→ Acid-base , minerals

Table 4-4: Evaluating Acid-Base Disorders
The 4-Step Method

Step	Questions	How to Determine Answer	What it Means
1) pH	Determine serum pH	Look at ABG results	pH > 7.45 = alkalemia pH < 7.35 = acidemia
2) Anion Gap	a) What is the anion gap?	$Na^+ - Cl^- - HCO_3^-$	If AG is > 13 = HAGMA
	b) What is change in AG? (measured – normal)	AG – 10	Use in Step 3
3) HCO_3	a) What is expected HCO_3^-?	25 – (ΔAG)	If actual HCO_3 > expected HCO_3 = metabolic alkalosis
	b) What is the change in HCO_3^-?		If actual HCO_3 < expected HCO_3 = NAGMA
4) pCO_2	a) What is expected pCO_2?	15 + measured HCO_3^-	If actual pCO_2 > expected pCO_2 = respiratory acidosis
	b) What is the change in pCO_2? (expected – measured)		If actual pCO_2 < expected pCO_2 = respiratory alkalosis

Table 4-3: HAGMAs and NAGMAs

Common Causes of HAGMA

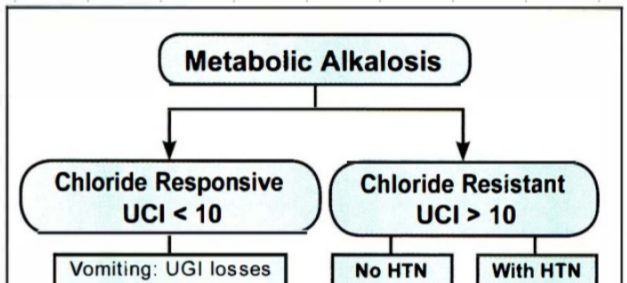
Severe CKD: decreased acid (especially NH_4) excretion—most common
Uremia: sulfate, phosphate, urate
Ketoacidosis: diabetic, alcoholic, starvation
Lactic acidosis: drugs, toxins, circulatory compromise
Poisonings: salicylates, methanol, ethylene glycol, propylene glycol

Common Causes of NAGMA

Renal tubular acidosis
Diarrhea
Carbonic anhydrase inhibitors
Hyperalimentation with TPN

Table 4-2: AG and OG in the Obtunded Patient

Anion Gap	Osmolal Gap	Consider
High	Very High	Methanol and ethylene glycol; ketoacidosis and lactic acidosis; chronic kidney disease
High	High	Ketoacidosis and lactic acidosis; chronic kidney disease
High	Normal	Salicylate poisoning; methanol or ethylene glycol ingestion after substrates have been converted to acid metabolites.
Normal	High	Isopropyl alcohol, acetone, or ethanol ingestion
Normal	Normal	Think carbon monoxide poisoning—before lactic acidosis develops.



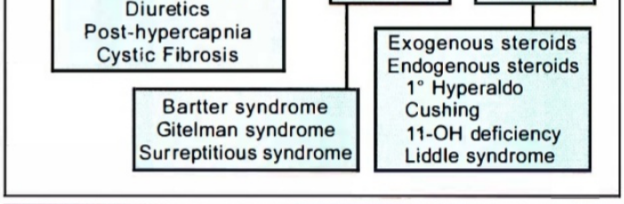


Figure 4-1: Metabolic Alkalosis: Chloride Responsive vs. Chloride Resistant Causes

