Hypoparathyroid

Hypoparathyroidism is present when secretion of parathyroid hormone is absent or deficient or peripheral tissues are resistant to the effects of the hormone. PTH stimulates bone resorption, inhibits renal excretion of calcium, and increases conversion to active vitamin D . The net effects of low PTH are: Decreased Calcium, Decreased Vitamin D, and Increased PO4. Symptoms of Hypoparathyroidism are related to hypocalcemia. A serum calcium concentration less than 4.5 mEq/L and an ionized calcium concentration lower than 2.0 mEq/L are indicative of hypoparathyroidism.

ANESTHETIC CONSIDERATIONS:

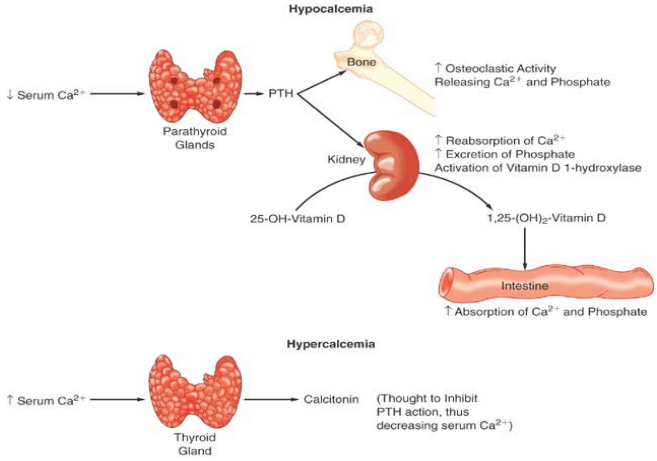
- Hypocalcemia:
  - Laryngospasm and Bronchospasm
  - QT prolongation
  - Decreased Myocardial Contractility and Hypotension
  - Seizures (not responsive to antiepileptics)
  - Psychiatric Symptoms – anxiety, dementia, psychosis
  - Extrapyramidal Syndromes

ANESTHETIC GOALS:

- Delay Surgery Until Symptoms of Hypocalcemia are controlled
- Treat Acutely with Calcium Infusion
- Correct Magnesium and Phosphate
- Avoid hyperventilation and alkalosis
- Avoid Drugs that Prolong QT interval
- Avoid Rapid infusions of blood (may worsen hypocalcemia)

ETIOLOGY AND PATHOPHYSIOLOGY

- PTH Homeostasis
  - PTH secretion increased by:
    - Low Calcium
    - Low Magnesium
  - Increased PTH causes: Increased ECF Calcium and Vitamin D
    - Bone – Release of Calcium and Phosphate by:
      - Increased Osteoclast Activity
      - Increased Bone Resorption
    - Kidney
      - Phosphate excretion and decreased reabsorption
      - Calcium and Magnesium Resorption
      - Increased 1-alpha-hydroxylase activity
  - Increased PTH causes Increased Calcium, Increased Vitamin D, and Decreased PO4



- Vitamin D Causes Increased Calcium and Increased PO4
- Decreased or Absent Parathyroid hormone
  - Accidental removal of parathyroid glands during thyroidectomy
  - Parathyroidectomy to treat hyperplasia
  - Therapy with iodine 131
  - Idiopathic (DiGeorge syndrome)
- Resistance of Peripheral Tissues to Effects of Parathormone
  - Congenital
    - Pseudohypoparathyroidism
      - Congenital disorder in which the release of parathormone is intact but the kidneys are unable to respond to the hormone
      - Affected patients manifest mental retardation, calcification of the basal ganglia, obesity, short stature, and short metacarpals and metatarsals
  - Acquired
    - Hypomagnesemia - suppresses PTH secretion and interfering with PTH action
    - CRF- leads to phosphorus retention and impaired $1,25(OH)_2D$ synthesis - treated with vitamin D
    - Malabsorbtion
    - Anticonvulsants (Phenytoin)
  - Other
    - Chronic Pancreatitis - suppression of PTH
    - Osteoblastic Metastases
    - Autoimmune disorders
    - Hemosiderosis or hemochromotosis
    - Neoplasia
    - Granulomatous disease
    - Autoimmune candidiasis related to multiple endocrine deficiency.
- The normal physiologic response to hypocalcemia is an increase in PTH secretion and $1,25(OH)_2D$ synthesis with an increase in Ca^{2+} mobilization from bone, GI absorption, and renal tubule reclamation

HISTORY AND PHYSICAL

- Clinical signs of hypocalcemia are:
  - clumsiness; convulsions; laryngeal stridor; depression; muscle stiffness; paresthesia (oral and perioral); parkinsonism; tetany; dry scaly skin, brittle nails, and coarse hair
  - Hallmark of hypocalcemia is increased neuronal membrane irritability and tetany
- Chvostek's sign - facial muscle twitching produced by manual tapping over the area of the facial nerve at the angle of the mandible.

