Nephrology Quick-Start Guide: Essential Foundations for Clinical Practice

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March 2026

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Learning Objectives

By the end of this handout, you will be able to: - Describe the functional anatomy of the nephron and explain how different segments contribute to urine formation - Interpret basic kidney function tests (creatinine, eGFR, BUN) and recognize their limitations - Classify acute kidney injury using KDIGO criteria - Understand normal electrolyte homeostasis and recognize common disorders - Apply a systematic approach to volume status assessment - Recognize indications for nephrology consultation

Part 1: Essential Kidney Anatomy & Physiology

The Nephron: Your Foundation

Each kidney contains ~1 million nephrons, the functional units that filter blood and regulate fluid/electrolyte balance.

Key Segments and Functions:

Segment	Key Functions	Physiologic Target
Glomerulus	Ultrafiltration; filters 180L/day	Protein barrier maintenance
Proximal Tubule	Reabsorbs 65% filtered Na+, all glucose, amino acids	Energy-dependent transport
Loop of Henle	Creates medullary concentration gradient; countercurrent multiplication	Urine concentration
Distal Tubule	Fine-tunes Na+, K+, Ca2+ balance; responds to thiazides	Electrolyte regulation
Collecting Duct	Final regulation of Na+, K+, water under hormonal control	ADH and aldosterone response

Critical Concept: Normal kidney functions extend beyond filtration: - **Endocrine:** Erythropoietin (RBC production), vitamin D activation (calcium homeostasis) - **Metabolic:** Gluconeogenesis, acid-base balance through ammonia production - **Regulatory:** Long-term blood pressure control through RAAS activation

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Part 2: Interpreting Kidney Function Tests

The "Imperfect Trinity" — What You Need to Know

Serum Creatinine (Scr) - Normal range: Men 0.7-1.3 mg/dL; Women 0.6-1.1 mg/dL - Produced at relatively constant rate from muscle metabolism - **Limitations:** Influenced by muscle mass, age, sex, medications (trimethoprim, cimetidine can falsely elevate) - Rule of thumb: A "normal" creatinine of 1.2 in a 75-year-old woman may represent significant kidney dysfunction

Estimated Glomerular Filtration Rate (eGFR) - Calculated from creatinine using CKD-EPI equation (preferred) - Accounts for age, sex, race - Normal: >90 mL/min/1.73m² - **Caveat:** Less reliable during acute kidney injury, extremes of body composition, rapidly changing function

Blood Urea Nitrogen (BUN) - Normal: 7-20 mg/dL - Influenced by protein intake, liver function, catabolism - BUN:Cr ratio helps differentiate prerenal azotemia (>20:1) from intrinsic kidney disease (10-15:1)

Alternative Markers: - **Cystatin C:** Less influenced by muscle mass; useful in elderly, children - **Spot protein-to-creatinine ratio (SPCR):** 1.0 ≈ 1 g protein/day - **Albumin-to-creatinine ratio:** <30 mg/g normal; 30-300 mg/g = microalbuminuria

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Part 3: Urinalysis — The Window into Kidney Disease

Quick Interpretation Framework

Physical Properties: - **Color:** Pale yellow to amber normal; brown/red = hematuria or pigmenturia - **Clarity:** Clear to slightly hazy; cloudy = infection, crystals, or proteinuria - **Specific Gravity:** 1.003-1.030; fixed at 1.010 = concentrating defect

Chemical Components Interpretation:

Finding	Significance	Next Step
Protein 1+ or more	Glomerular disease likely if quantified >150 mg/day	Quantify with SPCR or 24h urine
Blood positive, RBCs seen	Hematuria; dysmorphic cells = glomerular; uniform = lower urinary tract	Dysmorphic RBCs + casts = GN
Blood positive, NO RBCs	Hemoglobinuria or myoglobinuria (intravascular hemolysis or rhabdo)	Check rhabdo labs, LDH, urinalysis
Glucose (no diabetes)	Proximal tubule dysfunction or very high serum glucose	Check serum glucose; consider Fanconi
Ketones + Glucose	Diabetic ketoacidosis until proven otherwise	Stat VBG, electrolytes, insulin

Finding	Significance	Next Step
Nitrites positive	Bacterial infection (gram-negatives especially)	Urine culture; empiric abx if symptomatic
Leukocyte esterase	WBC present; suggests infection or inflammation	Culture; clinical context matters

Casts — The Pathognomonic Findings: - **RBC casts:** Pathognomonic for glomerulonephritis ☐ urgent evaluation for RPGN - **WBC casts:** Suggest pyelonephritis or interstitial nephritis - **Granular casts:** Coarse = acute tubular necrosis; fine = chronic processes - **Hyaline casts:** May be normal after exercise; increased numbers suggest reduced flow

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Part 4: Acute Kidney Injury (AKI) — Diagnosis & Classification

