

# Essential Nephrology Glossary: 100 Terms for Student Success

Andrew Bland, MD, FACP, FAAP

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## Essential Nephrology Glossary: 100 Terms for Student Success

### Learning Objectives

By the end of this glossary, you will: - Understand 100 essential nephrology terms used in clinical practice - Recognize terminology across glomerular disease, tubular dysfunction, and kidney failure - Apply correct definitions in clinical case discussions - Use this as a quick reference during rounds and study sessions

### Introduction

This glossary provides precise, clinically-relevant definitions of nephrology terminology. Terms are organized alphabetically with clinical correlations and cross-references to support comprehensive understanding.

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### A

**Acidosis:** Condition of blood pH <7.35 with increased acidity in tissues. In kidney disease, **renal tubular acidosis** occurs when kidneys fail to excrete sufficient acid or reabsorb sufficient bicarbonate, causing metabolic acidosis despite normal anion gap.

**Acute Interstitial Nephritis (AIN):** Inflammatory condition of kidney interstitium and tubules, typically drug-induced (NSAIDs, antibiotics, PPIs) or infection-related. Presents with AKI, fever, rash, and urinary eosinophils. Often reversible if offending agent removed promptly.

**Acute Kidney Injury (AKI):** Sudden decline in kidney function over hours to days, defined by KDIGO criteria: Scr rise  $\geq 0.3$  mg/dL, Scr 1.5x baseline, or oliguria. Classified as prerenal (hypoperfusion), intrinsic (direct kidney damage), or postrenal (obstruction).

**Acute Tubular Necrosis (ATN):** Damage to tubular epithelial cells from ischemia or nephrotoxins, causing AKI. Most common cause of intrinsic AKI. FeNa typically >2%. Often reversible with appropriate management.

**Albuminuria:** Presence of albumin in urine indicating glomerular permeability defect. Microalbuminuria (30-300 mg/24h) may be earliest sign of diabetic nephropathy; macroalbuminuria (>300 mg/24h) indicates significant glomerular disease.

**Aldosterone:** Mineralocorticoid hormone from adrenal cortex regulating sodium reabsorption and potassium secretion in distal nephron. Increased in volume depletion and hyperkalemia; suppressed by volume expansion and hypokalemia.

**Alkalosis:** Blood pH >7.45 with decreased acidity. Metabolic alkalosis (elevated  $\text{HCO}_3^-$ ) common in diuretic use or GI losses; respiratory alkalosis from hyperventilation. Both types impair potassium excretion.

**Alport Syndrome:** Hereditary disorder from type IV collagen mutations causing progressive glomerulonephritis, sensorineural hearing loss, and ocular abnormalities. Males more severely affected; presents with hematuria and progressive CKD.

**Amyloidosis (Renal):** Disorder with extracellular deposition of fibrillar protein (AL amyloid from light chains, AA amyloid from serum amyloid A) in kidney tissue. Causes nephrotic syndrome and progressive kidney dysfunction; treated by addressing underlying process.

**Anion Gap:** Calculated value [ $\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$ ] helping differentiate metabolic acidosis causes. Normal 8-12 mEq/L. Elevated anion gap suggests organic acid accumulation (lactic, ketoacids, uremia, toxins).

**Anti-GBM Disease:** Autoimmune disorder with antibodies against glomerular basement membrane causing rapidly progressive glomerulonephritis. Often presents with pulmonary hemorrhage (Goodpasture syndrome). Requires urgent plasma exchange.

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## B

**Bartter Syndrome:** Inherited tubulopathy affecting thick ascending limb (mimics chronic loop diuretic use). Features: hypokalemia, metabolic alkalosis, **normal blood pressure** (differentiates from Gitelman). Salt wasting and elevated renin with normal/low BP.

**Bowman's Capsule:** Cup-shaped epithelial structure surrounding glomerular capillary network. Collects ultrafiltrate from glomerulus; forms initial tubule. Parietal layer lines outer surface; visceral layer forms podocytes.

