infarction, pancreatitis, or drug ingestion (drugs that affect carbohydrate metabolism).

Usual Treatment

- Aggressive volume resuscitation with isotonic fluids to re-establish end-organ perfusion
- Insulin replacement and correction of electrolyte abnormality (start dextrose-containing fluids when serum glucose approaches 250 mg/dL to help to prevent hypoglycemia and cerebral edema)
- Identify and treat underlying cause of hyperglycemic state
- Frequent evaluation of volume resuscitation and metabolic status in an ICU setting

Assessment Points

System	Effect	Assessment by Hx	PE	Test
NEURO	Altered mental status, obtundation, coma, seizures	Progression of mental status changes over days	Mental status exam, airway reflexes	Head CT, CSF culture
CV	Hypovolemia and shock	Polyuria progressing to anuria, sense of thirst, headaches, dry mouth	Orthostatic hypotension, tachycardia, dry mucous membranes	CVP, PAP, ECHO
PULM	Hyperventilation if severe metabolic acidosis, hypoventilation if brainstem malperfusion		Resp rate and pattern of ventilation	ABG
ENDO	Insulinopenia, hyperglycemia, hyperosmolarity	DM, recent infection or stress		Serum glucose and osmolarity
RENAL	Polyuria progressing to anuria, metabolic acidosis, electrolyte abnormalities			BUN and Cr, FEN_a, UO, ABG, lytes

Key References: Maletkovic J, Drexler A: Diabetic ketoacidosis and hyperglycemic hyperosmolar state, *Endocrinol Metab Clin North Am* 42(4):677–695, 2013; Pichardo-Lowden A, Gabbay RA: Management of hyperglycemia during the perioperative period, *Curr Diab Rep* 12(1):108–118, 2012.

Perioperative Implications

Monitoring and Intravenous Access

- Large-bore IV access for volume resuscitation, particularly before induction.
- Invasive monitoring including arterial line and CVP may be useful to guide volume replacement and allow for frequent glucose and electrolyte sampling.

Induction

- Aggressive volume resuscitation before induction.
- Rapid sequence induction if altered mental status and concern for aspiration.
- Limited use of succinylcholine if metabolic acidosis and hyperkalemia are present.
- Be prepared for exaggerated hemodynamic changes with induction despite adequate volume resuscitation.
- Smaller doses than usual of induction agent if pt is obtunded.

Maintenance

- Closely follow serum glucose, lytes, and acid-base status.
- Continue volume resuscitation until UO is adequate and hemodynamics have stabilized.

Emergence

- Assessment of airway reflexes and ability to protect airway before tracheal extubation.
- Ensure metabolic and lyte status is corrected and pt meets the usual criteria for extubation

Postoperative Period

- Continued insulin therapy and observation for worsening hyperglycemia due to surgical stress response

Anticipated Problems/Concerns

- Comorbidities and diffuse end-organ damage increase morbidity and mortality of pts with HHS, particularly periop.

Hyperkalemia

Alan David Kaye | Mark R. Jones | Rachel J. Kaye

Risk

- Any pt with plasma K^+ concentration <5.5 mEq/L

Perioperative Risks

- Muscle weakness and paralysis
- Cardiac conduction system abnormalities
- CV collapse:
  - Peaked T waves (6–7 mEq/L)
  - ST depression
  - Prolonged P-R interval and widened QRS (10–12 mEq/L)
  - Ventricular fibrillation or asystole

Worry About

- Adverse effects are likely to accompany acute increases in K^+; chronic increases are better tolerated.
- Depolarizing muscle relaxants, especially if given to pts with burns, spinal cord transection, catatonia with immobility, or muscle trauma.
- Digitalis toxicity.
- Acidosis.

