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^+.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Dry mouth/mucous membranes		Mouth exam	
CV	Tachycardia/hypotension	Orthostatic changes	HR/BP	ECG
CNS	Restlessness, irritability, lethargy, seizures, coma		CNS exam	EEG
GI	N/V, diarrhea			
RENAL	Polyuria	Urinary frequency and color		Serum and urine Na^+, K^+, osmolarity

Key References: Sterns RH: Disorders of plasma sodium-causes, consequences, and correction, *N Engl J Med* 372(1):55–65, 2015; Bagshaw SM, Townsend DR, McDermid RC: Disorders of sodium and water balance in hospitalized patients, *Can J Anaesth* 56(2):151–167, 2009.

Perioperative Implications

Preoperative Preparation
- Correct lytes, replace H_2O deficit in controlled and calculated manner, assess neurologic status.
- Consider delaying elective surgery until serum Na^+ is normal. If surgery cannot be delayed, care must be taken to avoid rapid correction of Na^+.

Monitoring
- Electrolytes

Airway
- None

Maintenance
- Restore circulatory volume.
- Maintain urine output.
- Correct lytes.

Extubation
- Assess neurologic status to determine whether the pt is a candidate for extubation.
- Possible muscular weakness.

Adjuvants
- In central DI, vasopressin 5 U IVP will dramatically reduce UOP for 1–2 h, making it possible to catch up on IV fluids.
- Caution must be used to avoid too-rapid correction of Na^+.

Postoperative Period
- Assess for lethargy, irritability, muscular weakness, and confusion.
- Monitor serum Na^+.

Anticipated Problems/Concerns
- Too rapid correction and resultant neurologic effects

Hyperparathyroidism

Geoffrey L. Liu | Henry Liu

Risk
- Incidence in USA: 100,000 pts/y; increases with age
- Male:female ratio: 1:2; 0.8% in pregnancy
- Prevalence: 0.7% in general population; up to 3% in postmenopausal women
- Due to malignancy, vitamin D deficiency, sarcoidosis

Perioperative Risks
- Hypovolemia and electrolyte disturbances
- Increased risk of cardiac dysrhythmias secondary to hypercalcemia
- Aspiration from full stomach and/or mental change
- Postop hypocalcemia
- Airway compromise due to hematoma or recurrent laryngeal nerve injury

Worry About
- Signs of hypercalcemia and other electrolyte irregularities
- Intravascular volume changes
- Fluid overload and Na^+ retention in CV fragile pts
- Renal, cardiac, skeletal, and CNS abnormalities
- Pancreatitis due to hypercalcemia

Overview
- Endocrinopathy associated with elevation in PTH levels.
- Primary problem is hypercalcemia, leading to "moans, groans, and stones."
- Diagnosis supported by increased PTH level with hypercalcemia.
- Most pts with primary hyperparathyroidism are hypercalcemic but asymptomatic.
- Hyperparathyroidism in pregnancy, leading to high maternal and fetal morbidity (50%) and neonatal hypocalcemia and tetany.

Etiology
- Primary hyperparathyroidism usually due to benign parathyroid adenoma (80–90%), hyperplasia (15%), or parathyroid carcinoma (uncommon).
- May be manifestation of multiple endocrine neoplasia type I or IIa.
- Secondary hyperparathyroidism may be seen in pts with chronic renal disease.

Usual Treatment
- Parathyroidectomy shifting from standard four glands to only pathologic gland(s) removal.
- Advances in nuclear imaging to accurately localize parathyroid tumor(s), quick hormone assays, and radio-guided or video-assisted techniques facilitate minimally invasive parathyroidectomy, possibly under local/regional anesthesia.
- Medical treatment: Saline hydration, furosemide, and phosphate repletion in emergency situations to restore serum Ca^{2+} to a safe level (<14 mg/dL).
- Other Ca^{2+}-lowering modalities: calcitonin, cinacalcet, bisphosphonates (inhibit bone resorption), mithramycin (for more resistant hypercalcemia; toxic effects limit use), glucocorticoids, or hemodialysis.
- Pregnant women with primary hyperparathyroidism should be treated with parathyroidectomy, ideally in the second trimester.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Htn, dysrhythmias	Palpitation, headache	Abnormal HR, rhythm, increased BP	ECG, lytes, total and ionized Ca^{2+}, QT_c* interval
RESP	Decreased bronchial clearance of secretions	Cough	Adventitious sounds	
GI	Peptic ulcers, pancreatitis	Constipation, anorexia, N/V, epigastric pain		
RENAL	Nephrocalcinosis, nephrolithiasis, leading to renal abnormalities	Polyuria, polydipsia, hematuria		BUN, Cr
CNS	EEG abnormalities, seizures Lethargy	Depression, personality change, psychomotor retardation, memory impairment	Psychosis, disorientation Obtundation, coma	
MS	Hyporeflexia, osteopenia, osteitis fibrosa cystica	Weakness, bone pain	Muscular atrophy, arthritis Pathologic fractures	Bone density

* $QT_c = \frac{QT}{\sqrt{R-R}}$; *R-R,* R-R interval.

Key References: Kelz RR, Fraker DL: Hyperparathyroidism: what preoperative imaging is necessary? *Adv Surg* 49:247–262, 2015; Nahrwold ML, Nahrwold DA: Hyperparathyroidism. In Fleisher LA, Roizen MF, editors: *Essence of anesthesia practice,* ed 3, Philadelphia, PA, 2011, Elsevier.