1. Laryngeal innervation

Which of the following nerves should be anesthetized in order to perform an awake tracheostomy?

- (A) Superior laryngeal
- (B) Glossopharyngeal
- (C) Trigeminal
- (D) Recurrent laryngeal

The recurrent laryngeal nerve (RLN) and the superior laryngeal nerve (SLN) are branches of the vagus nerve (CN X).

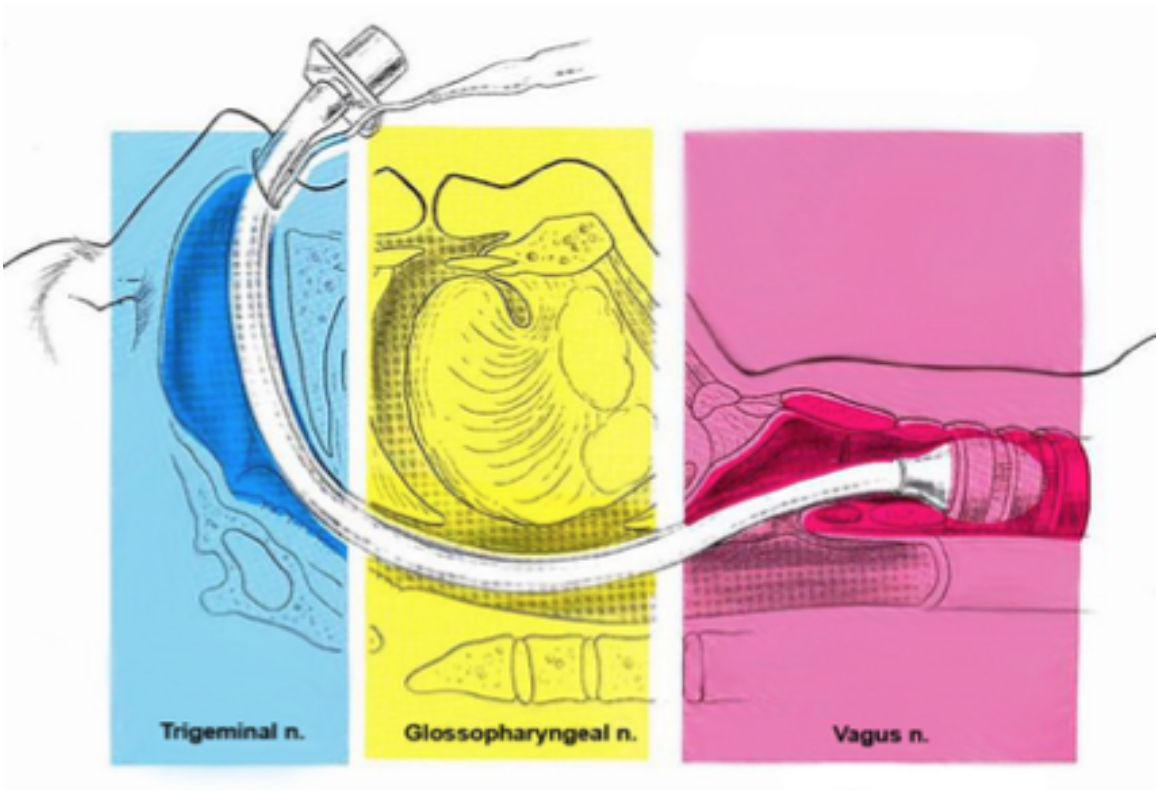
- **The recurrent laryngeal nerve** provides sensory innervation to all subglottic structures including the trachea. The recurrent laryngeal nerve also provides motor innervation to all muscles of the larynx except the cricothyroid muscle**. It is the only nerve required to be anesthetized in order to perform an awake tracheostomy.**

Post thyroidectomy, A unilateral RLN injury produces abductor vocal cord paralysis, so the affected cord assumes a paramedian position which causes postoperative hoarseness (may be a delayed presentation of a few weeks). Bilateral vocal cord paralysis, on the other hand, can manifest as partial vs. complete airway obstruction. Symptoms include respiratory distress with stridor. The situation often necessitates emergent reintubation or tracheostomy.