Overview

- Condition that can be due to increased total body K^+ content or alterations in distribution between intracellular and extracellular sites

Etiology

- Diminished renal excretion
- Acute oliguric renal failure
- Chronic renal failure
- Addison disease
- Hyporeninemic hypoaldosteronism
- Medications: Potassium-sparing diuretics, NSAIDs, heparin, K^+-containing antibiotics
- RAASi: Incidence of hyperkalemia <2% with RAASi monotherapy: Increased to 5% in pts receiving dual-agent RAASi therapy, and to 5%–10% when dual therapy was administered to pts with CKD
- Ingestion of K^+-rich foods, salt substitutes in pts with renal insufficiency
- Transcellular shifts
- Acidosis: Respiratory or metabolic
- Cell destruction: Trauma, burns, rhabdomyolysis, hemolysis, tumor lysis, or reperfusion of ischemic limb or organ
- Hyperkalemic periodic paralysis
- Diabetic hyperglycemia
- Depolarizing muscle relaxant causing K^+ release, especially in pts with burns, spinal cord transection, catatonia with immobility, muscle trauma, or denervating muscle
- Massive transfusion, particularly with irradiated blood
- Factitious hyperkalemia
- Tourniquet method of drawing blood
- Hemolysis of drawn blood due to delay in chemical determination

Usual Treatment

- Promote transfer of K^+ from ECF to ICF.
- Glucose and insulin: 25–50 g glucose with 10–20 units regular insulin/70 kg.
- Sodium bicarbonate: 50–100 mEq/70 kg.
- Hyperventilation: with each pH change of 0.1, there is an inverse change in K^+ of 0.5 mEq/L (goal $PaCO_2$ 25–30 mm Hg).
- Enhance K^+ elimination: Diuretics, exchange resins (Kayexalate), dialysis.
- Antagonism of cardiac effects: Ca^{++} gluconate—10–30 mL of a 10% solution over 10–20 min/70 kg counteracts cardiac effects.
- 50–250 mL hypertonic saline (3%–5%): Stabilizes membrane potential; effective only in hyponatremic pts.
- β_2-receptor agonists: 10–20 mg aerosol (nebulized) or 0.5 mg in 100 mL of 5% dextrose in water (IV) redistributes K^+ from ECF to ICF; use with caution in pts with CAD.
- New treatments: K^+ binders (patiromer and sodium zirconium cyclosilicate [ZS-9]) increase fecal potassium excretion; may offer better predictability, tolerability, and safety for pts with hyperkalemia; importantly, these new K^+ binders may allow the continued use of such medications as RAASis

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Tall peak T waves Decreased amplitude R wave Widened QRS complex Decreased and eventual disappearance of P wave QRS blends into T wave—"sine wave of hyperkalemia" Ventricular arrhythmia Cardiac arrest	 Possible hemodynamic instability CV collapse		ECG ECG ECG
MS	Weakness Paralysis			
ENDO	Increased aldosterone Insulin release Increased glucagon Epinephrine release		Increased BP, HR	K^+, renin, aldosterone, glucose

Key References: Kovesdy CP: Management of hyperkalaemia in chronic kidney disease, *Nat Rev Nephrol* 10(11):653–662, 2014; Seferovic PM, Pelliccia F, Zivkovic I, et al: Mineralocorticoid receptor antagonists, a class beyond spironolactone—focus on the special pharmacologic properties of eplerenone, *Int J Cardiol* 200:3–7, 2015.

Perioperative Implications

Preoperative Preparation

- Normal K^+ levels before elective surgery
- Avoid sedatives (decreased ventilation) prior to K^+ normalization

Monitoring

- ECG
- Plasma K^+ levels
- ABG concentration
- Peripheral nerve stimulator

Maintenance

- Adequate ventilation to avoid respiratory acidosis.
- Avoid metabolic acidosis: Arterial hypoxemia or excessive depths of anesthesia.
- IV fluids: Avoid lactated Ringer or others containing K^+.

Adjuvants

- Muscle relaxants: avoid depolarizing agents; increase K^+ 0.3–0.5 mEq/L with succinylcholine.
- Dose of nondepolarizing relaxants required is unclear; may need diminished dose.