- Chvostek's sign is positive in the absence of hypocalcemia in 10% to 15% of patients.
- Trousseau's sign - carpopedal spasm produced by 3 minutes of limb ischemia produced by a tourniquet.
- Inspiratory stridor

SYSTEM BASED CONSIDERATIONS

- Airway
  - Laryngeal Spasm
  - Perioral paresthesias
- Breathing/Respiratory
  - Inspiratory stridor (reflects neuromuscular irritability of the intrinsic laryngeal musculature)
  - Hypocalcemia worsened with hyperventilation
  - Bronchospasm
- CVS
  - Prolonged QT interval (QRS complex, PR interval, and cardiac rhythm usually remain normal)
  - Decreased Myocardial Contractility (Low Calcium)
  - Hypotension
  - Heart block (usually 2:1)
  - CHF
- Neuro
  - Convulsions
    - Cerebral tetany - generalized tetany followed by tonic spasms
    - Seizures due to hypocalcemia are NOT responsive to typical antiepileptics
  - Extrapyramidal syndromes
    - Calcifications may appear above the sella due to deposits of calcium in and around small blood vessels of the basal ganglia
    - Parkinsonian
  - Hyperreflexia
- GI
  - Steatorrhea
- GU
  - CRF (often a CAUSE of chronic hypocalcemia and secondary Hypoparathyroidism)
- Other
  - Fatigue and lethargy
  - Muscle Cramps
  - Impaired Insulin Release
  - Cataracts with chronic hypocalcemia
  - Avoid rapid infusion of blood
  - Tetany
  - Soft Tissue Calcifications
  - Preganancy
    - Hypocalcemia may be worse in preganacy due to hypoalbuminemia

DRUGS

- Changes in the calcium level may alter the duration of muscle relaxation
- Relative insensitivity to the effects of β-adrenergic agonists

INVESTIGATIONS

- Calcium, phosphate, and magnesium levels should be measured both preoperatively and postoperatively
  - Serum or ionized calcium concentration (less than 4.5 mEq/L or 2.0 mEq/L are indicative of hypoparathyroidism)
    - Hypocalcemia can also be caused by hypoalbuminemia (most common), hypomagnesemia, hypovitaminosis D, hungry bones after correction of hyperparathyroidism, anticonvulsant therapy, citrate infusion, or chronic renal disease)
- ECG
  - prolonged QT interval, Heart Block
- CBC, Electrolytes
- Other Tests as dictated by comorbid diseases and surgical procedure

TREATMENT

- Calcium Infusion - 10 to 20 mL of 10% solution
  - Oral: 500–100 mg elemental calcium q 6 hr
- vitamin D if Calcium is ineffective
- Magnesium if Hypomagnesemic
- Thiazide diuretics – cause sodium depletion without proportional potassium excretion, thereby tending to increase serum calcium concentrations
- Serum phosphate excess is corrected by the removal of phosphate from the diet and the oral administration of phosphate-binding resins (aluminum hydroxide).

PRE-OPERATIVE MANAGEMENT / OPTIMIZATION

- Bring symptoms under control before surgery and anesthesia
  - Calcium Infusion
  - Correction of any co-existing respiratory or metabolic alkalosis
  - Magnesium and Phosphate IV

ANESTHETIC OPTIONS

- No contraindications to any anesthetic technique

ANESTHETIC SETUP

- Standard CAS Monitors
- Ability to monitor calcium levels if clinical signs of hypocalcemia (arterial line)
- Other monitors as dictated by surgical procedure

INTRA-OPERATIVE MANAGEMENT

- As per surgical procedure
- Avoid Hyperventilation
- Possible prolonged response to Neuromuscular blocking agents
- Monitor QT and avoid QT prolonging drugs
    - Droperidol, Domperidone, Haldol, Sotalol, Chlorpromazine, Methadone, Procainamide, Amiodarone, Clarithromycin, Erthythromycin, Quinidine, Amitryptiline, Zofran

POST- OPERATIVE MANAGEMENT

- Monitor Calcium levels post-operatively
- Treat Hypocalcemia and symptoms thereof

COMPLICATIONS

- QT interval prolongation – Torsades
- Larnygeal Spasm
- Brosnchospasm
- Seizure
- Hypotension

REFERENCES

- Co-Existing, Miller, Barash, Toronto Notes, Chestnut