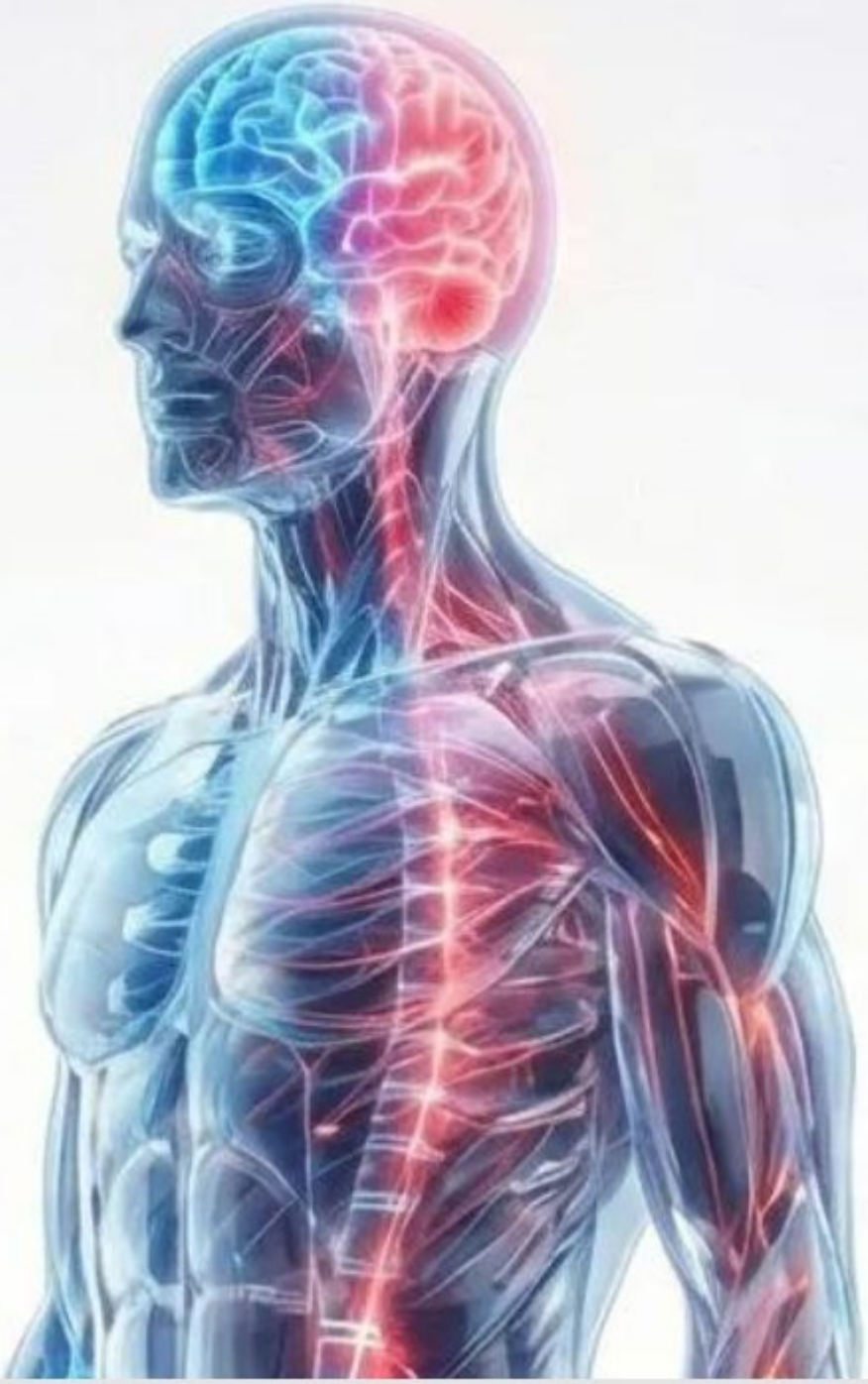
HYPO & HYPERNATREMIA

National Standard Treatment Guideline



Ministry of Health
Republic of Maldives

ADB

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Japan Fund for Prosperous and Resilient Asia and the Pacific

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World Health Organization
Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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HYPO AND HYPERNATREMIA

QUICK REFERENCE GUIDE

Electrolyte imbalance disrupts fluid and cellular function, with hyponatremia (serum sodium <135 mmol/L) being the most common inpatient disorder. It affects all ages - older adults due to impaired water clearance and drugs, and infants from feeding errors or illness. Causes include diuretics, SIADH, hypotonic fluids, and excess water intake. Severe cases can lead to seizures, coma, and increased mortality, while timely, controlled correction usually ensures recovery; chronic or prolonged episodes may cause cognitive or developmental deficits, especially in neonates and the elderly.

