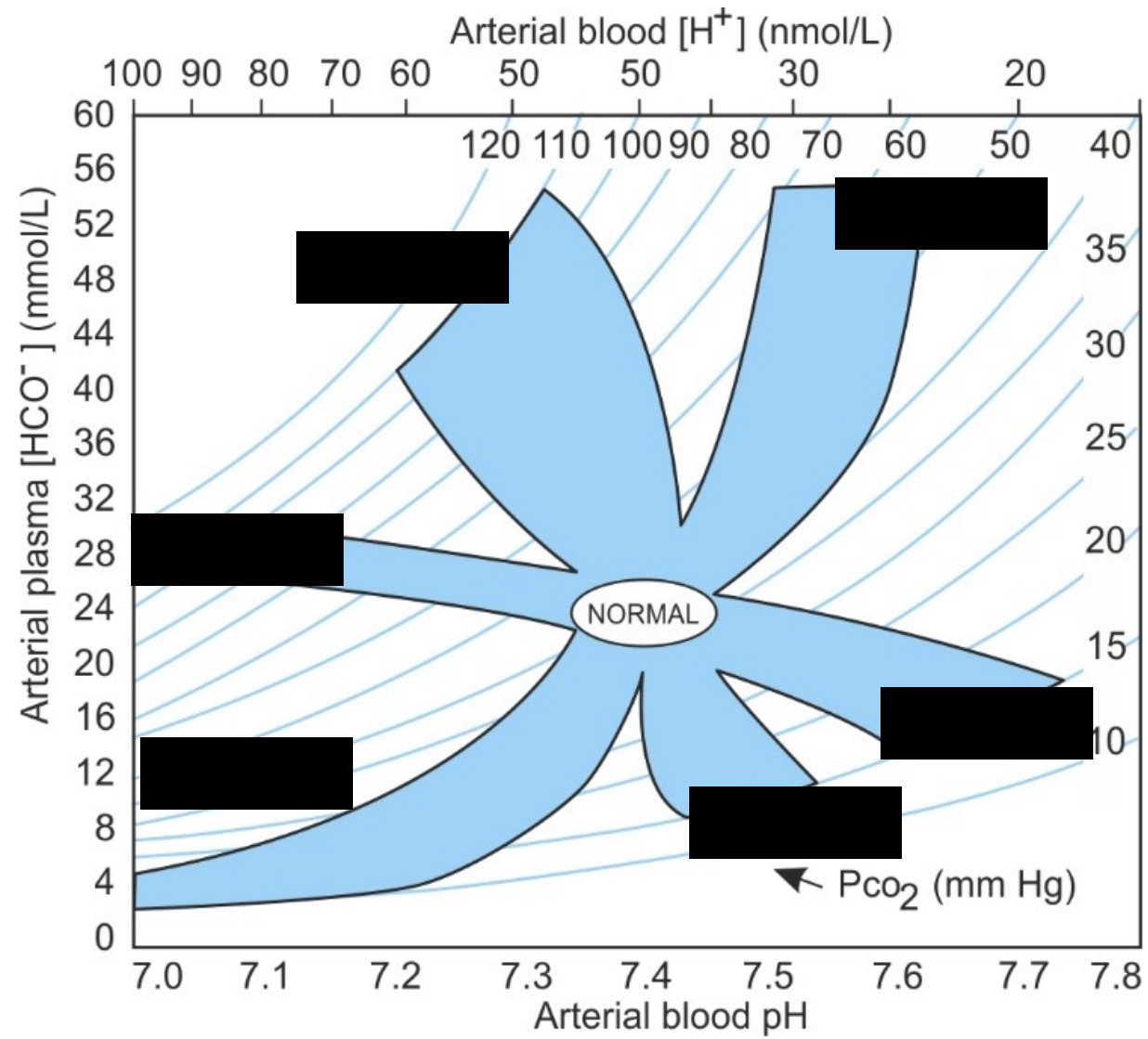


Acid-Base Balance

pH
CO₂
HCO₃⁻
Derived value in ABG:

	Causes	pH	Primary Change	Compensation
METABOLIC ACIDOSIS	HAGMA NAGMA			
METABOLIC ALKALOSIS	Hyperaldosteronism, Vomiting, Loop/thiazides Bartter-Gittleman			
RESPIRATORY ACIDOSIS	Hypoventilation Airway obstruction			
RESPIRATORY ALKALOSIS	Hyperventilation			

$$P_{CO_2} = 1.5[HCO_3] + 8 \pm 2$$



On laboratory investigations in a patient, pH=7.3, pCO₂=35 mm Hg, What is the likely acid base imbalance?

- A. Respiratory acidosis**
- B. Metabolic acidosis**
- C. Metabolic alkalosis**
- D. Respiratory alkalosis**

**A patient is having pH-7.12, HCO₃-28 and PCO₂-50 mm Hg.
What is the acid base disorder in this patient?**

- A. Metabolic acidosis with respiratory compensation**
- B. Metabolic alkalosis with respiratory compensation**
- C. Respiratory acidosis with renal compensation**
- D. Respiratory alkalosis with renal compensation**

**A patient is having pH-7.27, HCO₃-14 and PCO₂-28 mm Hg.
What is the acid base disorder in this patient?**

- A. Metabolic acidosis with respiratory compensation**
- B. Metabolic acidosis with respiratory acidosis**
- C. Metabolic acidosis with respiratory alkalosis**
- D. Respiratory alkalosis with renal compensation**

A patient who is a known case of CKD has complaints of vomiting. His ABG reports are as follows: pH-7.40, pCO₂- 40, HCO₃⁻-25. Na-145, chloride-100. What is the metabolic abnormality?

- A. Normal anion gap metabolic acidosis**
- B. High anion gap metabolic acidosis**
- C. No acid base abnormality**
- D. High anion gap metabolic acidosis with metabolic alkalosis**

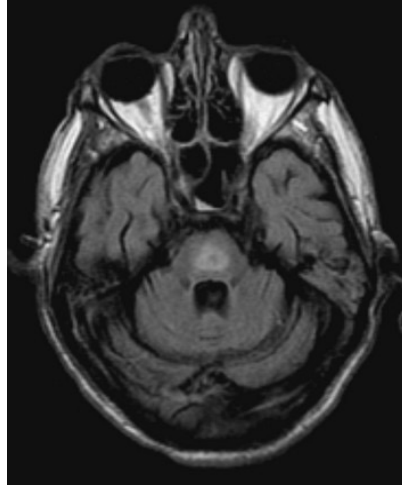
Electrolyte abnormalities

Serum osmolarity: $2(\text{Na}) + \text{glucose}/18 + \text{BUN}/2.8$

MANAGEMENT of HypoNa
Sodium deficit:

Max: 8 meq/l in a day
Severe-Seizures/coma:
Overcorrection:

Drugs causing HyperK
K sparing diuretics
ACE-/ARB
Digoxin
 β -blocker
Calcineurin inhibitors
Pentamidine
NSAIDs
Heparin
Succinylcholine
Trimethoprim



Free water deficit:

Management of Hyperkalemia:
1st step:

Transcellular spread:

Eliminate:

Most effective:

Binders:

- Sodium polystyrene sulfonate (SPS / Kayexalate)
- Patiromer
- Sodium zirconium cyclosilicate