Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Weakened tracheal rings, distorted/displaced trachea Ophthalmopathy	Snoring, hoarseness, neck pain	Ask pt to vocalize "e," examine airway and neck, look at the eyes, test for diplopia, note change over time in measure of eye protrusion	Check CXR (PA and lateral), lateral neck films; CT scan or US of neck
CV	Dysrhythmias, AFIB, sinus tachycardia, mitral valve prolapse CHF, cardiomyopathies	Palpitations; increased HR during sleep, DOE, orthostatic SOB	Standard exam	Rhythm strip or full ECG CV system is involved in either Hx or PE
GI	Weight loss, diarrhea, dehydration Hepatic enzyme abnormality due to medications	Dizziness on arising; Hx of diarrhea, constipation	Skin turgor; other measures of volume status such as orthostatic vital signs	Increased serum alkaline phosphatase
HEME	Mild anemia, thrombocytopenia; agranulocytosis secondary to propylthiouracil or methimazole		Skin/mucous membranes for infection/petechiae	CBC with platelet count and differential
CNS		Shaking, anxiety, emotional lability	Reflex speed, tremor, nervousness, mental status	
METAB	Need to assess if euthyroid and/or malnourished	Refer to all other systems, especially reflex speed, tremor, heat intolerance, fatigue, weakness, weight loss, anorexia, increased appetite	Reflex speed; HR	Free T_4

Key Reference: Roizen MF, Fleisher L: Anesthetic implications of concurrent diseases. In Miller RD et al, editors: *Anesthesia,* ed 7, New York, 2010, Elsevier, pp 1077–1080.

Perioperative Implications

- See Thyroidectomy, Subtotal.

Preoperative Preparation
- Assess if euthyroid.
- Assess for associated autoimmune diseases.

Preinduction/Induction
- Prehydrate liberally if CV status will tolerate it.
- Check and protect eyes.

Anesthetic Technique
- No one technique has proved superior.
- Hyperthyroidism is an associated risk factor for halothane hepatitis.

Monitoring
- Temperature. (Also place cooling blanket on OR table for possible treatment of thyroid storm.)
- Consider invasive monitoring if pt has dilated cardiomyopathy/thyroid storm/severe dysrhythmia.
- If head-up position is utilized, consider air embolus monitoring and therapy.

Airway
- Consider awake fiberoptic intubation if there are questions regarding adequacy of airway or distortion/involvement of the trachea.
- Consider armored tube or equivalent if tracheal rings are affected.

Induction/Maintenance
- Routine

Adjuvants
- Usually no requirement for muscle relaxants

Anticipated Problems/Concerns

- Thyroid storm is a life-threatening condition if hyperthyroidism has been severely exacerbated by illness or operation. Manifested by hyperpyrexia, tachycardia, and striking alterations in consciousness. Early signs include delirium, confusion, mania, excitement. Differential Dx: Malignant hyperthermia, pheochromocytoma crisis, NMS.
- Rx includes supportive care, methimazole or propylthiouracil followed in 1 h by iodides and propranolol or atenolol; these decrease conversion of the less active T_3 to the more active T_4.
- Surreptitious bleeding behind neck bandages or into chest if minimally invasive technique is used from axilla, can suddenly compromise airway function or result in CV collapse.
- Injuries to the recurrent laryngeal nerve after thyroidectomy usually result in damage to abductor fibers, resulting in hoarseness.
- Bullous glottic edema can require immediate reintubation.
- Occasionally late tetany (usually 2–3 d after thyroidectomy) can occur from accidental removal of or damage to parathyroid glands.

Hypertriglyceridemia

Andrew Bowdle

Risk

- Prolonged propofol infusion due to lipid vehicle
- Genetic defects in triglyceride metabolism
- Component of the metabolic syndrome (obesity, hypertriglyceridemia, low HDL, Htn, diabetes)

Perioperative Risks

- Associated with atherosclerosis, coronary, and cerebrovascular disease.
- Hyperglycemia (metabolic syndrome) increases risk of surgical wound infection.
- Severe hypertriglyceridemia may cause acute pancreatitis.

