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

Sara M. Skrlin

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,

acting as a vasodilator, a Ca^{2+} antagonist, and acting on the myocardium to slow the rate of conduction through the SA node. In addition to correction of hypomagnesemia, Mg^{2+} replacement can be used for the following:

- CV: Myocardial protection, decreases CHF, improves contractility, diastolic relaxation, attenuates or prevents tachycardic arrhythmias, minimizes changes in heart rate and BP during intubation, decreases the risk of postop atrial fibrillation in cardiac surgery.
- Neurologic: Has neuroprotective effects, but the degree to which it is clinically useful is uncertain.
- Endocrine: Attenuates insulin resistance; helps in hemodynamic control in pheochromocytomas by causing arteriolar vasodilation and decreasing the hypertensive response to catecholamines.
- Obstetric: Widespread use in treatment of preeclampsia/eclampsia and decreases risk of cerebral palsy in preterm infants.
- Pulm: Bronchodilation in severe asthmatic.
- Anesthesia: May decrease need for inhalation agent to maintain same BIS level.
- Pain: May decrease the need for postop opiates through its blockade of NMDA receptors.
- MS: Relaxes muscle rigidity and decreases autonomic dysfunction in tetanus.
- Intoxication and recreational drugs: Helpful to treat catecholamine excess and hypertension associated with cocaine and methamphetamines.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CNS	Seizures, cerebral vasospasm (after SAH)	Lethargy, SAH (vasospasm)	Altered mental status	Plasma Mg^{2+}, TCD, cerebral angiogram
CV	Arrhythmias, torsades, wide QRS, CHF (impaired diastolic relaxation)	Tachyarrhythmia, Htn, dyspnea		ECG, plasma Mg^{2+}, BNP, ECHO
MS	Hypocalcemia (decreased secretion and resistance to PTH)	Weakness, tetany	Chvostek and Trousseau signs	Plasma Ca^{+2}, Mg^{2+}
ENDO	Insulin resistance, may affect lipid profile	Diabetes (types 1 and 2) Hyperlipidemia		Glucose, plasma Mg^{2+}, HDL, triglycerides
RESP	Bronchospasm	Asthma	Wheezing	Plasma Mg^{2+}
RENAL	Hypokalemia (K^+ loss from loop of Henle)	Alcohol abuse, nephrotoxins (antibiotics, chemo), diuretics		Cr, BUN Plasma K^+, Mg^{2+}

Key References: Herroeder S, Schönherr ME, De Hert SG, et al.: Magnesium—essentials for anesthesiologists, *Anesthesiology* 114(4):971–993, 2011; Ayuk J, Gittoes NJ: Treatment of hypomagnesemia, *Am J Kidney Dis* 63(4):691–695, 2014.

Perioperative Implications

Preoperative Preparation

- Check serum Mg^{2+} level (<1.7 mg/dL is hypomagnesemia).
- Obtain 12-lead ECG.
- Start replacing Mg^{2+} (e.g., 2 g IV over 20 min; faster replacement safe but may cause burning at the IV site).

Monitoring

- Standard ASA monitors.
- Plasma Mg^{2+} levels (normal range 1.7–2.5 mg/dL).
- TOF monitoring (replacing Mg^{+2} potentiates NMB agents).
- Consider BIS (or other depth of anesthesia) monitoring because replacing Mg^{2+} may alter anesthetic requirement.

Induction

- Mg^{2+} IV bolus during induction is safe (e.g., 2–4 g IV bolus).
- May cause mild and transient drop in BP.
- Replacing Mg^{2+} minimizes changes in heart rate and BP during intubation.
- Hypomagnesemia may cause or exacerbate bronchospasm.

Maintenance

- Replacing Mg^{2+} attenuates or prevents tachyarrhythmias and may convert some types of malignant arrhythmias.
- Insulin resistance may occur in the hypomagnesemic pt.

Emergence

- Replacing Mg^{2+} attenuates shivering.
- Replacing Mg^{2+} maintains hemodynamic stability.
- Titrate NMB agents and reverse residual NMB, especially if Mg^{2+} was replaced intraop.
- Hypomagnesemia may worsen bronchospasm in asthmatic pts.

Postoperative Period

- Actual efficacy of Mg^{2+} as an analgesic adjuvant is currently unclear, but Mg^{2+} replacement may lead to decreased opioid consumption.
- Increased catecholamine levels may exacerbate arrhythmias in the hypomagnesemic pt.

Anticipated Problems/Concerns

- Although Mg^{2+} replacement is usually well tolerated, potential problems with overdose include
  - Levels above 8–10 mg/dL may cause diaphragmatic weakness, and above 10–12 mg/dL may cause widening of QRS and conduction blocks. These levels are rarely reached with recommended doses and in the absence of decreased GFR.
  - Potentiation of neuromuscular blockade.

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Hyponatremia

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Risk

- Preop hyponatremia is a prognostic marker for increased 30-d mortality, major cardiac events, wound infection, and pneumonia.
- Premenopausal women, especially those undergoing procedures associated with rapid irrigant absorption, are at particularly high risk of both symptomatic hyponatremia and osmotic demyelination with Na^+ correction.
- Conditions associated with SIADH or adrenocortical insufficiency (Addison disease).
- Elderly taking diuretics.
- Pts with liver, heart, or renal failure.
- Hyponatremia especially common in elderly pts and associated with increased morbidity and mortality.
- Up to 10–15% of men undergoing TURP.
- Infants and/or children receiving multiple tap H_2O enemas.

Perioperative Risks

- Risk of CV collapse with adrenocortical insufficiency and inability to cope with stress of surgery.
- Iatrogenic dilution in TURP and endoscopic gynecologic procedures associated with CNS, cardiopulmonary, and skeletal muscle abnormality.
- Increased ADH secretion extremely common periop and may cause further decrease in serum Na^+.
- Isotonic saline (0.9%) will result in free H_2O gain and decrease in serum Na^+ in presence of SIADH.

Overview

- Normal serum sodium levels are 135 to 145 mEq/L. Hyponatremia defined as serum Na^+ >135 mEq/L; most common cause is an excess of total body H_2O, usually associated with low serum osmolality (<275 mOsmol/kg).
- Hyponatremia can be associated with low, normal, or high tonicity (tonicity defined as the contribution to osmolality of solutes that cannot freely cross cell membranes).
- There are at least two systems of classification for hyponatremia with low serum osmolality: one according to the level of inappropriately elevated or suppressed ADH and the other according to the volume status (hypovolemia, normovolemia, or hypervolemia).
- Sodium and water balance in the body regulated by the kidney affects the plasma tonicity (ADH mechanism) and effective arterial blood volume (renin-angiotensin-aldosterone system).