**BUN (Blood Urea Nitrogen):** Nitrogenous waste product of protein metabolism; normal 7-20 mg/dL. Elevated in kidney disease but influenced by protein intake, liver function, and catabolism. BUN:Cr ratio helps classify azotemia (prerenal >20:1, intrinsic 10-15:1).

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## C

**Calcitriol:** Active form of vitamin D (1,25-dihydroxycholecalciferol) produced in kidney proximal tubule. Essential for intestinal calcium absorption and bone mineralization. Decreased production in CKD contributes to renal osteodystrophy.

**Central Venous Catheter:** Temporary vascular access placed in internal jugular, subclavian, or femoral vein for hemodialysis when permanent access unavailable. Higher infection risk than AVF; used during transition to permanent access.

**Chronic Kidney Disease (CKD):** Progressive irreversible decline in kidney function staged 1-5 by eGFR. Stage 5 = kidney failure (eGFR <15). Complications include HTN, bone disease, anemia, metabolic acidosis.

**Clearance:** Volume of plasma completely cleared of a substance per unit time (mL/min). Creatinine clearance approximates GFR. Substance must be freely filtered and not reabsorbed or secreted for accurate GFR estimation.

**Collecting Duct:** Final portion of nephron where fine regulation of sodium, potassium, and water balance occurs under hormonal control. Principal cells respond to aldosterone and ADH; intercalated cells regulate acid-base balance.

**Continuous Renal Replacement Therapy (CRRT):** Slow, continuous dialysis form used in critically ill patients with hemodynamic instability. Better solute removal than peritoneal dialysis; permits fluid removal without hypotension.

**Countercurrent Multiplication:** Mechanism in loop of Henle creating osmotic gradient in renal medulla enabling urine concentration. Descending limb: water permeable, solute impermeable. Ascending limb: solute reabsorption, water impermeable.

**Creatinine:** Waste product of muscle metabolism used to estimate kidney function. Normal: men 0.7-1.3 mg/dL, women 0.6-1.1 mg/dL. Production relatively constant but influenced by muscle mass, age, medications. Less reliable in acute changes.

**Cystatin C:** Low molecular weight protein produced by all nucleated cells, freely filtered by glomerulus. Serum level as alternative GFR marker less affected by muscle mass than creatinine; useful in elderly and children.

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## D

**Diabetic Nephropathy:** Kidney disease from diabetes mellitus; leading cause of ESRD in developed countries. Progressive: microalbuminuria  $\square$  overt proteinuria  $\square$  declining GFR. SGLT2i and GLP-1 agonists now standard preventive therapy.

**Dialysate:** Solution containing water, electrolytes, and buffers used in dialysis. Composition matches or differs from plasma to achieve desired concentration gradients. Waste products move from blood to dialysate via diffusion.

**Dialysis Adequacy:** Measure of dialysis effectiveness in waste product removal. Assessed by Kt/V (urea clearance  $\times$  time/volume of distribution; target >1.2) or urea reduction ratio (target >65%). Inadequate dialysis linked to worse outcomes.

**Dialysis Disequilibrium Syndrome:** Neurological disorder during or after hemodialysis from rapid osmotic shifts. Presents with headache, nausea, restlessness; severe cases include seizures or coma. Prevented by slower dialysis in first sessions.

**Distal Convoluted Tubule (DCT):** Nephron segment between loop of Henle and collecting duct where fine electrolyte balance occurs. Thiazides block sodium-chloride cotransporter here; parathyroid hormone regulates calcium reabsorption.

**Diuretic:** Medication increasing urine production. Classes: loop diuretics (furosemide—block thick ascending limb), thiazides (block DCT), potassium-sparing (block collecting duct), osmotic

(mannitol—increases filtrate osmolality).