Anticipated Problems/Concerns

- Acute increases in K^+ leading to acute ECG changes or adverse cardiac effects. Rx (see Usual Treatment).
- Avoid use of depolarizing muscle relaxants in pts with burns, neuropathies, paraplegia or quadriplegia, muscle trauma, or catatonia with immobility.

Hypermagnesemia

Alan David Kaye | Lien Tran | Erik M. Helander

Risk

- Pts with renal insufficiency, especially those receiving Mg^{2+}-containing cathartics or antacids.
- Parturients on $MgSO_4$ therapy.
- "Runaway" infusion of Mg^{2+} during transportation to the OR can cause acute, life-threatening hypermagnesemia. Risk of developing very high serum Mg^{2+} levels in such cases can be reduced by always using a small-volume buretrol device in pts receiving IV Mg^{2+} therapy.

Therapeutic Uses

- Treatment of preeclampsia, eclampsia, and preterm labor.
- Evidence indicates that Mg^{2+} therapy reduces the risk of cerebral palsy in women at risk of preterm delivery.
- Treatment of ventricular dysrhythmias, especially torsades de pointes.
- Treatment of severe asthma in pts who have not responded to initial therapy.
- Treatment of migraine.
- Lowers risk of metabolic syndrome.

Perioperative Risks

- Potentiates nondepolarizing neuromuscular blocking agents.
- May increase risk of modest hypotension during administration of regional anesthesia.
- Potentiates hypotension associated with use of volatile anesthetics, CCBs, and butyrophenones.
- Can exacerbate local anesthetic toxicity.
- Hypermagnesemia may be associated with increased in bleeding time and TEG changes, although no clinically significant coagulopathies have been attributed to Mg^{2+}.

Worry About

- Intraop hypotension
- Muscle weakness (especially respiratory)
- Excessive sedation
- Myocardial depression and cardiorespiratory arrest with very high levels

Overview

- Defined as an elevated Mg^{2+} concentration in plasma, in excess of 1.1 mmol/L.
- Equivalent Mg^{2+} concentrations in the three unit systems in common use: mg/dL, mEq/L, mmol/L.
  - Normal serum level 1.8–2.4 mg/dL, 1.5–2.0 mEq/L, 0.75–1.0 mmol/L.
  - Therapeutic level 4.8–8.4 mg/dL, 4–7 mEq/L, 2–3.5 mmol/L.
  - Neuromuscular toxic level greater than 12 mg/dL, greater than 10 mEq/L, greater than 5 mmol/L.
- Mg^{2+} elimination is dependent on GFR; with GFR less than 30 mL/min, pts are at significant risk.
- Signs and symptoms vary with plasma concentration and become more serious as the plasma concentration increases greater than 4 mmol/L.
- CV, respiratory, and MS systems are predominantly affected.
  - Pts with chronic renal failure frequently have Mg^{2+} levels up to 3 mmol/L but are seldom symptomatic.
  - Acidemia will decrease serum level at which side effects occur; e.g., in presence of acidemia, cardiac arrest can occur at a serum level of 8–10 mmol/L.

Etiology

- Pts with chronic renal failure who are receiving Mg^{2+}-containing antacids or laxatives
- Often iatrogenic; for example, excessive administration of $MgSO_4$ infusion to parturient pts with preterm labor or pregnancy-induced Htn
- Less common causes: Addison disease, myxedema, excessive tissue breakdown, or lithium therapy

Usual Treatment

- Discontinue Mg^{2+} therapy and delay nonessential surgery.
- Fluid load and diuretic therapy in pts with normal renal function.
- Adults: IV calcium gluconate 1 g (temporary but effective).
- Neonates: IV calcium gluconate 100–200 mg/kg over 5 min and continuous infusion 100–300 mg/kg per d.
- Peritoneal dialysis or hemodialysis for persistent or life-threatening hypermagnesemia.
- Assist ventilation/protect airway if necessary.