KDIGO Staging System

Definition: Increase in serum creatinine OR decrease in urine output within 48 hours

Stage	Serum Creatinine	Urine Output	Clinical Significance
Stage 1	1.5-1.9x baseline OR ≥0.3 mg/dL increase	<0.5 mL/kg/hr × 6-12 hrs	Mild; still reversible
Stage 2	2-2.9x baseline	<0.5 mL/kg/hr × 12-24 hrs	Moderate; requires intervention
Stage 3	≥3x baseline OR ≥4.0 mg/dL	<0.3 mL/kg/hr × 24 hrs OR anuria ≥12 hrs	Severe; may need RRT

AKI Etiologies — The Prerenal-Intrinsic-Postrenal Framework

Prerenal AKI (55-60% of cases) - Mechanism: Decreased renal perfusion - Key findings: FENa <1%, Scr/BUN ratio >20:1, oliguria - Common causes: Hypovolemia, shock, medications (ACEi/ARB in specific settings) - Response to IV fluids: Responsive

Intrinsic AKI (35-40% of cases) - Acute Tubular Necrosis (ATN): Ischemic or nephrotoxic; most common intrinsic cause - Acute Interstitial Nephritis (AIN): Drug-induced (NSAIDs, antibiotics, PPIs); eosinophiluria suggests drug reaction - Glomerulonephritis: RBC casts + dysmorphic RBCs pathognomonic - Vascular: Renal infarction, thrombosis, dissection

Postrenal AKI (5% of cases) - Mechanism: Urinary obstruction - Findings: Hydronephrosis on ultrasound - Common causes: Stones, BPH, malignancy, strictures - Response: Urgent decompression

Key Decision Tool: FENa Calculation

Fractional Excretion of Sodium (FENa) = (Urine Na × Serum Cr) / (Serum Na × Urine Cr) × 100

- FENa <1% ☐ Prerenal (conserving sodium)
- FENa >2% ☐ Intrinsic (wasting sodium)
- **Exception:** High FENa can occur in prerenal if already on diuretics

Pro Tip: When AKI is recognized early and prerenal factors identified, aggressive IV hydration can prevent progression to established AKI.

Part 5: Electrolyte Disorders at a Glance

Sodium Disorders

Hyponatremia (Na <135 mEq/L) - Represents water excess relative to sodium - Classify by volume status first (hypovolemic, euvolemic, hypervolemic) - Acute vs chronic: Acute (<48 hrs) carries seizure risk; chronic adaptation prevents symptoms - **Danger:** Rapid correction causes osmotic demyelination syndrome (ODS) - Correction rate: <8 mEq/L per 24 hours for chronic hyponatremia

Hypernatremia (Na >145 mEq/L) - Represents water deficit - Causes: Inadequate free water intake, excessive free water losses (DI) - Central DI: Responds to desmopressin; nephrogenic DI: Does not respond - Water replacement must be gradual; too-rapid correction causes cerebral edema

Potassium Disorders

Hyperkalemia (K >5.0 mEq/L) — THE EMERGENCY - ECG changes correlate poorly with symptom severity - **Emergencies: Peaked T waves ☐ widened QRS ☐ loss of P wave ☐ sine wave ☐ asystole** - Treatment: "ABCs" = Antagonize (calcium gluconate), Shift (insulin/glucose, beta-agonists), Remove (diuretics, dialysis, binders) - Risk factors: eGFR <15, RAAS inhibitors, NSAIDs, K-sparing diuretics, cell breakdown states

Hypokalemia (K <3.5 mEq/L) - Causes: Diuretics, GI losses, cellular shifts - Cardiac arrhythmias: Increased U waves, flattened T waves, ST depression - **Critical rule:** Every 1 g of KCl deficit ≈ 2-3 mEq/L drop in serum K - Refractory hypokalemia: Check magnesium (need to replete Mg for K to normalize)

Calcium Disorders

Hypercalcemia — Think VITAMINS TRAP - **V**itamins (A, D), **I**ntoxication, **T**umor, **A**ddison, **M**ilk-alkali, **I**mmunity, **N**eoplasm, **S**arcoidosis - **T**hyroid, **R**etinol, **A**ntacids, **P**heo - Presentation: "Stones, bones, groans, and psychiatric overtones" - Severity guide: Mild (<11 mg/dL) vs Symptomatic (>13 mg/dL)

Hypocalcemia — Think PRIMARY CAUSE - **P**ancreatitis, **R**enal disease (CKD), **I**nfections, **M**alabsorption, **A**lkaline load, **R**Y = Vitamin D deficiency - Check PTH to differentiate PTH-mediated (PTH low) from vitamin D deficiency (PTH elevated) - Acute symptomatic: Calcium gluconate IV (not calcium chloride peripherally — risk of necrosis)

Magnesium (Often Overlooked!)

