DISORDERS OF ELECTROLYTE (CATION) HANDLING

Electrolyte Disorder	Etiology	Pathophysiology and Presentation	Lab Findings and Diagnosis	Treatment
HYPOKALEMIA	**Transcellular Shifts** Primary metabolic or respiratory alkalosis Insulin Catecholamines Familial hypokalemic periodic paralysis **GI Loss** Reduced intake Emesis, NG tube (increased HCO3- filtration and activation of RAAS) Diarrhea (direct) **Cutaneous Loss** Diaphoresis Burns **Renal Loss** Diuretic therapy (Loops + Thiazides) Primary Hyperaldosteronism Secondary Hyperaldosteronism Type I RTA: impaired H+ secretion Type II RTA: impaired HCO3- reabsoprtion DKA	**Neuromuscular Dysfunction** Muscle weakness, myalgia, ischemia (loss of local K+ vasodilation), rhabdomyolysis **Cardiac Dysfucntion** Hyperpolarization → delayed ventricular repolarization PVCs, unstable ventricular tachyarrhythmias Digitalis toxicity **Metabolic Effects** Glucose intolerance (suppression of insulin) Failure to thrive **Renal** Polydipsia + Polyuria Medullary gradient collapse → Polyuria Nephrogenic DI Ammonia excess → metabolic alkalosis PCT and DCT vacuolization → progressive renal failure	Serum K+ < 3.5 mEq/L Urinary: FEK > 10% → RENAL LOSS FEK < 6% → NON-RENAL LOSS EKG: ST depression, flat T waves, prominent U waves, prolonged QT interval	**Hypokalemic + Hypochloremic Metabolic Alkalosis**: IV KCl at 10 – 20 mmol/hr **Hypokalemia + Metabolic Acidosis**: KHCO3, K+ Citrate
HYPERKALEMIA	**Transcellular Shifts** *Primary* metabolic or respiratory acidosis Hyperglycemia (osmotic shift) β-adrenergic blockers Myocyte necrosis, surgery, trauma Transfusion with stored whole blood **GI Excess** Increased intake **IV K+ therapy** **IV Heparin**: antagonizes the release of aldosterone	**Neuromuscular Dysfunction** Muscle weakness and paralysis **Cardiac Dysfunction** Depolarization → Na+ accomodation → reduced myocardial excitability → delayed atrial and ventricular depolarization + rapid repolarization **Metabolic Effects** NML AG metabolic acidosis	Serum K+ > 5.0 mEq/L EKG: peaked T-waves, effaced P waves, widened QRS, monomorphic VTach	First-line: calcium gluconate to stabilize cardiac muscle Insulin + glucose Infuse bicarbonate B2-agnosist nebulizer Cation exchangers + sorbitol Dialysis

	Renal Retention Renal failure Addison's Disease (aldosterone deficiency) *Type IV RTA: Hyporeninemic Hypoaldosteronism* Decreased aldosterone → decreased K+ secretion → intracellular alkalosis → decreased NH3 → metabolic *acidosis* **Drugs** ACE inhibitors, ARBs, NSAIDs, K+-sparing diuretics			
HYPONATREMIA + HYPOVOLEMIA	GI Loss: emesis, diarrhea Renal Loss: diuretics, osmotic dieresis Aldosterone deficiency (Addison's disease) Interstitial edema (bowel obstruction, pancreatitis, burns) ECF depletion → secretion of ADH → increased water retention in medullary CD → incomplete volume expansion → hyponatremia Decreased ECF volume Increased ICF volume	Cerebral edema with potential herniation Lethargy, confusion, stupor, personality changes Neuromuscular hyperexcitability	Serum Na+ < 135 mEq/L Urine Osm > Plasma Osm → indicates normal response to ADH Orthostatic hypotension, tachycardia	IV NML (0.9%) saline Restrict free water
HYPONATREMIA + HYPERVOLEMIA	CHF Hepatic cirrhosis Nephrotic syndrome Decreased vascular volume → decreased renal perfusion → activation of RAAS → Na+ retention (insufficient) → ECF expansion Increased ECF volume Increased ICF volume		Serum Na+ < 135 mEq/L Urine Osm > Plasma Osm	TX underlying disease Restrict free water Vasopressin receptor blockade
HYPONATREMIA + EUVOLEMIA	SIADH Paraneoplastic, head trauma,	With excessively rapid Na+ replenishment: central pontine myelinolysis (osmotic	Serum Na+ < 135 mEq/L	Restrict free water **Furosemide** → collapse

	meningitis, stroke, subdural hemorrhage Psychogenic polydipsia Hypothyroidism Cortisol deficiency NML total sodium with excess water retention NML intravascular volume Increased ICF volume	demyelination syndrome)	Urine Osm >> Plasma Osm	medullary gradient IV NML saline → replace isotonic renal loss V2 receptor antagonists
HYPERNATREMIA + HYPOVOLEMIA	GI Loss: emesis, diarrhea Burns Diaphoresis Diuretics, Osmotic Diuresis Hypotonic ECF loss → increased serum osmolarity → increased ADH secretion → renal water retention → insufficient volume expansion Hypovolemia → activation of RAAS Decreased ECF volume Increased TB Na+ Decreased TBW	Cellular shrinkage → cerebral vascular hemorrhage Confusion, increased neuromuscular excitability, seizures	Serum Na+ > 145 mEq/L Urine Osm >> Serum Osm	IV NML saline → restore intravascular volume Taper saline with BP normalization Then IV 5% dextrose solution (D5W)
HYPERNATREMIA + HYPERVOLEMIA	Hypertonic fluid intake Hyperaldosteronism			
HYPERNATREMIA + EUVOLEMIA	Central DI Trauma, neurosurgery, CNS malignancy, granuloma Nephrogenic DI X-linked inheritance Acquired: polycystic disease, obstructive uropathy, hypokalemia, hypercalcemia, pyelonephritis,		Water deprivation test to distinguish central and nephrogenic DI Fluid restriction → determine maximum urine osmolarity → subcutaneous ADH → no rise in urine osm: nephrogenic → increase: central	Low-sodium diet +Low-dose thiazides These increase the absorption of sodium → reduce diuresis Recombinant ADH

	Lithium Diaphoresis Lack of function of ADH → diuresis → normal sodium retention → normal ECF volumes with concentration of Na+		This is inaccurate with renal diseases leading to dissipated medullary gradients	