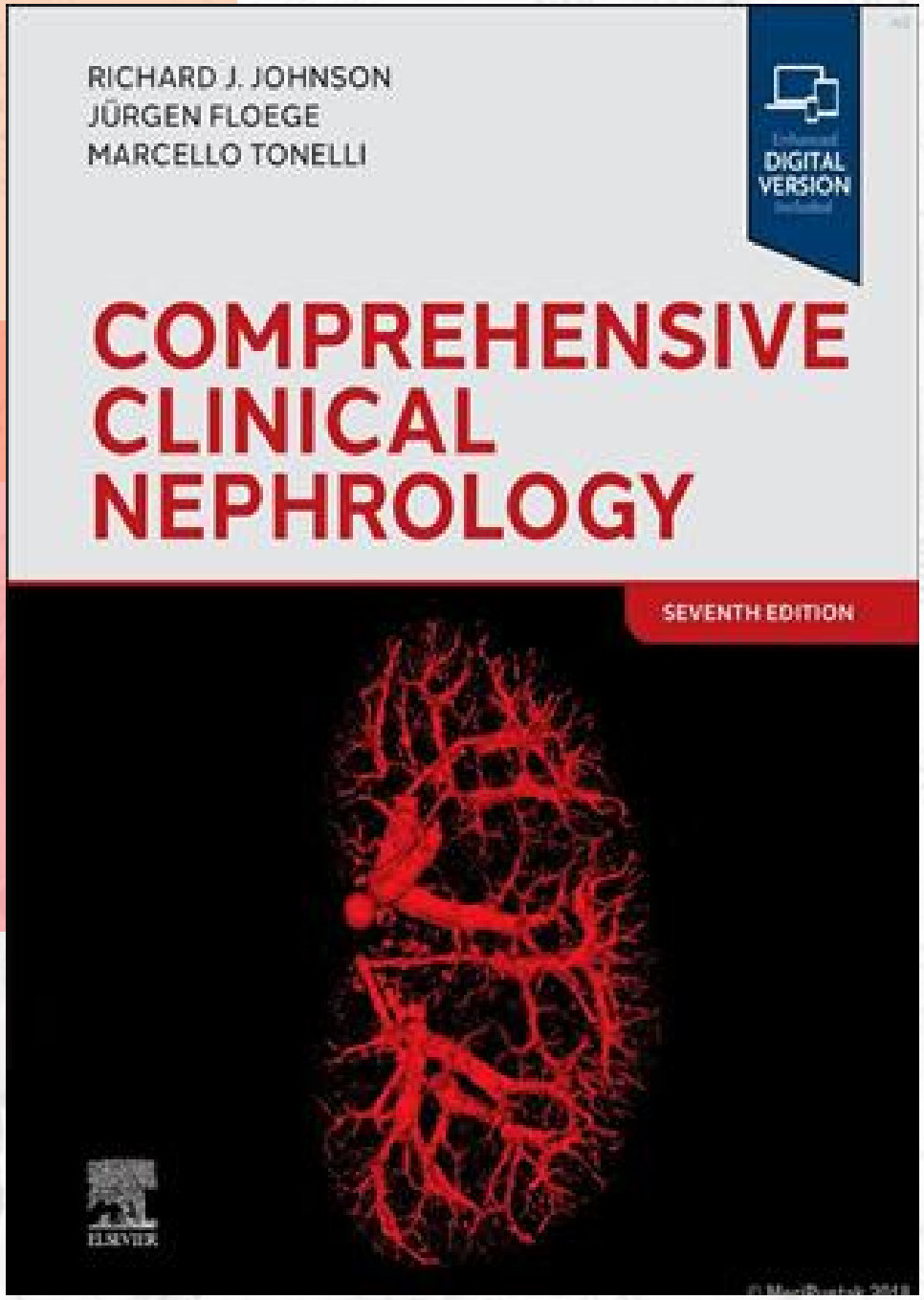
STEM-S

Last Minute Revision

LMR NOTES



INI-SS NEPHROLOGY

PRESENTED BY
Stem-S

Nephrology

CORE CONCEPT

- Kidney = cortex + medulla + collecting system
- Functional unit = nephron (~1–1.5 million/kidney)
- Blood flow = unique double capillary system
- Juxtaglomerular apparatus (JGA) = BP + GFR regulator
- Countercurrent system = urine concentration mechanism

GROSS ANATOMY

• LOCATION & EXTENT

Feature	Detail
Position	Retroperitoneal
Vertebral level	T12–L3
Right vs Left	Right lower (~1–2 cm)
Movement	Moves with respiration

• SIZE & SHAPE

Parameter	Value
Length	10–12 cm
Width	5–7 cm
Thickness	3 cm
Weight	~150 g
Shape	Bean-shaped

• EXTERNAL FEATURES

Feature	Description
Anterior surface	Related to viscera
Posterior surface	Muscles + ribs
Lateral border	Convex
Medial border	Concave (hilum present)

HILUM ARRANGEMENT : Anterior → Posterior = V A P

• COVERINGS

Layer (inside → out)	Feature
Capsule	True capsule
Perirenal fat	Protection
Renal fascia (Gerota)	Anchors kidney
Pararenal fat	Outer layer

- **GLOMERULAR FILTRATION BARRIER**

Layer	Function
Endothelium	Blocks cells
GBM	Charge barrier (−ve)
Podocytes	Size barrier

CLINICAL LINK

- Charge loss → Minimal change disease
- Structural damage → Nephritic syndrome

JUXTAGLOMERULAR APPARATUS (JGA)

Specialized structure at vascular pole of glomerulus
Senses NaCl (macula densa) + perfusion
Regulates GFR + BP via RAAS
Key for tubuloglomerular feedback (TGF)
Links tubule ↔ arteriole