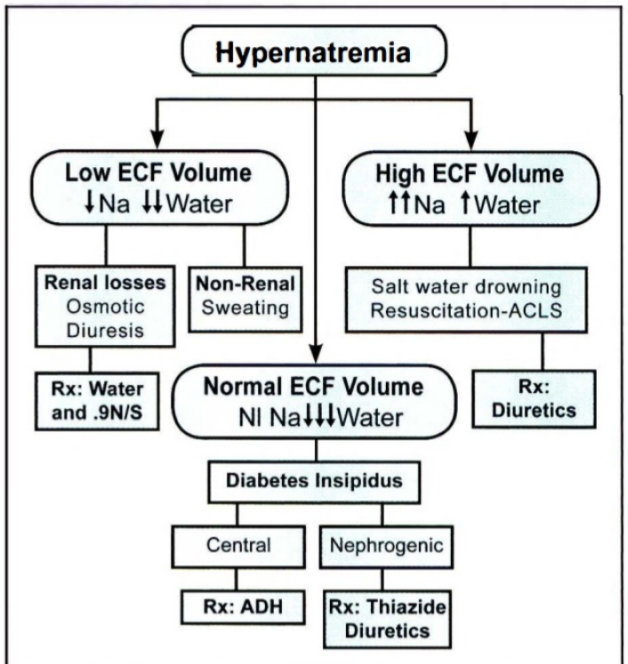


Figure 4-3: Causes of Hypernatremia

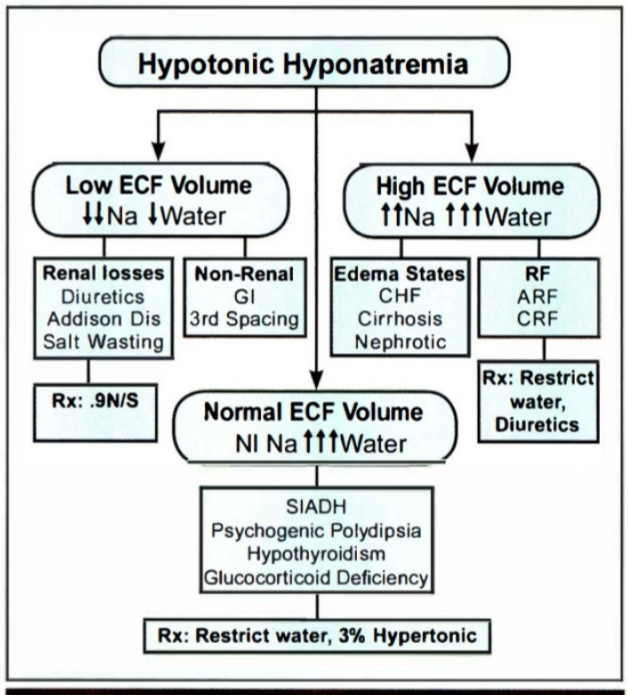


Figure 4-2: Causes of Hypotonic Hyponatremia

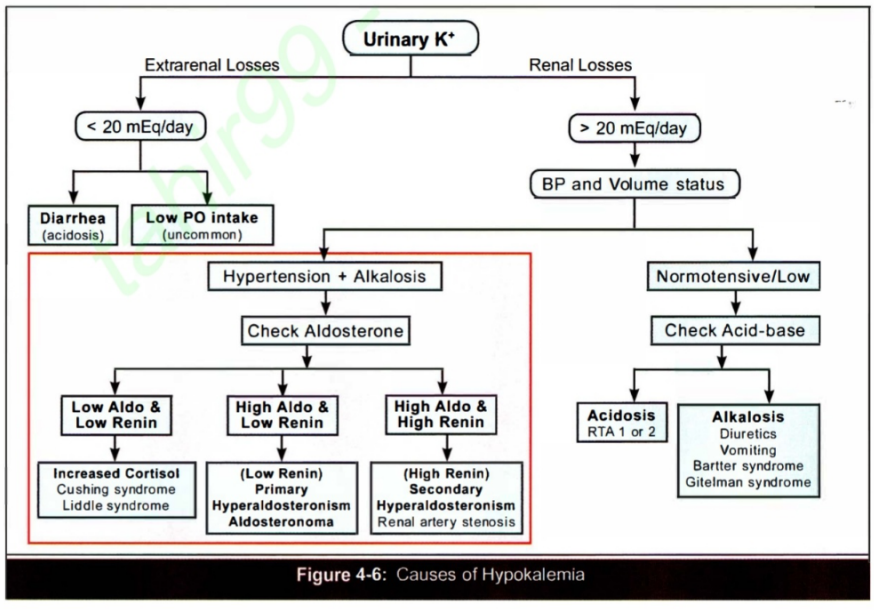


Figure 4-6: Causes of Hypokalemia

Table 4-8: Comparison of Bartter and Gitelman Syndromes

	Bartter Syndrome	Gitelman Syndrome
Onset	Infants/Children	Early adults
Low K, metabolic alkalosis	+	+
Low Mg	+	+
Hypocalciuria	–	+
Chondrocalcinosis	–	+
Defect	Ascending loop	Distal convoluted tubule
Looks like patient is taking …	A loop diuretic	A thiazide diuretic

RTAs.