Worry About

- Coronary and cerebrovascular disease
- Pancreatitis
- Blood sugar control in metabolic syndrome
- Propofol infusion syndrome if hypertriglyceridemia is due to prolonged propofol infusion (hypertriglyceridemia due to propofol may occur with or without other features of propofol infusion syndrome, including rapidly progressive myocardial failure, bradycardia, ECG changes resembling Brugada syndrome, lactic acidosis, rhabdomyolysis, elevated serum creatine kinase, urea and potassium, elevated liver enzymes, hepatomegaly, and lipemic blood)

Overview

- High triglycerides are strongly associated with coronary artery atherosclerosis.
- Normal <150 mg/dL, borderline high 150–199 mg/dL, high 200–499 mg/dL, very high >500.
- >1000 mg/dL: Severe hypertriglyceridemia may cause acute pancreatitis.
- Prolonged and/or high-dose propofol infusion may produce hypertriglyceridemia.

Etiology

- Primary hypertriglyceridemia is caused by a variety of disorders of triglyceride metabolism.
- Secondary hypertriglyceridemia is caused primarily by obesity, diabetes, nephrotic syndrome, hypothyroidism, pregnancy, restrogen replacement, tamoxifen, beta-blockers, immunosuppressive medications, HIV antiretroviral agents, and retinoids.

Usual Treatment

- Diet and weight loss if due to obesity
- Lipid-lowering drugs: Statins for triglycerides <500 mg/dL (mainly to reduce risk of coronary artery atherosclerosis), fibrates for triglycerides >500 mg/dL
- If due to propofol infusion, discontinue or reduce infusion

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Atherosclerosis	Hx of coronary disease or heart failure	JVD, peripheral edema, S_3, S_4	ECG, CXR, coronary CT, ECHO, stress test, coronary angiography
ENDO	Associated with altered glucose metabolism	Hx of diabetes, ketoacidosis; hypothyroidism		Blood glucose, HgbA1C; thyroid function tests when applicable
RENAL	Caused by nephrotic syndrome, renal failure	Urinary frequency		BUN, Cr, lytes
CNS	Atherosclerosis, cerebrovascular disease	TIA or stroke	Neurologic exam	CT angiography, head CT
GI	Fat accumulation in liver and spleen; acute pancreatitis	Abd discomfort, pain	Hepatosplenomegaly, obesity	Amylase, lipase
DERM			Cutaneous xanthoma	

Key References: Bowdle A, Richebe P, Lee L, et al.: Hypertriglyceridemia, lipemia and elevated liver enzymes associated with prolonged propofol anesthesia for craniotomy, *Ther Drug Monit* 36(5):556–559, 2014; Brinton EA: Management of hypertriglyceridemia for prevention of atherosclerotic cardiovascular disease, *Cardiol Clin* 33(2):309–323, 2015.

Perioperative Implications

Preoperative Preparation

- Blood glucose.
- Severe hypertriglyceridemia should be controlled prior to elective surgery due to risk of pancreatitis.

Monitoring

- Determined based on coexisting coronary or cerebrovascular disease
- Blood glucose

Airway

- If obese, increased risk of difficult intubation.
- If obese, rapid sequence intubation may be advisable due to aspiration risk. Diabetes may cause gastroparesis.

Induction

- Determine based on coexisting coronary or cerebrovascular disease.
- Avoid propofol if history of propofol infusion syndrome (rare).

Maintenance

- Propofol infusion may cause hypertriglyceridemia if prolonged and/or high dose. Consider avoiding propofol infusion if preexisiting very high or severe hypertriglyceridemia.

Extubation

- Consider aspiration risk, such as obesity and diabetic gastroparesis (same as for induction). Extubate awake if at risk.

