Hypoglycemia		
Treatment	 IV Dextrose: "Hawaii 5-0 Rule" 10 cc/kg bolus of D5W, 5 cc/kg bolus of D10W, 2 cc/kg bolus of D25W Glucagon (can use if no IV access and patient unable to take PO's): 0.03 mg/kg (max 1 mg) IM, IV, or subQ. Effective for hypoglycemia caused by hyperinsulinemia. Does not work if glycogen stores are depleted or w/ glycogen storage diseases 	

	Diabetes Insipidus
PowerPlan/ Ordersets	DMICU DI orderset, Endo AMB DI Plan
Definition	Failure to produce or respond to antidiuretic hormone, leading to excessive free water loss and subsequent hypernatremia.
Etiology	Central: Failure of posterior pituitary to secrete ADH Nephrogenic: Failure of kidney to respond to ADH
Presentation	Polyuria, nocturia, increased thirst, polydipsia
Diagnostic Studies	Chem 10, UA, serum osm, urine osm Lab criteria Serum Na >145 mEq/L Serum osmolarity > 300 mosm/kg Urine osmolarity < 300 mosm/kg Urine output > 4 ml/kg/hr Water deprivation test Osmo Receptor Volume Carotid Sinus Baroreceptors ADH Vasoconstriction V1R
Treatment	Central Diabetes Insipidus: vasopressin IV vs PO/intranasal/SC ddAVP Post-op patients/ICU: vasopressin infusion at 1 milliunit/kg/hr Titrate drip q5-10 minutes to max rate 10 milliunits/kg/hr w/ goal urine output <2 ml/kg/hr Replace fluid deficits w/ NS to avoid hyponatremia Check serum sodium and osm every hour Non-operative, non-ICU patients: ddAVP either PO 0.05 mg BID or intranasal 5-30 mcg/day (3 mo-12 yr) or 10-40 mcg/day (>12 yr) and titrate to goal of daily breakthrough diuresis. Nephrogenic DI: Low salt diet, thiazide diuretics, access to water Can try ddAVP if only partial nephrogenic

Syndrome of Inappropriate ADH (SIADH)		
Definition	Inappropriate antidiuretic hormone release \rightarrow hyponatremia, hypoosmolality, and inappropriately concentrated urine	
Etiology	CNS disorders: post-operative, infection, stroke, hemorrhage, trauma, Tumors (usually adults), particularly lung cancer (small cell), Drugs: carbamazepine, cyclophosphamide, others. Pulmonary disease: pneumonia, Surgery, HIV	

SIADH continued on next page \rightarrow

Endocrinology

	Syndrome of Inappropriate ADH (SIADH)
Pathophysiology	 ADH binds to V2R receptors in collecting tubules causing aquaporin-2 water channels to move from cytosol to luminal membrane. Leads to increased water reabsorption. Excessive/unregulated release of ADH from posterior pituitary or ectopic release (such as in lung cancer) leads to inappropriate retention of free water leading to hyponatremia.
Presentation	Decreased UOP, hyponatremia, low serum osm and high urine osm Patients typically have euvolemic hyponatremia, so do not have peripheral edema/ascites
Diagnostic Studies	Chem 10, UA, Serum osmolality (low) and urine osmolality (usually high), urine sodium (usually above 40 mEq/L)
Treatment	 Fluid restriction is mainstay of therapy. Goal to increase serum sodium by 6-8 mEq/L/day. Risk of central pontine myelinolysis w/ rapid correction. Start w/ restriction to 2/3 maintenance fluids daily (1 L/m2/day) Increased solute intake Can use hypertonic saline in conjunction w/ loop diuretic for symptomatic hyponatremia (seizures, AMS) To calculate the necessary dose of 3% hypertonic saline: mEq sodium infused = [desired plasma sodium (mEq/L) – actual plasma sodium (mEq/L)] x 0.6 x weight (kg) Each mL of 3% hypertonic saline has just over 0.5 mEq of sodium Give slowly (over 3-4 hours), goal not to inc plasma Na by more than 3 mEq/L/hr Given until symptoms resolve of serum Na reaches 125 mEq/L