Table 4-6: Renal Tubular Acidoses (RTAs)

	Type of Acidosis	Urine pH	Serum K^+	Misc.	Mechanism	Main Causes
Type 1 Distal	NAGMA (severe, but responds to HCO_3 treatment)	> 5.5	Low-nl	Stones, nephrocalcinosis	In intercalated cells: Decreased H^+ secretion in late distal tubule and cortical collecting duct	Autoimmune (SLE, Sjögren's, RA) Hereditary hypercalciuria Drugs (amphotericin B, lithium)
Type 2 Proximal	NAGMA (mild, but difficult to correct with HCO_3 treatment)	Varies	Low-nl	Fanconi's	Decreased resorption of HCO_3^- in proximal tubule	MM Acetazolamide Amphotericin B Heavy metals Amyloidosis
Type 4 Distal	NAGMA (mild, responds to HCO_3 treatment)	< 5.5	High	Diabetes	In principal cells: Decreased Na^+–K^+ exchange in late distal tubule and cortical collecting duct; reduced NH_4^+ excretion	Diabetic nephropathy Chronic interstitial nephritis NSAIDs ACEI Obstructive uropathy Spironolactone

Table 4-7: RTAs — Serum and Urine Chemistry

	Plasma				Urine		
	Na^+	K^+	Cl^-	HCO_3^-	pH	K^+	Na^+
Normal	135–145	3.5–5	95–105	22–30	Variable	25–100	100–260
A	140	2.6	113	17	7.9	50	100
B	140	5.5	117	13	6	50	100
C	140	4.0	115	15	6	50	100
D	140	4.0	105	15	6	50	100
E	140	4.0	115	15	6	10	10

HTN & drugs

Table 4-10: Identifiable Causes of Hypertension

Labile HTN, medullary thyroid cancer, primary hyperparathyroidism (MEN2)	Pheochromocytoma
Continuous abdominal bruit	Renovascular HTN
Decreased BP in lower extremities or absent/delayed femoral pulses	Coarctation of the aorta
Abdominal or flank masses	Polycystic kidneys
Elevated creatinine or	Renal parenchymal

abnormal urinalysis	disease
Hypercalcemia	Hyperparathyroidism Granulomatous disease
Hypokalemia	Hyperaldosteronism