- **The superior laryngeal nerve**, provides sensory innervation to the epiglottis, the base of the tongue, and supraglottic mucosa down to the false vocal cords. Also provides motor innervation to the cricothyroid muscle, which tenses and adducts the vocal cords. Injury to the nerve can produce changes in voice quality

- **The glossopharyngeal nerve** provides sensory innervation to the posterior one third of the tongue, pharynx, and the superior surface of the epiglottis. Blockade of the glossopharyngeal nerve will abolish the gag reflex .

- **The trigeminal nerve** provides sensory innervation to the nasal mucosa and nasal cavity.



Hypothyroidism

Considerations

- Possible difficult airway:
    - Enlarged goiter: anatomical deviation/obstruction
    - Anterior mediastinal mass
    - Recurrent laryngeal nerve involvement
    - Prior neck radiation
- Aspiration risk
- Physiologic manifestations:
    - Cardiovascular: congestive heart failure, ↓ CO (↓ contractility/rate), hypotension, pericardial effusion, autonomic instability, hypovolemia
    - Respiratory: hypoventilation, ↓ response to hypoxemia/hypercarbia
    - Electrolytes: hyponatremia

    - Endocrine: hypoglycemia, adrenal insufficiency (cortical atrophy)
    - Hypothermia
    - ↓ metabolic rate
- Interactions with anesthetic:
    - ↓ MAC
    - Delayed emergence
    - Sensitivity to respiratory depressents
    - Perioperative endocrine supplementation (thyroid, steroids)
- Potential for myxedema coma
- Thyroid surgery:
    - Shared airway
    - Post-operative airway obstruction (recurrent laryngeal nerve injury, tracheomalacia, hematoma, hypocalcemia)

Optimization

- Euthyroid patient preoperatively
- Optimize volume status, give steroids, & manage glucose & sodium

Conflicts

- Thyroid replacement & coronary artery disease (can precipitate myocardial ischemia)
- Potential for over-sedation vs. difficult airway (post-operative analgesia)

Myxedema Coma

- Life-threatening form of hypothyroidism (mortality > 50%) precipitated by stress
- Exaggerated features of hypothyroidism:
    - ↓ LOC
    - Risk of aspiration
    - ↑ sensitivity to neuromuscular blockers & sedatives
    - ↓ cardiac output/heart rate, congestive heart failure, pulmonary edema
    - Respiratory depression
    - Hypothermia
    - Metabolic: SIADH, hypoglycemia, adrenal suppression

    - High risk for delayed emergence & need for post-operative ventilation
- Treatment:
    - IV thyroxine
    - IV T3 0.2mcg/kg q6h (onset 6-24 hrs)
    - T4 200-300mcg IV over 5-10 mins then 100mcg IV q24
    - Risk of precipitation of myocardial ischemia with IV T3/T4 supplementation in those with CAD
    - Hydrocortisone 100mg IV then 25mg q6h (common association with adrenal suppression)
    - Passive rewarming with blankets
    - Post-operative ventilation, fluids, pressors, inotropes
    - ICU & endocrinology consult

Dx – Myxedema coma

Definition

Myxedema coma typically presents as decreased mental status and hypothermia in a patient with either chronic, severe hypothyroidism or any form of hypothyroidism in the setting of a significant physiological stressor (infection, MI, sedative medications/opioids). Patients may also demonstrate hypotension, bradycardia, hypoventilation, hyponatremia, and hypoglycemia. The term “myxedema” refers to non-pitting edema that can be seen in the face, hands and lower extremities. This is secondary to mucin deposition in the skin. This deposition may also occur in the airway, necessitating intubation. Diagnosis is made by history and physical exam, and treatment with thyroid hormone replacement (both T4 and T3) and glucocorticoids (often, coexisting adrenal insufficiency can be observed) should be initiated as soon as possible: prior to the availability of the results of laboratory investigation. Major clues are a known history of hypothyroidism or the presence of a thyroidectomy scar. Serum TSH, free T4 and cortisol levels should be checked to confirm diagnosis. If the patient has primary hypothyroidism (majority of myxedema coma cases), TSH will be high and free T4 will be low. Normal/low TSH and low free T4

indicate secondary hypothyroidism (hypothalamic or pituitary dysfunction).