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## E

**Edema:** Swelling from excess fluid in body tissues. Common in nephrotic syndrome (hypoalbuminemia), heart failure (poor venous return), and liver disease (portal hypertension). Grade 1 (barely visible) through 4 (pitting edema).

**Electrolytes:** Ions in body fluids ( $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Cl}^-$ ,  $\text{HCO}_3^-$ ,  $\text{Ca}^{2+}$ ,  $\text{PO}_4^{3-}$ ,  $\text{Mg}^{2+}$ ) tightly regulated by kidneys. Small changes in serum concentration cause significant effects on cardiac and neurologic function.

**End-Stage Renal Disease (ESRD):** Advanced kidney failure (stage 5 CKD,  $\text{eGFR} < 15$ ) requiring renal replacement therapy (dialysis or transplantation). Patients need definitive therapy to survive.

**Erythropoietin:** Hormone produced primarily by kidney interstitial fibroblasts stimulating red blood cell production in bone marrow. Deficient production in CKD causes anemia; recombinant EPO (ESA) used therapeutically with careful hemoglobin targets.

**Estimated Glomerular Filtration Rate (eGFR):** Mathematical estimate of kidney function from serum creatinine, age, sex, and race using CKD-EPI equation (preferred). Normal  $> 90$  mL/min/1.73m<sup>2</sup>. Used for CKD staging.

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## F

**Fabry Disease:** X-linked lysosomal storage disorder from alpha-galactosidase deficiency causing progressive kidney damage. Features proteinuria, declining GFR, extrarenal manifestations (pain, skin lesions, cardiac disease). Enzyme replacement therapy available.

**Fanconi Syndrome:** Generalized dysfunction of proximal tubule causing aminoaciduria, glycosuria, phosphaturia, bicarbonaturia, and tubular proteinuria. Associated with monoclonal light chain disease, cystinosis, and medications.

**Focal Segmental Glomerulosclerosis (FSGS):** Pattern of glomerular sclerosis affecting some (focal) glomeruli and portions (segmental) of those glomeruli. Common cause of nephrotic syndrome. Associated with podocyte dysfunction; may be primary or secondary.

**Fractional Excretion of Sodium (FENa):** Percentage of filtered sodium excreted in urine:  $\text{FENa} = [(\text{UNa} \times \text{PCr}) / (\text{PNa} \times \text{UCr})] \times 100$ .  $\text{FENa} < 1\%$  suggests prerenal cause;  $\text{FENa} > 2\%$  suggests intrinsic kidney disease.

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## G

**Gitelman Syndrome:** Inherited tubulopathy affecting distal convoluted tubule (similar to thiazide use). Features: hypokalemia, metabolic alkalosis, hypomagnesemia, **hypocalciuria** (reduces kidney stones vs. Bartter). Normal/low BP.

**Glomerular Filtration Rate (GFR):** Volume of fluid filtered by glomeruli per minute; best measure of overall kidney function. Normal ~100-120 mL/min/1.73m<sup>2</sup>. Declines naturally ~1 mL/min/year with aging.

**Glomerulonephritis:** Inflammation of glomeruli often immune-mediated. Presents with hematuria, proteinuria, reduced kidney function. Classified by histology (proliferative, membranous, crescentic). Requires immunosuppression or plasma exchange in severe cases.

**Glomerulus:** Network of specialized capillaries in Bowman's capsule where ultrafiltration occurs. Comprises three layers: fenestrated endothelium, basement membrane, podocytes. Damaged in most primary kidney diseases.

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## H

**Hematuria:** Presence of blood in urine; either gross (visible) or microscopic (<5 RBC/hpf is normal; >5 is abnormal). Dysmorphic RBCs and RBC casts indicate glomerular source; uniform RBCs suggest lower urinary tract.

**Hemolytic Uremic Syndrome (HUS):** Microangiopathic hemolytic anemia + thrombocytopenia + AKI, often following Shiga toxin-producing bacterial infection (e.g., E. coli O157:H7). Treated supportively; plasma exchange in atypical HUS.