- **COMPONENTS**

Component	Location	Function	Key Mediators
Macula densa	DCT	Senses NaCl	Adenosine, NO
JG (Granular) cells	Afferent arteriole	Secrete renin	β1 receptors
Lacis (Extraglomerular mesangial)	Between A & E arterioles	Signal transmission	Support

HYPERNATREMIA (Na^+ >145 mEq/L)

CORE CONCEPT

- Always = water deficit > sodium excess
- Reflects hyperosmolality → cellular dehydration (brain shrinkage)
- Key determinant = volume status (hypovolemic / euvolemic / hypervolemic)
- Most cases = impaired thirst or limited water access
- Rapid correction → cerebral edema → seizures

CAUSES (CLASSIFICATION)

1. Hypovolemic Hypernatremia (↓ Na, ↓↓↓ water)

- GI loss: diarrhea, vomiting
- Renal loss: **osmotic diuresis (glucose, urea), diuretics**
- Skin: burns, sweating

2. Euvolemic Hypernatremia (normal Na, ↓ water)

- **Central DI (↓ ADH)**
- **Nephrogenic DI (ADH resistance)**
- Insensible loss (fever, ventilation)

3. Hypervolemic Hypernatremia (↑ Na, ↑ water)

- **Iatrogenic Na excess ($NaHCO_3$, hypertonic saline)**
- Mineralocorticoid excess (rare cause)

By Etiology

- Primary: MCD, FSGS, MN, IgA nephropathy
- Secondary: DM, SLE, infections, amyloidosis

• MECHANISM OF SYMPTOMATOLOGY IN GLOMERULAR DISORDERS

PROTEINURIA → EDEMA (NEPHROTIC AXIS)

STEPWISE MECHANISM

Podocyte injury (MCD, FSGS, MN)
→ Loss of slit diaphragm (nephrin, podocin)
→ ↑ GBM permeability
→ **Massive albuminuria (>3.5 g/day)**
→ ↓ plasma oncotic pressure
→ Fluid shifts to interstitium
→ **EDEMA**

TWO PARALLEL THEORIES (IMPORTANT MCQ)

A. UNDERFILL THEORY

↓ albumin
→ ↓ plasma oncotic pressure
→ fluid → interstitium
→ ↓ effective circulating volume
→ ↑ RAAS
→ Na + water retention → edema

B. OVERFILL THEORY (MODERN CONCEPT)

Primary renal Na retention (ENaC activation)
→ Na retention
→ water retention
→ edema (independent of RAAS)

2. HEMATURIA (NEPHRITIC AXIS)

MECHANISM

Immune complex deposition / inflammation
→ GBM damage
→ RBC leakage into urine
→ Dysmorphic RBCs + RBC casts

Maintenance Therapy (≥2–3 years)

- MMF or Azathioprine
- Continue low-dose steroids (taper)
- HCQ lifelong

3. FLARES / RESCUE

- High-dose IV steroids (methylprednisolone)
- Rituximab / Belimumab for refractory disease

4. SUPPORTIVE CARE

- BP control (ACEi/ARB)
- Proteinuria management
- Lipid-lowering therapy
- Anticoagulation if antiphospholipid syndrome

HIGH-YIELD PEARLS

- ANA sensitive, anti-dsDNA specific
- Lupus nephritis → major prognostic determinant
- Class IV LN → aggressive immunosuppression
- Hydroxychloroquine = standard for all patients
- Complement levels correlate with disease activity
- Flares precipitated by UV, infection, pregnancy
- Steroid-sparing agents → MMF, Azathioprine, Rituximab

CHANNELopathies (RENAL FOCUS)

CORE CONCEPT (≤5 lines)

- Channelopathies = ion channel / transporter defects
- Cause electrolyte + acid-base disorders
- Often genetic → tubular dysfunction
- Key = pattern recognition of electrolytes

Common: Na^+, K^+, Cl^-, Ca^{2+} channel defects

STEP 4: RESISTANT HTN

Defined:

- BP uncontrolled on ≥3 drugs (incl. diuretic)

Management

- Add spironolactone
- Evaluate:
  - Volume overload
  - OSA
  - Renal artery stenosis

DRUG TABLE (HIGH-YIELD)

Drug	Mechanism	Benefit	Trap
ACEi/ARB	↓ intraglomerular pressure	↓ proteinuria	↑ K, ↑ Cr
Diuretics	↓ volume	BP control	Electrolyte imbalance
CCB	Vasodilation	Add-on	Edema
Finerenone	Anti-fibrotic	CKD benefit	Hyperkalemia
SGLT2i	↓ hyperfiltration	Renoprotection	Initial ↓ GFR

HIGH-YIELD PEARLS

- HTN both cause & consequence of CKD
- ACEi/ARB = cornerstone
- Target SBP <120 (KDIGO)
- Loop diuretics in eGFR <30
- Proteinuria = key treatment driver
- Resistant HTN → think volume overload

SECONDARY HYPERTENSION

A. INITIAL (ALL PATIENTS)

- Stop nephrotoxins
- Optimize volume
- Monitor electrolytes
- Treat underlying cause

B. ETIOLOGY-SPECIFIC

1. PRERENAL

- IV fluids (NS)
- Treat shock

2. ATN

- Supportive
- Avoid overload
- No routine diuretics

3. AIN

- Stop offending drug
- Steroids if needed

4. POSTRENAL

- Relieve obstruction immediately ⭐
  - Catheter
  - Stent

V. NON-DIALYTIC MANAGEMENT ⭐ ⭐ ⭐

FLUID MANAGEMENT

- Hypovolemia → fluids
- Hypervolemia → diuretics