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.

Acknowledgment

The author would like to acknowledge Drs. Mehmet Ozcan and James Feld's contributions to this chapter in the previous edition.

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).

- Change in tonicity causes free H_2O shift leading most importantly to cerebral intracellular volume changes (edema in hypotonic hyponatremia). Extracellular volume may be decreased, normal, or increased.

Etiology

- Most common causes of severe hyponatremia in adults: Postop state, thiazide diuretics, clinical scenarios associated with SIADH, polydipsia in psychiatric pts, TURP.
- Multiple tap H_2O enemas most common cause in infants and children.
- Hypervolemic hyponatremia:
  - Cirrhosis, nephrotic syndrome.
  - Cardiac failure.
  - Renal failure postop.
  - TURP.
- Normovolemic hyponatremia:
  - SIADH (associated with CNS, pulm diseases/malignancy, pain, drugs, stress).
  - Endocrine: Glucocorticoid deficiency, hypothyroidism.
  - Pseudohyponatremia syndrome, for example, factitious hyponatremia (normotonic hyponatremia): Hyperlipidemia states (e.g., chylomicronemia) or hyperproteinemia.
- Hypovolemic hyponatremia:
  - Renal loss (diuretics, mineralocorticoid deficiency, osmotic diuresis, cerebral salt wasting, ketonuria, renal tubule acidosis/metabolic acidosis).
  - Extrarenal loss (trauma, vomiting/diarrhea, burns, pancreatitis).
- Diagnosis:
  - Laboratory: Urine osmolarity, serum osmolarity, urine sodium concentration.
  - Imaging: Consider head CT and CXR.
- Approach to treatment:
  - Based on overall risk stratification.
  - Duration: Acute = hyponatremia developing within last 24 h; subacute = hyponatremia present for 24–48 h; chronic = hyponatremia present for greater than 48 h.
  - Severity: Mild (130–135 mEq/L); moderate (125–129 mEq/L); profound (125 mEq/L or less).
  - Symptoms: Absent or present.

Usual Treatment

- Sodium levels are corrected by treating the underlying disorder, fluid restrictions, administering oral or IV sodium chloride, or vasopressin receptor antagonist therapy, depending on the etiology and severity of hyponatremia.
- Goal of sodium correction: increase plasma sodium concentration 6 mEq/L (not to exceed 12 mEq/L) in first 24 h, followed by goal increase of 8 mEq/L every 24 h after to serum sodium 130 mEq/L.
- Severe symptomatic hyponatremia: immediate treatment with IV infusion of hypertonic 3% saline with goal of 6 mEq/L increase in serum sodium over several h (not to exceed 12 mEq/L in 24 h).
- Asymptomatic or moderate hyponatremia with mild-to-moderate symptoms:
  - Hypovolemic hyponatremia: nonemergent therapy with IV infusion of isotonic saline with goal of 6 mEq/L (not to exceed 12 mEq/L) slowly over 24 h.
  - Hypervolemic hyponatremia: salt and fluid restrictions, ± loop diuretics with goal of 6 mEq/L (not to exceed 12 mEq/L) slowly over 24 h. Vasopressin (V2) receptor antagonist can also be considered.
  - Close monitoring of serum electrolytes and fluid intake/output during correction therapy is critical (every 2–4 h). Also pt's mental status should also be reassessed regularly.
  - Dose of sodium required to correct a deficit may be calculated using the following formula:

$$\text{Dose (mEq)} = (\text{Weight [kg]} \times (140 - [\text{Na}]) [\text{mEq/L}]) \times 0.6.$$

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Dysrhythmias	Palpitations		Oscillation, ECG (wide QRS, increased ST, VT/VF)
	CHF, hypervolemia	DOE, orthopnea	S_3, rales	CXR
	Hypovolemia	Lightheadedness, weakness	Orthostatic hypotension, CVP, tachycardia	BP, CVP
RESP	Pulm edema	DOE	S_3, rales	CXR
CNS	Confusion, restlessness, gait disturbance, lethargy, seizures, visual disturbances, obtundation, coma			Serum Na^+ <115–120 mEq/L associated with profound symptoms
MS	Weakness, cramps	Weakness, cramps	Weakness, hyporeflexia	Reflexes
RENAL	Free H_2O retention Salt wasting			Urine Na^+, serum, and urine osmolality

Key References: Spasovski G, Vanholder R, Allolio B, et al.: Clinical practice guideline on diagnosis and treatment of hyponatraemia, *Nephrol Dial Transplant* 29(Suppl 2):i1–i39, 2014; Leung AA, McAlister FA, Rogers SO Jr, et al.: Preoperative hyponatremia and perioperative complications, *Arch Intern Med* 172(19):1474–1481, 2012.

Perioperative Implications

Preinduction

- Ensure medical optimization of comorbid diseases (hyponatremia greater risk with increasing severity of disease: ASA III and IV).
- Caution with sedatives.
- Preop lytes in high-risk procedures.
- Consider regional in TURP to facilitate monitoring of mental status.
- Increased ADH and volume changes associated with surgical trauma likely to decrease Na^+ further.
- Identify irrigating solution and prepare for irrigant-specific side effects.

Monitoring

- TURP:
  - Metal status with regional technique.
  - Automatic versus manual fluid monitoring system for fluid absorption.
  - TURP procedures of long duration and with significant bleeding or increased hydrostatic pressure of irrigant predictive for large amounts of irrigation fluid absorption (increased vigilance required).
  - Consider EEG with GA.
  - Consider invasive monitoring (CVP/PA cath/TEE) with development of TURP syndrome and CHF in elderly pts.
- Hyponatremic pts undergoing therapy to correct serum Na^+:
  - Serum Na^+.
  - Urine output.
  - Mental status.
  - ECG.

General Anesthesia

- Sodium concentration >130 mEq/L usually considered safe for pts undergoing general anesthesia, whereas moderate-to-severe hyponatremia should be postponed.
- For general anesthesia serum sodium levels should be corrected to greater than 130 mEq/L, even when no neurologic symptoms are present.
- Isotonic fluids for volume resuscitation.
- Ensure adequate depth of anesthesia as pain and/or stress associated with ADH release.

Regional Anesthesia

- Spinal or epidural with T10 block for TURP.
- Prepare for emergency airway protection if respiratory distress, seizures, obtundation.

Postoperative Period

- Monitor mental status for agitation, confusion, somnolence.
- Adequate pain management to reduce sympathetic-related Na^+ and water retention.
- Close monitoring for administration of hypotonic solutions.
- Monitor daily serum Na^+ (strict monitoring in high-risk pts).
- Initiate appropriate therapy in symptomatic or severely hyponatremic pts.
- Avoid too rapid correction and associated demyelination syndrome.
- Restore blood and/or volume loss if necessary.