Postoperative Period

- Blood sugar control.
- Monitoring as dictated by coronary and cerebrovascular disease.
- Consider risk of obstructive sleep apnea if obese.
- If intubated and sedated postop, consider avoiding propofol infusion if preexisting very high or severe hypertriglyceridemia.

Anticipated Problems/Concerns

- Complications due to comorbidities: Obesity, diabetes, coronary artery disease, cerebrovascular disease, obstructive sleep apnea.
- Severe hypertriglyceridemia may cause acute pancreatitis.

Hypokalemia

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Risk

- Defined as plasma K^+ <3.5 mEq/L.
- Common conditions and/or treatments place pts at increased risk, including
  - Those on diuretics (especially loop and thiazide diuretics) to treat Htn, CHF, and so forth.
  - Those experiencing significant GI fluid loss (e.g., vomiting, diarrhea, or gastric suction).
  - Those with increased serum pH (metabolic or respiratory alkalosis).

Perioperative Risks

- Increased risk of cardiac dysrhythmias (with greater concern in those with preexisting heart disease and in setting of acute onset hypokalemia)
- Increased risk of muscle weakness (which includes possible respiratory muscle weakness and prolonged neuromuscular blockade)
- Increased risk of GI hypomotility

Worry About

- Cardiac dysrhythmias are the most worrisome complication of hypokalemia.
- Many medications regularly used in periop treatment can cause or worsen hypokalemia (e.g., diuretics, antibiotics, β_2-agonists, epinephrine).
- Pts requiring significant/urgent K^+ replacement may require central line placement.
- Over-replacement: Any pt requiring K^+ replacement may be at risk for hyperkalemia and thus the malignant dysrhythmias associated with hyperkalemia.

Overview

- K^+ ions have essential role in maintaining cellular resting membrane potentials and in generating functional activity in muscle cells, neurons, and cardiac tissue.
- Overall, intracellular K^+ concentration is approximately 30 times greater than extracellular K^+ concentration; this ratio is maintained by cell membrane Na^+/K^+ ATPase.
- Decreases in extracellular K^+ impairs nml gradients required for membrane potential/action potential transmission.
- Acute/rapid decreases in serum K^+ concentration create more concerning derangements in cellular membrane potential physiology than chronic or slowly developing decreases in K^+.

Etiology

- Inadequate K^+ intake: Seen in eating disorders, inability to eat, "tea and toast" diet, alcoholism, and those receiving K^+-poor TPN
- Increased K^+ excretion:
  - Renal losses: Mineralocorticoid excess (primary or secondary hyperaldosteronism, Cushing disease, congenital adrenal hyperplasia), hyperreninism, congenital renal disorders (Bartter/Gitelman/Liddle syndromes), medication-induced (loop and thiazide diuretics, carbonic anhydrase inhibitors, amphotericin B, some penicillins, gentamicin)
  - GI losses: Vomiting, diarrhea, NGT/OGT suction, villous adenoma, ureterosigmoidostomy
- Intracellular K^+ shifts: alkalosis (metabolic or respiratory), medication induced (insulin administration, β_2-agonists, epinephrine, terbutaline, ritodrine), refeeding syndrome, periodic paralysis, barium toxicity

Usual Treatment

- Identify and attempt to correct the underlying factors causing the hypokalemia (e.g., adjust diet intake, review medications, lower pH of pts with alkalosis by treating primary disorder).
- K^+ repletion: It is reported that each 10 mEq of K^+ given will raise serum K^+ by 0.1 mEq/L.
  - Oral K^+: Can use K^+ paired with gluconate, phosphate, chloride, or citrate, with delivery via tablet or solution.
  - IV K^+: Most commonly as K^+ chloride. Careful repletion required via programmable infusion pump to avoid hyperkalemic complications; patients receiving >10–20 mEq/h should have cardiac monitoring in place. Peripheral IV administration can cause burning sensation and vascular epithelium damage; consider placement of central line.
- Coexisting hypomagnesemia: requires correction before repletion of potassium will be successful.