**Hemodialysis:** Procedure removing waste products and excess fluid from blood using semipermeable membrane. Blood circulates through dialyzer where diffusion and ultrafiltration remove uremic toxins. Typically 3-4 hours, 3x/week.

**Henle's Loop:** U-shaped nephron segment between proximal and distal tubules. Descending limb permeable to water; ascending limb permeable to solutes. Creates medullary osmotic gradient essential for urine concentration.

**Hydronephrosis:** Dilation of renal pelvis and calyces from urine outflow obstruction. Can be unilateral or bilateral. Risks: UTI, pain, progressive kidney damage if prolonged. Diagnosed by ultrasound or CT.

**Hyperkalemia:** Elevated serum potassium (>5.0 mEq/L). ECG changes: peaked T waves □ widened QRS □ loss of P wave □ sine wave. Caused by decreased renal excretion, cellular shift, or increased intake.

**Hypernatremia:** Elevated serum sodium (>145 mEq/L) indicating water deficit. Causes: inadequate free water intake, excessive losses (diabetes insipidus), or sodium excess. Water replacement must be gradual.

**Hyperphosphatemia:** Elevated serum phosphate common in CKD from decreased renal excretion. Contributes to secondary hyperparathyroidism and renal osteodystrophy. Managed with dietary restriction, binders, and dialysis.

**Hypertension:** Elevated blood pressure; both risk factor for kidney disease and consequence of it. Tight BP control (especially in CKD) reduces proteinuria and slows GFR decline. RAAS inhibitors first-line.

**Hypokalemia:** Low serum potassium (<3.5 mEq/L). Cardiac effects: U waves, flattened T waves, ST depression, arrhythmias. Common from diuretics, GI losses, cellular shifts. Magnesium must

be repleted for correction.

**Hyponatremia:** Low serum sodium (<135 mEq/L) representing water excess. Classified by volume status: hypovolemic (GI losses), euvolemic (SIADH), hypervolemic (CHF, cirrhosis, renal failure). Acute vs. chronic guides correction rate.

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## I

**IgA Nephropathy:** Most common primary glomerulonephritis worldwide from IgA immune complex deposition in glomerular mesangium. Presents with hematuria; variable proteinuria and progression. Treated with ACEi/ARB, corticosteroids in severe cases.

**Interstitial Nephritis:** Inflammation of kidney tissue between tubules, commonly medication-induced (NSAIDs, antibiotics, PPIs). Presents with AKI, fever, rash, eosinophiluria. Often reversible if offending agent removed promptly.

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## J

**Juxtaglomerular Apparatus:** Specialized structures at vascular pole of glomerulus containing: granular cells (secreting renin), macula densa (sensing sodium delivery), and extraglomerular mesangial cells. Responds to decreased renal perfusion by releasing renin.

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## K

**KDIGO Guidelines:** Kidney Disease: Improving Global Outcomes—international consensus clinical practice guidelines for evaluation, classification, and management of kidney disease. References standard AKI staging, CKD classification, GN treatment.

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## L

**Loop Diuretics:** Medications (furosemide, bumetanide, torsemide) blocking sodium-potassium-chloride cotransporter in thick ascending limb of loop of Henle. Potent diuretics with hypokalemia and hyperuricemia risks.

**Lupus Nephritis:** Kidney involvement in systemic lupus erythematosus causing immune complex glomerulonephritis. Classified into six WHO classes (I-VI) with varying prognosis. Treated with immunosuppression (mycophenolate, cyclophosphamide).

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## M

**Macula Densa:** Specialized cells in distal tubule near glomerulus involved in tubuloglomerular feedback. Senses sodium chloride delivery; signals juxtaglomerular cells. Plays critical role in renin-angiotensin-aldosterone system activation.

**Membranoproliferative Glomerulonephritis (MPGN):** Pattern of glomerular injury with mesangial proliferation and capillary wall thickening. Associated with viral infections (hepatitis C), systemic diseases (lupus), and immune complex deposition.