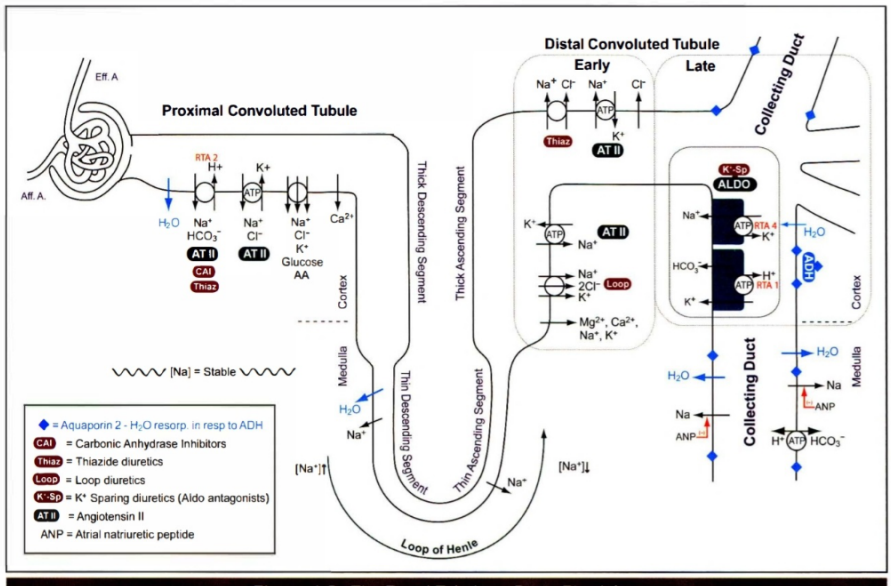


Figure 4-5: The Renal Tubule — Basic Physiology

→ AKI

Table 4-11: Acute Kidney Injury Labs and Clues

Category	Causes	FE_{Na}	$FE_{Uric\ acid}$	FE_{Urea}	U_{Osm}	Urine Na^+	Urine Sediment	Suspect in Patient with ...
Prerenal	Volume depletion Decreased EABV* NSAIDs ACEI	< 1%	< 12%	< 35%	> 400 mOsm/L	< 20	Normal Granular casts Hyaline casts	Bleeding CHF Cirrhosis/hepatorenal Abdominal compartment syndrome (ACS) Nephrotic syndrome GI fluid loss (nausea/vomiting/diarrhea)
Intrinsic renal	Diseases of, or damage to, the glomeruli, tubules, or interstitium	ATN* > 2% GN* < 1%	> 20%	> 50%	300–350 mOsm/L	> 20	Red cell casts and/or protein (GN) Dirty brown casts (ATN*) Eos (AIN*)	Infections SLE Vasculitis Drugs (aminoglycosides, amphotericin, cisplatin, NSAIDs) Contrasts/IV dyes Atheroembolism Heroin Myeloma Diabetes HTN Hypotension, shock
Postrenal	Obstruction	Varies	Varies	Varies	Normal	Normal	Hematuria	Elderly males Colicky pain

Fractional excretion**	Level indicating prerenal AKI	Changed by diuretics
FE_{Na}	< 1	Yes
FE_{Urea}	< 35	No
$FE_{Uric\ acid}$	< 12	No

*EABV = effective arterial blood volume
ATN = acute tubular necrosis
GN = glomerulonephritis
AIN = acute interstitial nephritis
**Recent diuretics use can alter the FE_{Na} and, in this setting, FE_{Urea} and $FE_{Uric\ acid}$ are more reliable.

Glomerular disease

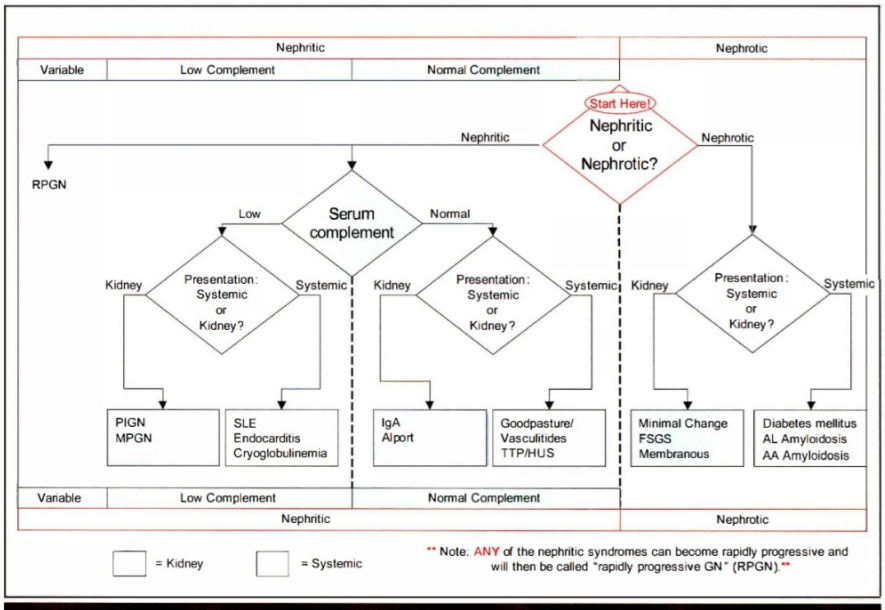


Figure 4-7: A Strategy for Workup of the Glomerular Disorders

Table 4-12: Summary Table of Glomerulonephritides — Nephritic Syndrome (1 of 2)