- Normal: 1.7-2.2 mg/dL
- **Critical:** ~30% of hypokalemia is magnesium depletion; can't fix K+ without repleteing Mg
- PPI use is common cause of Mg wasting
- Hypomagnesemia worsens hypokalemia and increases arrhythmia risk

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Part 6: Volume Status Assessment — The Clinical Skill

Determining Volume Status (Critical for Treatment Selection)

Sign/Symptom	Hypovolemia	Euvolemia	Hypervolemia
Vital Signs	Orthostatic changes	Stable	Hypertension
JVP	Flat (<2 cm)	2-8 cm	Elevated (>8 cm)
Lungs	Clear	Clear	Rales, pulmonary edema
Extremities	Dry, poor turgor	Normal	Peripheral/sacral edema
Mucous Membranes	Dry	Moist	Normal
Urine Output	Oliguric	Normal	Variable

Physical Exam Pearls: - Assess JVP with head of bed at 30-45° - Check skin turgor at forearm (not back of hand in elderly) - Orthostatic vital signs: Drop >20 systolic or >10 diastolic = significant hypovolemia - Weight changes: 1 L fluid ≈ 1 kg body weight

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Part 7: Chronic Kidney Disease (CKD) Staging

KDIGO Staging by eGFR

Stage	eGFR	Description	Management Focus
1	≥90	Normal/high	Treat underlying cause
2	60-89	Mildly decreased	Slow progression
3a	45-59	Mildly to moderately decreased	Control BP, proteinuria
3b	30-44	Moderately to severely decreased	Prepare for RRT
4	15-29	Severely decreased	Dialysis education begins
5	<15	Kidney failure	RRT or conservative care

Key CKD Complications (Remember: These Accelerate Decline)

1. **Hypertension:** Needs aggressive control (target SBP <120 in many)

2. **Proteinuria:** Every gram/day ≈ faster GFR decline
3. **Bone disease:** PTH-vitamin D axis dysregulation
4. **Anemia:** EPO production declines
5. **Acidosis:** Contributes to bone loss
6. **Malnutrition:** Wasting affects outcomes

Clinical Pearl Summary

"The Big 5" — Always Consider in Any Kidney Patient:

1. **Volume Status** — Hypovolemia looks like "prerenal," hypervolemia looks like "CHF"
2. **Medications** — ACEi/ARB, NSAIDs, diuretics, antibiotics are common culprits
3. **Obstruction** — Never miss a post-renal cause; simple ultrasound is quick
4. **Infection/Inflammation** — Pyelonephritis, endocarditis present as AKI
5. **Baseline Renal Function** — You must know the baseline Cr to interpret new values

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Practice Questions

Question 1: A 72-year-old man with baseline creatinine 1.4 mg/dL presents with creatinine 1.9 mg/dL. Which statement is MOST accurate? - A) His eGFR has increased by 5 mL/min/1.73m² - B) He meets KDIGO Stage 1 AKI criteria - C) His actual kidney function loss may be much greater due to lower baseline muscle mass - D) He can safely receive IV contrast without premedication

Answer: B is correct (1.9/1.4 = 1.36, which is between 1.5-1.9x baseline = Stage 1). C is a clinical pearl—in elderly patients with lower muscle mass, creatinine changes underestimate kidney function loss.

Question 2: Patient with hyponatremia (Na 126), euvolemic, urine osmolality 650 mOsm/kg (high). Most likely diagnosis? - A) Cerebral salt wasting (hypovolemic) - B) SIADH (euvolemic) - C) Nephrogenic DI (hypervolemic) - D) Primary polydipsia

Answer: B. Euvolemic + high urine osmolality = kidney appropriately concentrating urine but body can't handle the water load = SIADH. Treatment: fluid restriction.

Question 3: A patient on lisinopril presents with K 6.8 mEq/L and peaked T waves on ECG. Which intervention provides IMMEDIATE cardiac membrane stabilization? - A) IV insulin 10 units with 25 g dextrose - B) IV calcium gluconate 10 mL of 10% solution - C) Sodium polystyrene sulfonate 15 g PO - D) Hemodialysis

Answer: B. Calcium antagonizes hyperkalemia's membrane effects immediately (within seconds). Other treatments lower K+ but take longer. This is the emergency step.

Key References

- KDIGO Clinical Practice Guidelines for AKI and CKD (most current)
- Brenner & Rector's The Kidney, 11th Edition

- National Kidney Foundation Kidney Disease Statistics (annual)
- UpToDate: "Evaluation of acute kidney injury in hospitalized adults"

Related Student Resources

- nephrology glossary student handout — 100 key terms explained
- structure of renal disease student handout — Framework for organizing renal pathology
- nephrology study topics guide — Curated UpToDate reading list

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Last updated: February 2026. This handout covers foundational material suitable for PA students, medical students, and early-stage nephrology rotations.