**Membranous Nephropathy:** Common cause of nephrotic syndrome in adults characterized by uniform glomerular basement membrane thickening from subepithelial immune deposits. May be primary (often phospholipase A2 receptor positive) or secondary (malignancy, infection, medication).

**Mesangial Cells:** Specialized cells in glomerulus providing structural support and contractile regulation. Also phagocytose immune complexes and produce inflammatory mediators. Proliferation occurs in various glomerulonephritides (IgA, MPGN, lupus).

**Metabolic Acidosis:** Low blood pH (<7.35) from decreased bicarbonate. Common in advanced kidney disease from impaired ammonia production and hydrogen ion excretion. Anion gap elevated in uremia.

**Microalbuminuria:** Small amounts of albumin in urine (30-300 mg/24h). Early marker of kidney damage and cardiovascular risk. Presence predicts progression to overt proteinuria and ESRD, especially in diabetes.

**Minimal Change Disease:** Most common cause of nephrotic syndrome in children, characterized by normal-appearing glomeruli on light microscopy but **foot process effacement** on electron microscopy. Highly responsive to corticosteroids.

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## N

**Nephrectomy:** Surgical removal of kidney, either partial (nephron-sparing) or complete. Indicated for renal cancer, severe infection, trauma, or donation. After unilateral nephrectomy, remaining kidney undergoes compensatory hypertrophy.

**Nephritic Syndrome:** Clinical presentation with hematuria, proteinuria (typically <3.5 g/day), hypertension, and reduced GFR. Typically caused by glomerulonephritis. Differs from nephrotic syndrome by lower proteinuria and RBC casts.

**Nephron:** Functional unit of kidney consisting of glomerulus and tubular system. Each kidney ~1 million nephrons. Specialization of different nephron segments allows coordinated filtration and reabsorption functions.

**Nephrosclerosis:** Hardening of renal vessels and parenchyma from chronic hypertension or aging, causing progressive kidney function loss. “Benign nephrosclerosis” from HTN; “malignant nephrosclerosis” from severe HTN with acute kidney damage.

**Nephrostomy Tube:** Percutaneous catheter placed directly into kidney pelvis to drain urine when normal outflow obstructed. Temporary measure; allows time for definitive treatment of obstruction (stone, stricture, malignancy).

**Nephrotic Syndrome:** Clinical syndrome with heavy proteinuria (>3.5 g/24h), hypoalbuminemia, lipiduria (fatty casts, oval fat bodies), edema, and hyperlipidemia. Often caused by membranous disease, FSGS, or minimal change disease.

**Nephrotoxicity:** Kidney damage from medications, toxins, or substances. Common agents: aminoglycosides, amphotericin B, NSAIDs, ACEi/ARB (in specific settings), contrast dye, cisplatin. Prevention via adequate hydration and dose adjustment.

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## O

**Oliguria:** Reduced urine output (<400 mL/24h in adults; <0.5 mL/kg/hr). Common in acute kidney injury, severe dehydration, or advanced CKD. Ominous sign requiring urgent evaluation and management.

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## P

**Parathyroid Hormone (PTH):** Regulates calcium and phosphate homeostasis. Levels increase in CKD as compensatory response to hypocalcemia and hyperphosphatemia. Elevated PTH drives renal osteodystrophy.

**Peritoneal Dialysis:** Dialysis modality using peritoneum as semipermeable membrane. Dialysate infused into peritoneal cavity; diffusion and ultrafiltration remove waste. More gradual than hemodialysis; permits greater residual kidney function.

**Podocyte:** Specialized epithelial cell in visceral layer of Bowman's capsule forming filtration slits. Foot processes interdigitate to create slit diaphragm preventing protein leakage. Damaged in most nephrotic syndromes.