II. Thyrotoxicosis – Rx

Definition

Thyroxicosis is a hypermetabolic state occurring when there is an overabundance of circulating thyroid hormone (T3/T4). The condition has several common causes including Graves' Disease, thyroid nodule, thyroiditis, amiodarone, and post-partum thyroiditis. Treatment begins with antithyroid medications followed by potentially surgical excision or radioactive iodine therapy.

Antithyroid agents

- Thyrostatic medications (carbimazole, methimazole, propylthiouracil) inhibit the iodination of thyroglobulin (production of T4) by inhibition of thyroperoxidase. Additionally propylthiouracil prevents the peripheral conversion of T4 to T3. Thyrostatics take weeks to become effective.
- B-blockers provide immediate relief of symptoms until adequate treatment can be established. D-propranolol also inhibits thyroxine deiodinase blocking the conversion of T4 to T3, although this is thought to be minimal.

Surgery (total or partial thyroidectomy) is typically reserved for patients who are intolerant of antithyroid medications or radioactive iodine.

Radioactive Iodine is contraindicated during pregnancy or when breastfeeding. Radioactive iodine acts by restricting or destroying overactive thyroid tissue. Radioactive iodine uptake is increased in thyroid cells especially overactive thyroid cells yielding minimal widespread side effects. Patients may have a brief period of thyroiditis for a few days following treatment and may benefit from B-blockers.

Thyroid storm: Thyroid storm presents with body temperature to over 40 degrees Celsius (104 degrees Fahrenheit), tachycardia, arrhythmia, vomiting,

diarrhea, dehydration, coma, and death. It is most commonly seen after illness or surgery.

It requires immediate treatment with resuscitation, pharmacologic treatment with an intravenous beta-blocker such as propranolol followed by a thioamide such as methimazole, an iodinated radiocontrast agent or iodine, and an intravenous steroid such as hydrocortisone.

Thyroidectomy: complications

Definition

Major postoperative complications include wound infection, bleeding, airway obstruction (compressing hematoma, tracheomalacia), hypocalcemia, thyroid storm (uncommon, usually associated with Grave's disease) and recurrent laryngeal nerve injury.

1) **Hypocalcemia**

Incidence is 3-5%. Surgery can lead to trauma to the parathyroids, devascularization of the glands with resultant ischemia, or inadvertent excision of these small structures. Hypoparathyroidism with subsequent decreased production of parathyroid hormone leads to decreased serum calcium. ***Acute hypocalcemia generally presents at 24-48 hours as laryngeal stridor and airway obstruction.*** First symptoms are usually tingling in the lips and fingertips. Additional findings may develop, including carpopedal spasm, tetany, laryngospasm, seizures, QT prolongation and cardiac arrest. Chvostek's sign is facial contractions elicited by tapping the facial nerve in the per-auricular area. Trouseau's sign is carpal spasm on inflation of a blood pressure cuff. CPAP is often effective for associated airway compromise, and 1 gram of calcium gluconate given slowly usually alleviates symptoms.

2) Airway obstruction (compressing hematoma, trachiomalacia)

Incidence of hematoma is 1-2%, tracheomalacia incidence is <1%. Acute airway obstruction from hematoma may occur immediately postoperatively and is the most frequent cause of airway obstruction in the first 24 hours.

Definitive therapy is opening the surgical incision to evacuate the hematoma. Re-intubation may be lifesaving for persistent airway obstruction. Consider awake fiberoptic intubation.

3) Recurrent laryngeal nerve injury

Incidence of injury is 0.77% for unilateral damage resulting in hoarseness and 0.39% for bilateral damage with associated aphonia and airway obstruction.

4) Wound infection

Incidence is 0.2-0.5%.

***Airway obstruction: In the first 24 hours is most likely from compressive hematoma. After 24 hours consider laryngeal dysfunction secondary to hypocalcemia.**

III. Hyperparathyroidism: signs

Definition

Epidemiology: Primary hyperparathyroidism occurs in about 0.1% of the population, most commonly begins in the third to fifth decades of life, and occurs two to three times more frequently in women than men.

Etiology: Primary hyperparathyroidism usually results from enlargement of a single gland, commonly an adenoma and very rarely a carcinoma. Hypercalcemia almost always develops.