Hyponatremia is defined as serum sodium <135 mmol/L. Hypotonic hyponatremia: serum osmolality <275 mOsm/kg. Acuity: acute <48 h; chronic ≥48 h or unknown.

Biochemical severity

- Mild: 130-134 mmol/L
- Moderate: 125-129 mmol/L
- Severe (profound): <125 mmol/L

Symptom - based severity

- No/mild symptoms: nausea, headache, subtle confusion, unsteadiness.
- Moderate symptoms: vomiting, marked confusion, agitation, somnolence.
- Severe symptoms (hyponatremic encephalopathy): seizures, coma, respiratory arrest, signs of raised ICP → treat immediately with hypertonic saline.

Presenting features

- Nausea, headache, confusion, lethargy; gait imbalance.
- Severe: seizures, decreased level of consciousness, respiratory arrest.
- Clues to cause: diuretics (esp. thiazides), SSRIs/carbamazepine/NSAIDs/ desmopressin; HF/cirrhosis/CKD; hypothyroidism/adrenal insufficiency; high water intake or hypotonic IV fluids.

Red flags (both adults and children)

- Seizure, coma, respiratory compromise, signs of raised ICP.
- Serum Na^+ <120 mmol/L or rapid fall with neurologic symptoms.
- Shock, severe dehydration, or suspected adrenal crisis.

Initial assessment

- ABCs; bedside glucose; focused neuro exam.
- Volume status: orthostasis, JVP, edema/ ascites, mucous membranes.
- Labs: serum Na^+/osmolality, K^+, creatinine/BUN, glucose; urine Na^+/ osmolality; TSH and AM cortisol if unclear.

Emergency management (severe symptoms, or rapid decline with Na^+ typically <125 or a sharp fall from baseline)

- 3% NaCl 100 mL IV over 10 min, repeat 1-2 times until seizures stop or Na^+ increases to 4-6 mmol/L.
- Then slow correction to target (below). Treat precipitants (e.g., hypoxia, hypoglycemia).

Cause - directed therapy

- Hypovolemic: 0.9% NaCl 250-500 mL boluses to euvolemia; replace GI losses; stop offending diuretics.
- Euvolemic (SIADH): fluid restriction ~800-1,000 mL/day; isotonic maintenance; treat trigger; consider oral salt and/or loop diuretic if urine Osm high; vaptans only with inpatient monitoring when other measures fail.
- Hypervolemic (HF/cirrhosis/nephrotic): fluid/Na restriction, loop diuretics; disease-specific therapy.

Monitoring and safety

- Serum Na^+ q4-6 h during active correction; neuro checks q1-2 h initially.
- Correction limits: after the initial 4-6 mmol/L "rescue," keep total rise ≤8-10 mmol/L in 24 h.
- If Na^+ rises too fast: pause therapy, give DDAVP and small D5W to re-establish a safe slope.
- Track input/output (I/O), weight; correct K^+/Mg^{2+}.

Discharge

- Stable Na^+ for ≥24 h; cause addressed; outpatient Na^+ checks arranged; clear medication and fluid plan.

Children (neonates and infants included)
Presenting features

- Infants: irritability, poor feeding, vomiting, high-pitched cry, apnea; bulging fontanelle (raised ICP).
- Older children: headache, nausea, confusion, ataxia; seizures can occur early.
- Triggers: gastroenteritis with excess free water, over-diluted formula, hypotonic IV fluids, CNS/ respiratory infections (SIADH), adrenal disorders; adolescents - exercise-associated hyponatremia, drug-induced SIADH.

Symptom - based severity (pediatric cues)

- No/mild symptoms: irritability, mild lethargy, headache, nausea.
- Moderate symptoms: vomiting, confusion, ataxia, reduced responsiveness.
- Severe symptoms (hyponatremic encephalopathy): seizures, apnea, coma, bulging fontanelle, respiratory compromise → urgent hypertonic saline, weight-based.

Initial assessment

- ABCs; bedside glucose; pediatric neuro checks (alertness, cry, feeding, tone, fontanelle).
- Volume status: capillary refill, extremity temperature, weight change.
- Labs: serum Na^+/osmolality, K^+, creatinine/BUN, glucose; urine Na^+/osmolality; blood gas if ill; add 17-OHP/cortisol ± renin/aldosterone if salt-wasting suspected.