**Polycystic Kidney Disease (PKD):** Hereditary disorder characterized by progressive multiple renal cysts. Autosomal dominant (ADPKD) most common; autosomal recessive (ARPKD) presents in infancy. Leads to progressive kidney enlargement and failure.

**Post-Streptococcal Glomerulonephritis (PSGN):** Immune-mediated glomerulonephritis following streptococcal infection. Classically presents 1-2 weeks after strep pharyngitis with hematuria, proteinuria, HTN. Usually self-limited with complete recovery.

**Potassium:** Principal intracellular cation with serum normal range 3.5-5.0 mEq/L tightly regulated by kidneys. Aldosterone increases renal potassium excretion; beta-blockers and ACEi/ARBs decrease it.

**Proteinuria:** Abnormal amount of protein in urine (normal <150 mg/24h). Indicates kidney damage. Quantified as 24-hour urine protein or spot protein-to-creatinine ratio. Marker of disease severity and progression.

**Proximal Convoluted Tubule (PCT):** First portion of renal tubule where ~65% of filtered sodium, water, glucose, amino acids, and phosphate are reabsorbed. Damaged first in ATN and interstitial nephritis.

**Pyelonephritis:** Bacterial infection of kidney parenchyma and renal pelvis. Presents with flank pain, fever, costovertebral angle tenderness, pyuria. Can progress to sepsis if untreated. Requires urgent antibiotic therapy.

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## R

**Rapidly Progressive Glomerulonephritis (RPGN):** Severe form of glomerulonephritis with rapid loss of kidney function over days to weeks. Characterized by crescent formation on biopsy. Classified by immunofluorescence: anti-GBM, immune complex, ANCA-associated.

**Renal Artery Stenosis:** Narrowing of renal artery (atherosclerotic or fibromuscular dysplasia) causing renovascular hypertension and ischemic nephropathy. Diagnosed by duplex ultrasound, CTA, or MRA. Treatment: balloon angioplasty or stent.

**Renal Biopsy:** Procedure obtaining kidney tissue via percutaneous needle (ultrasound-guided) for diagnostic evaluation. Enables light microscopy, immunofluorescence, and electron microscopy. Standard for diagnosis of proteinuria and hematuria of unclear cause.

**Renal Osteodystrophy:** Bone disease from CKD secondary to altered calcium, phosphorus, and vitamin D metabolism. Features: high-turnover bone (secondary hyperparathyroidism), low-turnover bone (from excessive PTH suppression), mixed disease.

**Renal Replacement Therapy (RRT):** Treatments partially replacing kidney function in ESRD: hemodialysis, peritoneal dialysis, kidney transplantation. Choice depends on residual kidney function, patient preference, vascular access, and contraindications.

**Renal Tubular Acidosis (RTA):** Group of disorders characterized by kidney inability to maintain normal acid-base balance. Type 1 (distal): impaired H<sup>+</sup> secretion; Type 2 (proximal): impaired HCO<sub>3</sub><sup>-</sup> reabsorption; Type 4: hyporenin-hypoaldosteronism.

**Renin-Angiotensin-Aldosterone System (RAAS):** Hormone system regulating blood pressure, sodium balance, and fluid volume. Activation: low renal perfusion → renin release → angiotensin II → aldosterone → sodium retention and vasoconstriction.

**Rhabdomyolysis:** Breakdown of skeletal muscle releasing myoglobin into circulation. Myoglobin is toxic to tubular cells, causing AKI through direct toxicity and cast formation. Causes: statins, excessive exercise, crush injury, malignant hyperthermia.

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## S

**Secondary Hyperparathyroidism:** Excessive parathyroid hormone production secondary to hypocalcemia and hyperphosphatemia, common in CKD. Drives renal osteodystrophy. Managed with vitamin D, phosphate binders, calcimimetics, parathyroidectomy if severe.

**Sodium:** Principal extracellular cation responsible for maintaining extracellular fluid volume and osmolality. Kidneys regulate total body sodium (through RAAS) and water balance (through ADH) to maintain homeostasis.