Important Facts: The normal total serum calcium level is 8.6 to 10.4 mg/dL, as measured in most laboratories. Fifty percent to 60% is bound to plasma proteins or is complexed with phosphate or citrate. The value is dependent on the albumin level, with a decline of 0.8 mg/dL for each 1-g/dL drop in albumin. Binding of calcium to albumin is dependent on pH: binding decreases with acidic pH and increases with alkaline pH. It should be noted that serum calcium and not ionized calcium decreases with decreases in albumin levels. PTH and vitamin D3 work to keep the level stable within 0.1 mg/dL in any individual.

Causes: To remember the causes, a useful mneumonic is CHIMPANZEE's. **C**alcium excess (administration) **H**yperparathyroidism **I**mmobility / **I**atrogenic **M**etastasis / **M**ilk-alkali syndrome **P**aget's disease **A**ddison's disease **N**eoplasms **Z**ollinger Ellison syndrome **E**xcess vit D **E**xcess vit A **S**arcoidosis

Symptoms: Many of the prominent symptoms of hyperparathyroidism are a result of the hypercalcemia that accompanies it. Regardless of the cause, hypercalcemia can produce any of a number of symptoms, the most prominent of which involve the renal, skeletal, neuromuscular, and GI systems —anorexia, vomiting, constipation, polyuria, polydipsia, lethargy, confusion, formation of renal calculi (in approximately 60-70% of patients with hyperparathyroidism), pancreatitis, bone pain, and psychiatric abnormalities. Peptic ulcer disease is more common in these patients due to increased production of gastrin and gastric acid. Mnemonic "Stones, Groans, Moans, Psychiatric Overtones"

Symptoms of Hyperparathyroidism

- Neuro: confusion, lethargy, psychiatric abnormalities
- GI: anorexia, vomiting, constipation, pancreatitis
- GU: polyuria, polydipsia, formation of renal calculi (in approximately 60-70% of patients with hyperparathyroidism)
- Other: bone pain

Treatment: diuresis and administration of normal saline to dilute plasma calcium. These primary treatments also are useful because sodium inhibits the renal reabsorption of calcium.
Additional therapies include 1)bisphosphonates (pamidronate is the most commonly used) 2)calcitonin 3)ambulation 4)hemodialysis 5)treatment of the underlying condition.
Certain conditions, including numerous cancer-related hypercalcemias, can be treated with calcium-lowering agents, such as mithramycin and glucocorticoids.
Anesthetic management: avoidance of thiazide diuretics and maintenance of hydration and urine output with sodium-containing fluids. Monitoring the patient by means of electrocardiograms is useful to detect cardiac conduction abnormalities with shortened P–R or Q–T interval, with or without widening of the QRS complex. Patients who have muscle weakness should receive decreased doses of nondepolarizing muscle relaxants.

For a more detailed explanation of why the symptoms listed above in bold occur, see below:

Sustained hypercalcemia can result in tubular and glomerular disorders, including proximal (type II) renal tubular acidosis. Polyuria and polydipsia are common complaints.

Skeletal disorders related to hyperparathyroidism are osteitis fibrosa cystica, simple diffuse osteopenia, and osteoporosis. The rate of bone turnover is five times higher in patients with hyperparathyroidism than in normal controls. Patients may have a history of frequent fractures or complain of bone pain, the latter especially in the anterior margin of the tibia.

Because free intracellular calcium initiates or regulates muscle contraction, neurotransmitter signaling, hormone secretion, enzyme action, and energy metabolism, abnormalities in these end organs are often symptoms of hyperparathyroidism. Patients may experience profound muscle weakness, especially in proximal muscle groups, as well as muscle atrophy. Depression, psychomotor retardation, and memory impairment may occur. Lethargy and confusion are frequent complaints.

Peptic ulcer disease is more common in these patients than in the rest of the population. Production of gastrin and gastric acid is increased. Anorexia, vomiting, and constipation may also be present.

Approximately a third of all hypercalcemic patients are hypertensive, but the hypertension usually resolves with successful treatment of the primary disease. Long-standing hypercalcemia can lead to calcifications in the myocardium, blood vessels, brain, and kidneys. Cerebral calcifications may cause seizures, whereas renal calcifications lead to polyuria that is unresponsive to vasopressin.