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

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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.

Assessment Points

The side effects of hypermagnesemia are more serious as the serum level of magnesium increases.

System	Signs and Symptoms	Serum Mg^{2+} Concentration (mmol/L)
GENERAL	Normal	0.7–1.1 (normal range)
CV	Warmth, flushing, headache, nausea, dizziness	2.5–4.0
	Decreased AV and intraventricular conduction	3.7–4.9
	ECG: Prolonged PR and widening of QRS	
	Hypotension	
	Cardiopulmonary arrest*	>8.9
CNS	Confusion or sedation	3.7–4.9
MS	Absent deep tendon reflexes	3.7–4.9
	Profound muscle weakness	5–6.95

*The ability of this degree of hypermagnesemia to cause cardiac arrest is uncertain if ventilatory support and normal acid-base balance are maintained.

Key References: Jahnen-Dechent W, Ketteler M: Magnesium basics, *Clin Kindey J* 5(Suppl 1):i3–i14, 2012; Herroeder S, Schönherr ME, De Hert SG, et al.: Magnesium–essentials for anesthesiologists, *Anesthesiology* 114(4):971–993, 2011.

Perioperative Implications

Preoperative Preparation

- Discontinue $MgSO_4$ unless being used to treat seizures or ventricular dysrhythmias.
- Check serum level.
- ECG, Cr, and lytes.

Monitoring

- Routine

Airway

- Does not affect onset or duration of succinylcholine; fasciculations may not be observed.
- Reduce dose of nondepolarizing NMBs by one-third to one-half.

Preinduction/Induction

- Avoid sedative premedications.
- Ensure full denitrogenation of lungs.
- Avoid precurarization or priming dose of NMB.

Maintenance

- May decrease requirement for anesthetics owing to decreased neurotransmitter release.

Extubation

- Ensure full return of train-of-four, ability to sustain head lift, and vital capacity lesser than 10 mL/kg.
- Ensure pt responsiveness.

Adjuvants

- Hypermagnesemia may exacerbate hypotension associated with hypovolemia, CCBs, volatile inhalation anesthetics, butyrophenones, lumbar epidural, or subarachnoid anesthesia.
- Treat with IV calcium gluconate 1 g, IV fluids, and diuretics.

Postoperative Period

- Beware of excessive sedation, weakness, hypoventilation, and cardiac arrest.
- May cause or aggravate neonatal hypotonia and hypotension.
- May reduce postop analgesic requirements by antagonism of *N*-methyl-D-aspartate.

Anticipated Problems/Concerns

- Hypermagnesemia potentiates action of nondepolarizing NMBs by inhibiting release of acetylcholine from motor nerve terminal, decreasing sensitivity of postjunctional membrane, and reducing excitability of muscle fibers.
- Many common anesthetic drugs exacerbate weakness and sedation associated with hypermagnesemia.
- Potentiates local anesthetic toxicity.
- Excessively high plasma Mg^{2+} concentrations can cause cardiorespiratory arrest.

Hypernatremia

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Risk

- Older age, infants, prior brain injury, DM, surgery, diuretic therapy, altered mental status, insufficient water intake, DI, hypertonic sodium solution (including sodium bicarbonate), hyperalimentation, hyperaldosteronism, Cushing syndrome, and hypothalamic injury

Perioperative Risks

- Increased incidence of morbidity and mortality, seizures, coma, cerebral bleeding, and subarachnoid hemorrhage

Worry About

- Increased risk of hospital death, residual and/or permanent neurologic disability
- If Na^+ corrected too rapidly, cerebral edema, seizures, and death

Overview

- Hypernatremia is a relative deficit of body H_2O in relation to body sodium content.
- Serum Na^+ is preserved within a fine physiologic range (138–142 mEq/L).
- Sodium metabolism is regulated by the kidney through the interaction of the RAAS, sympathetic nervous system, atrial natriuretic peptide, brain natriuretic peptide, effective circulating volume, and serum H_2O content. H_2O metabolism is tightly regulated by arginine vasopressin.
- Most commonly found in pts with impaired sense of thirst (brain injury, altered mental status), lack of access to H_2O, diuretic therapy, and severe GI losses of H_2O.

Etiology

- Lack of access to H_2O
- Impaired thirst mechanism
- DI (central and nephrogenic)
- Osmotic diuresis (mannitol, glucose); diuretics (furosemide, thiazides)
- Insensible losses from the dermal or respiratory systems
- GI losses from diarrhea or osmotic cathartics (lactulose, sorbitol), vomiting, or nasogastric suctioning
- Seizures or severe exercise (transient intracellular shift of H_2O)
- Excess sodium administration; hyperalimentation
- Hyperaldosteronism and Cushing syndrome

Usual Treatment

- H_2O replacement (see later); central DI can be treated with desmopressin (5–20 µg intranasal once or twice per day), nephrogenic DI can be treated with thiazide diuretics.
- Free H_2O deficit = 0.6 × weight (kg) × ([current Na^+/140]–1).
- Total body water is approximately 0.6 and 0.5 times the lean body weight for men and women, respectively. Replace ½ of the free H_2O deficit over the first 24 h as an initial starting point. Note that the free H_2O deficit does not take into account ongoing losses, so ultimately the rate of H_2O replacement must be guided by serial measurements of serum Na^+.
- Rate of correction of Na^+ to level of 145 mmol/L:
  - If hypernatremia developed acutely, Na^+ can be corrected rapidly (1 mmol/L per h with a limit of 12 mmol/L per 24 h).
  - If hypernatremia developed slowly, Na^+ can be corrected at a maximum rate of 0.5 mmol/L per h (or in the case of life-threatening complications, at 1 mmol/L per h with a limit of 12 mmol/L per 24 h).
  - Measurement of Na^+ at least every 4–6 h, and adjustment of the rate of H_2O replacement is important to ensure safe and expeditious correction of Na^+.