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## T

**Thin Basement Membrane Disease:** Benign condition with glomerular basement membrane thinning. Presents with persistent microscopic hematuria; stable kidney function. Excellent prognosis; no treatment required. Requires kidney biopsy to distinguish from IgA nephropathy.

**Tubuloglomerular Feedback:** Mechanism where macula densa senses sodium chloride delivery to distal tubule, signaling juxtaglomerular cells. Controls renin secretion and afferent arteriolar vasoconstriction, auto-regulating glomerular filtration.

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## U

**Urea:** Nitrogenous waste product of protein metabolism. Serum concentration measured as BUN (blood urea nitrogen). Elevated in kidney disease but non-specific; influenced by protein intake, liver function, catabolism.

**Urea Reduction Ratio (URR):** Measure of hemodialysis adequacy calculated as percentage reduction in blood urea during session. Target  $\geq 65\%$ . Used clinically alongside Kt/V; URR  $>75\%$  generally indicates adequate dialysis.

**Uremia:** Clinical syndrome of advanced kidney failure from accumulation of nitrogenous waste products and uremic toxins. Features: uremic pericarditis, encephalopathy, peripheral neuropathy, platelet dysfunction. Reversed by dialysis or transplantation.

**Urinalysis:** Examination of urine for physical, chemical, and microscopic properties. Essential diagnostic tool in kidney disease providing information about glomerular function, tubular integrity, infections, and metabolic disorders.

**Urinary Casts:** Cylindrical structures formed in renal tubules from Tamm-Horsfall protein. Visible on microscopic urinalysis. Different types indicate different pathologies: RBC casts = glomerulonephritis, WBC casts = infection/inflammation, granular = tubular damage.

**Urinary Tract Infection (UTI):** Infection involving any part of urinary system caused by bacteria (mostly E. coli). Lower UTI (cystitis): dysuria, frequency; upper UTI (pyelonephritis): fever, flank pain. Diagnosed by urinalysis and culture.

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## V

**Vascular Access:** Methods to access bloodstream for hemodialysis: arteriovenous fistula (native, preferred), arteriovenous graft (synthetic), central venous catheter (temporary). Adequate access crucial for dialysis adequacy; fistulas have longest survival.

**Vesicoureteral Reflux:** Abnormal retrograde flow of urine from bladder to ureters and kidneys. Predisposes to recurrent UTIs and scarring. Classified grade 1-5 by cystography. Lower grades often resolve spontaneously; higher grades may require surgical intervention.

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## Cross-Reference Clinical Correlations

**For Acute Kidney Injury:** See AKI, ATN, AIN, prerenal, postrenal, intrinsic, FENa  
**For Chronic Kidney Disease:** See CKD, ESRD, eGFR, creatinine, proteinuria, RTA  
**For Electrolyte Disorders:** See hyperkalemia, hypokalemia, hyponatremia, hypernatremia  
**For Glomerular Disease:** See glomerulonephritis, nephrotic syndrome, nephritic syndrome, RPGN  
**For Renal Replacement:** See hemodialysis, peritoneal dialysis, RRT, CRRT

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## Study Tips

1. **Group by pathophysiology:** Organize terms by body system affected (electrolytes, filtration, acid-base)
  2. **Use clinical cases:** Create patient scenarios requiring terminology knowledge
  3. **Make flashcards:** Front = term, back = definition + one clinical correlation
  4. **Connect to treatment:** Each disorder suggests specific management; learn together
  5. **Use in rounds:** Practice explaining patient findings using precise terminology
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## Key References

- Brenner & Rector's The Kidney, 11th Edition
  - Comprehensive Clinical Nephrology (Feehally, Floege, Tonelli)
  - KDIGO Clinical Practice Guidelines (current)
  - UpToDate Nephrology Topic Reviews
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*This glossary complements clinical education and rounds discussions. Use as quick reference during patient encounters and study sessions.*