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.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CNS	Muscle weakness Cramping/myalgia	Decreased mobility, falls, decreased ADL Complaints of muscle pain	Decreased muscle strength	TOF intraoperative
RESP	Resp muscle failure	SOB, hypoventilation, ventilator dependence	Poor inspiratory effort, low TV	ABG, NIF
CV	Dysrhythmias Vasomotor instability	Complaints of palpitations, syncope, cardiac arrest Syncope, falls, disorientation	 Refractory shock, hypotension	ECG
GI	Decreased GI motility	Constipation, abdominal pain	Loss of bowel sounds, abd tenderness and distention	KUB
RENAL	Polyuria Polydipsia Increased renal ammonia Edema and sodium retention	Frequent urination Frequent drinking		 Urine ammonia Urine sodium

Key References: Gennari FJ: Hypokalemia, *N Engl J Med* 339(7):451–458, 1998; Wong KC, Schafer PG, Schultz JR: Hypokalemia and anesthetic implications, *Anesth Analg* 77(6):1238–1260, 1993.

Perioperative Implications

Preoperative Preparation

- Obtain serum K^+ concentration preop if pt presents with risk factors for hypokalemia.
- Attempt to identify and/or address the etiology of hypokalemia.
- For elective cases, replete serum K^+ concentration to >2.6 before going to OR. Discuss concerns and implications with pt/family, and surgical team.
- Have ACLS medications on hand and transport with cardiac monitoring.

Monitoring

- ECG/continuous cardiac monitoring (watch for T wave flattening, U waves, PVC, VT/VF).
- BP cuff or arterial line (watch for hypotension related to vasomotor insufficiency).
- Periodic ABG and lyte panels as needed (watch for pH and K^+ trend).
- Twitch monitor (watch for prolonged neuromuscular blockade).

Maintenance

- Judicious use of medications associated with causing or exacerbating hypokalemia.
- Control glucose and fluid volume.
- Avoid hyperventilation and respiratory alkalosis.

Anticipated Problems/Concerns

- Pts with symptomatic hypokalemia (especially with cardiac symptoms) that are not well controlled after initial treatments may need elective surgical procedures delayed.
- Cardiac dysrhythmias are of greatest concern in hypokalemia because these can be lethal. Risk is greatest when hypokalemia is acute and serum K^+ <3.0.
- Preop problems: ECG changes and volume status (related to diuretics or polydipsia).
- Intraop problems: Persistent hypotension after induction (related to refractory vasomotor response to catecholamines), prolonged neuromuscular blockade, respiratory muscle weakness.

Hypomagnesemia

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Risk

- 12% of all hospitalized pts as well as 44–60% of all pts admitted to medical/surgical and pediatric ICUs, are hypomagnesemic.
- Associated with
  - Poor nutrition.
  - GI losses: Diarrhea and severe vomiting; malabsorption (steatorrhea, bowel resection, intestinal fistulas, celiac disease); acute pancreatitis; medications (proton pump inhibitors, laxatives).
  - Renal losses: Medications (loop/thiazide diuretics, aminoglycosides, amphotericin B, cisplatin, foscarnet, cyclosporine); familial renal Mg^{2+} wasting syndromes; uncontrolled diabetes mellitus; metabolic acidosis; alcohol abuse.
  - Miscellaneous: Prolonged IV therapy; massive blood transfusions; digitalis.

Perioperative Risks

- Arrhythmias (atrial, ventricular, prolonged QT, and torsades de pointes). Hypomagnesemia should be corrected prior to elective procedures due to the potential for malignant arrhythmias.
- Worsening cardiac ischemia and CHF.
- Increased susceptibility to seizures, bronchoconstriction, and vasospasm.
- Refractory hypokalemia and hypocalcemia.
- Resistance to vasodilators.
- Aggravates insulin resistance in the diabetic pt.

Worry About

- Weakness, lethargy, paresthesias, muscle spasms.
- Seizures (especially in preeclampsia).
- Arrhythmias (especially torsades de pointes).
- During treatment of hypomagnesemia: Burning at IV site, overall sense of warmth and flushing. Transient and mild hypotension may occur if $MgSO_4$ is given too fast. Administration of Mg^{2+} will also potentiate the neuromuscular blockade with all nondepolarizing drugs.

Overview

- Normal range of plasma Mg^{2+} is 1.7–2.4 mg/dL. Most symptomatic pts have levels <1 mg/dL.
- Mg^{2+} levels are not routinely checked in screening tests. Hypomagnesemia should be suspected, especially in chronic diarrhea, alcoholism, malnutrition, long-term hospitalization, and hypoalbuminemia.
- Mg^{2+} is primarily an intracellular ion. Plasma levels may not reflect the true magnitude of deficit. Intracellular shift may occur with the administration of insulin and thyroid hormone.
- Normomagnesemic Mg^{2+} depletion has been described; if clinical suspicion of hypomagnesemia is present, Mg^{2+} should be administered, even with normal plasma levels.
- If it is unclear from the pt's history, a 24-h urine sample may help to differentiate renal from nonrenal causes. Mg^{2+} loss of less than 3–4 mEq/d supports a renal etiology.
- Alternatively, a fractional excretion of Mg^{2+} can be calculated in a spot urine sample.
  - $FE_{Mg} = [(U_{Mg} \times P_{Cr})/(0.7 \times P_{Mg}) \times U_{Cr}] \times 100$, where U_{Mg}/U_{Cr} and P_{Mg}/U_{Cr} denotes urinary and plasma concentrations of Mg^{2+} and Cr.
  - Usually, FE_{Mg} greater than 2% indicates renal Mg^{2+} wasting.

Usual Treatment

- Chronic hypomagnesemia may be treated with oral magnesium.
- Acute administration of 1-2 g $MgSO_4$ IV over 20-30 min for pts with symptoms. Significantly decreased Mg^{2+} levels may require 4-8 g $MgSO_4$ IV over the next 24 h.
- If Mg^{2+} replacement is needed, give at the beginning of an anesthetic because $MgSO_4$ may interfere with neuromuscular blockade reversal.
- Torsades de pointes can be treated with 1–2 g $MgSO_4$ IV push over 5–20 min.
- Usual doses for preeclampsia are 4–6 g bolus over 15-20 min followed by 1–2 g/h, targeting a plasma level around 6 mg/dL.
- Each g of $MgSO_4$ has 98 mg of elemental Mg^{2+} (equivalent to 4 mmol or 8 mEq).
- As long as renal function is intact, excessive Mg^{2+} levels will be cleared over several h. In pts with kidney disease, Mg^{2+} replacement should be done cautiously.

Therapeutic Uses

- Mg^{2+} has multiple functions including, but not limited to, decreasing acetylcholine in motor nerve terminals,