Emergency management (severe symptoms)

- 3% NaCl 2-4 mL/kg IV over 10-20 min (max ~100 mL); may repeat once or twice until symptoms improve or Na^+ rise to 4-6 mmol/L.
- Seizure protocol in parallel; treat precipitant.

Cause - directed therapy

- Hypovolemic: 0.9% NaCl 10-20 mL/kg bolus; reassess; replace ongoing GI losses; antiemetics.
- Euvolemic (SIADH): fluid restriction ~60-80% of maintenance (weight-based); isotonic maintenance; treat trigger; consider oral NaCl 1-3 mmol/kg/day in divided doses ± loop diuretic if urine Osm high. Avoid vaptans/demeclocycline.
- Hypervolemic: careful fluid/Na restriction, loop diuretics; disease-specific care with specialist input.

Monitoring and safety

- Serum Na^+ q2-4 h during active correction (neonates/infants may need q1-2 h early).
- Correction limit: total rise ≤8 mmol/L in 24 h after initial 4-6 mmol/L rescue.
- If Na^+ rises too fast: DDAVP 0.3 µg/kg IV/SC + weight-calculated D5W; recheck in 2-4 h.
- Nutrition: avoid overly tight restriction without a feeding plan; confirm correct formula preparation.
- Strict I/O, hourly urine output if severe; daily weights (more frequent in neonates).

Discharge

- Stable Na^+ for ≥24 h; caregiver teaching on red flags (vomiting, headache, confusion, seizures), exact fluid/feed plan; outpatient Na^+ checks q2-4 days until normal.

HYPONATREMIA

INTRODUCTION

Electrolytes govern fluid balance, nerve transmission, muscle contraction, and acid-base status. Sodium sets extracellular osmolality and supports neuronal signaling, muscle function, and blood pressure. Potassium stabilizes the resting membrane potential in nerve and cardiac cells. Minor shifts can cause headache, confusion, or cramps; major disturbances can precipitate seizures, coma, or lethal arrhythmias. Early recognition and careful correction prevent acute harms such as cerebral edema in sudden hyponatremia and rhythm deaths in hypo- or hyperkalemia and reduce longer-term problems like gait instability and cognitive decline.

Hyponatremia, defined as serum sodium below 135 mmol/L, is the most common inpatient electrolyte disorder, affecting roughly 15-20% of admissions and up to 30% in some settings. It reflects excess body water relative to sodium and ranges from incidental lab finding to life-threatening cerebral edema. Older adults are vulnerable because of reduced free-water clearance, comorbid illness, and drugs such as diuretics, selective serotonin reuptake inhibitors, and anticonvulsants, leading to higher risks of falls, fractures, prolonged stays, and mortality. Children, especially neonates and infants, are at risk from gastroenteritis, hypotonic IV fluids, over-diluted formula, excess free water, and syndrome of inappropriate antidiuretic hormone due to central nervous system or respiratory infections; rapid onset heightens seizure risk. Timely identification in primary care or at admission, accurate volume assessment, targeted tests, and age-appropriate therapy are key.

SCOPE OF THESE GUIDELINES

These guidelines cover hyponatremia & hypernatremia from initial evaluation through follow-up in adults though these guidelines provide approach to management of electrolyte imbalance in children.

Intended users

Physicians, nurses, and allied healthcare professionals at primary, secondary, and tertiary care levels.

Applicability by Healthcare Level

- **Primary Care:** Early recognition, basic volume assessment, and referral criteria.
- **Secondary Care:** Full diagnostic workup, initial fluid therapy, and monitoring protocols.
- **Tertiary Care:** Specialist consultation, use of vaptans, and management of complex or refractory cases.

DEFINITIONS

Hyponatremia is defined as a serum sodium concentration below 135 mmol/L in both adults and children. It reflects either excess body water relative to sodium or sodium loss exceeding water loss. In pediatric patients, causes often include gastroenteritis, inappropriate use of hypotonic IV fluids, excessive free-water intake, over-diluted formula feeds, or SIADH secondary to CNS or respiratory infections. Neonates and infants are at higher risk due to immature renal water handling, higher baseline water turnover, and complete dependence on caregivers for fluid provision.

