**Hypocalcemia in Pediatrics**

**Physiology of Calcium Homeostasis**

* Calcium (Ca) is maintained primarily by parathyroid hormone (PTH) and calcitriol (1,25 dihydroxyvitamin D) through actions on the bone, kidney, and GI tract
* PTH:
	+ Secreted in response to low Ca (sensed by the calcium-sensing receptor on parathyroid cells).
	+ Actions:
		- Bone: PTH mobilizes Ca stores and promotes bone resorption by osteoclasts
		- Kidney: PTH increases Ca reabsorption in distal tubule, increases Phosphorus (Phos) excretion, and activates 1-alpha hydroxylase which converts 25-hydroxyvitamin D to 1,25-vitamin D
		- Net effect = Increased serum Ca, decreased serum Phos
* Calcitriol:
	+ GI tract (primary source of calcium): calcitriol enhances intestinal Ca and Phos absorption
	+ Net effect = Increase serum Ca and serum Phos



serum Ca and Phos

**Hypocalcemia**: Defined as [Ca] 2 standard deviations below mean (for age) adjusted for albumin

* Symptoms/Signs: neuromuscular irritability (perioral numbness, paresthesia of hands/feet, muscle cramping, tetany, seizures), diaphoresis, hyperventilation, QT prolongation
* History**:** dietary assessment, medications (including OTC), surgical history (thyroidectomy), family history of hypocalcemia
* Physical Exam: Trousseau’s sign, Chvostek’s sign, mucocutaneous candidiasis
* Lab Evaluation: total Ca (with albumin), ionized Ca, Phos, magnesium, PTH, 25-OH vitamin D
	+ Also consider: 1,25-OH vitamin D, urine calcium/creatinine, alkaline phosphatase
* Differential diagnosis:
	+ *Neonatal Hypocalcemia*
		- Early transient (first 72 hours): maternal factors (diabetes, vit D deficiency, hyperparathyroidism, preeclampsia), neonatal factors (prematurity, low birth weight, birth asphyxia, sepsis or illness)
		- Late transient (>72 hours): high phosphate load in formula
	+ *Low PTH*
		- Abnormal parathyroid gland development or PTH synthesis (DiGeorge syndrome)
		- Activating Ca-sensing receptor mutation
		- Postsurgical or radiation damage to parathyroid gland
		- Parathyroid gland infiltration
		- Autoimmune hypoparathyroidism (isolated vs. APS1: the other two major components are adrenal insufficiency and mucocutaneous candidiasis)
		- Other: Mitochondrial disorders, HDR (hypoparathyroidism, deafness, renal anomaly) syndrome; hypomagnesemia; hyperphosphatemia
	+ *Elevated PTH*
		- * Vitamin D deficiency (poor intake, low sun exposure, decreased absorption)
			* Loss of calcium from circulation (tumor lysis, pancreatitis, etc)
			* Defects in vitamin D metabolism or action (liver disease, medications such as antiepileptic drugs and calcium chelators, kidney disease, 25-hydroxylase deficiency)
			* Vitamin D dependent rickets: 1-alpha hydroxylase deficiency, vitamin D resistance
			* Pseudohypoparathyroidism
* Management:
	+ Based on underlying etiology
		- Treat hypoparathyroidism with calcitriol, calcium
		- Treat vitamin D 25OH deficiency with Vitamin D3
	+ Acute treatment:
		- IV Calcium Gluconate (if symptomatic), 100 mg/kg (max 2g)
		- PO Calcium (if asymptomatic), 50-100 mg/kg/day elemental Ca, divided q6-8h
		- Treat concurrent hypomagnesemia