Classification / Complement Level		Name	Presentation	Urine	Notes	Treatment
NEPHRITIC SYNDROME Primarily kidney presentation	Low	PIGN	Post-strep (skin or throat)	Red cells Proteinuria (1–3 g/d) +/– Red cell casts +/– White cell casts	Latent period between infection and symptoms. Complements return to normal after a short period. Check for antibodies of antecedent strep infection.	Treat the infection and supportive care.
		MPGN			Complements stay low > 3 months. Primary MPGN = rare, usually assoc w/ HCV	No good Rx; steroids; frequent renal failure
	Normal	IgA (Mesangial proliferative)	Classic presentation: gross hematuria with viral illness (no latent period)		Best prognosis for pts with intermittent hematuria, may progress in pts with proteinuria	If proteinuria or reduced GFR: ACEIs or ARBs, Steroids
		Alport	GN, deafness, congenital eye disease		X-linked recessive most common	ACEIs or ARBs
		ANCA + renal-limited vasculitis	Rapidly progressive GN, HTN		Nonspecific presentation	High-dose steroids with cytotoxics
NEPHRITIC SYNDROME Systemic presentation	Low	Immune complex GN	SLE Endocarditis Cryoglobulinemia/ HCV/MPGN HBV		MPGN pathology common	Treat the cause.
	Normal		Vasculitis: (microscopic polyangiitis, granulomatous polyangiitis) TTP/HUS		Symptoms depend on type of vasculitis	High-dose steroids with cytotoxics TTP: + Plasmapheresis
NEPHRITIC SYNDROME RPGN	Variable	1) Anti-GBM 2) Immune complex 3) Pauci-immune (usually + ANCA)	Acute, rapidly progressive, renal failure +/– signs of systemic disease (depending on cause)		Requires urgent renal biopsy that documents glomerular crescents and demonstrates type of antibodies; check ANCA and anti-GBM ab; consider ANA, anti-dsDNA, cryoglobulins, anti-streptococcal Ab	High-dose methylprednisone → prednisone + cytotoxics +/– plasmapheresis

Table 4-13: Summary Table of Glomerulonephritides — Nephrotic Syndrome (2 of 2)

Classification	Name	Causes	Urine	Notes	Treatment
NEPHROTIC SYNDROME Primarily kidney	Minimal change disease	Sudden onset of severe nephrotic syndrome. Can be associated with Hodg-	> 3 g/d proteinuria Oval fat droplets Edema	10–15% of nephrotic syndrome in adults Only abnormal finding on bx: loss of foot	Steroids +/– cytotoxics

presentation		kin's; use of NSAIDs, lithium	Hyperlipidemia Complications: DVT bacterial infections	processes on electron microscopy	
	FSGS	Primary (idiopathic) causes most common esp. in African-Americans Secondary causes: heroin use, HIV/AIDS, reflux nephropathy, obesity		Pts who respond to steroids have best prognosis	Steroids Cyclosporine ACEIs or ARBs
	Membraneous	Most common cause is idiopathic. Secondary causes: NSAIDs, chronic HBV, solid tumors, SLE		Idiopathic may be associated with antibodies to the PL2receptor	Mild cases: no treatment Treat underlying disease Steroids +/– cytotoxics ACEIs or ARBs
NEPHROTIC SYNDROME Systemic presentation	Diabetes	Diabetic with nephrotic -range proteinuria		Most common secondary cause of nephrotic syndrome; eye disease usually precedes kidney disease.	ACEIs or ARBs +/– low-protein diet; control of blood glucose, BP, and lipids
	AA amyloidosis AL amyloidosis	Injection drug use Multiple myeloma		Look for other clues to myeloma: hypercalcemia, anemia, obtain SPEP and UPEP and measure serum free light chains	Treat underlying disease.

Glomerular diseases associated with low complement levels: Postinfectious GN, lupus nephritis (SLE), cryoglobulinemia, MPGN (hepatitis C and B), infective endocarditis