Hypernatremia

Hypernatremia is defined as a serum sodium concentration above 145 mmol/L in both adults and children. It indicates a relative water deficit or, less commonly, sodium excess. In pediatric patients, common causes include diarrhea, vomiting, high-solute formula feeds, inadequate breastfeeding, congenital or acquired diabetes insipidus, and iatrogenic sodium administration. Infants are especially vulnerable because of limited ability to communicate thirst, immature renal concentrating capacity, and proportionally greater insensible water losses.

Other common electrolyte emergencies in both adults and children are defined by specific serum concentration cut-offs, though normal ranges and urgency thresholds may vary slightly by age.

Hypokalemia (<3.5 mmol/L) increases susceptibility to ventricular arrhythmias, muscle weakness, and paralytic ileus; in pediatrics, levels below 3.0 mmol/L can precipitate rapid deterioration, especially in those with underlying cardiac disease.

Hyperkalemia (>5.0 mmol/L in adults; >5.5 mmol/L in children and neonates) slows cardiac conduction and can lead to bradycardia, conduction blocks, and sudden cardiac arrest.

Hypocalcemia is defined as an adjusted serum calcium below the lower limit of the age-specific reference range, typically <2.15 mmol/L in adults, <2.0 mmol/L in older children, <1.9 mmol/L in infants, and <1.75 mmol/L in neonates. It may present with tetany, seizures, prolonged QT interval, or laryngospasm, with neonates and infants at higher risk of rapid symptom onset due to smaller calcium reserves and higher requirements during growth.

Hypercalcemia is defined as an adjusted calcium above the age-adjusted upper limit, >2.6 mmol/L in adults and older children, >2.75 mmol/L in infants, and >2.9 mmol/L in neonates, and can cause arrhythmias, altered mental status, hypotonia, polyuria, and dehydration.

Hypomagnesemia is generally <0.7 mmol/L in adults and older children, <0.65 mmol/L in infants, and <0.62 mmol/L in neonates. It can trigger neuromuscular irritability, refractory hypocalcemia, seizures, and ventricular arrhythmias.

Hypermagnesemia is >1.05 mmol/L in adults and older children, >1.1 mmol/L in infants, and >1.2 mmol/L in neonates, and may cause hypotonia, respiratory depression, conduction blocks, and cardiac arrest, particularly in newborns exposed to high maternal magnesium during labor.

Evaluation Principles for Both Disorders

- Assess volume status (hypovolemic, euvolemic, hypervolemic) as the first step in guiding management.
- Measure plasma osmolality to confirm true hypo- or hypernatremia and rule out laboratory artifacts such as pseudohyponatremia from severe hyperlipidemia or hyperproteinemia.
- In pediatrics, integrate weight trends, feeding history, and caregiver-reported intake/output into the assessment to identify potentially reversible causes.

HYPONATREMIA
CAUSES, RISK FACTORS & TRIGGERS (ADULTS)

Causes (by mechanism)

- **Hypovolemic:** GI losses (vomiting/diarrhea), diuretics especially thiazides, third spacing (pancreatitis, burns), adrenal insufficiency.
- **Euvolemic:** Syndrome of inappropriate antidiuretic hormone (SIADH) from CNS disease (stroke, SAH), pulmonary disease (pneumonia), malignancy (small-cell lung), drugs (SSRIs, carbamazepine, cyclophosphamide, MDMA), hypothyroidism, primary polydipsia, low-solute diet (beer potomania/tea-and-toast).
- **Hypervolemic:** Heart failure, cirrhosis, nephrotic syndrome, advanced chronic kidney disease.

Risk factors

- Older age, low body mass, female sex (thiazide sensitivity), renal impairment.
- Recent surgery, pain/nausea, psychiatric illness with polydipsia.
- Chronic low solute intake, strict low-salt diets.

Common triggers

- Hypotonic IV fluids, rapid free-water intake.
- New or dose-increased drugs that enhance ADH effect or thirst (SSRIs, opioids, NSAIDs, desmopressin).
- Acute CNS or pulmonary infections; adrenal crisis.

Children (including neonates and infants)

Causes (age - patterned)

- **Neonates/infants:** Gastroenteritis with excess free water, over-diluted formula or inappropriate water supplementation, hypotonic maintenance IV fluids, SIADH from bronchiolitis/pneumonia/meningitis, congenital adrenal hyperplasia or adrenal insufficiency, renal/cerebral salt wasting.
- **School - age/adolescents:** Exercise-associated hyponatremia (over-drinking hypotonic fluids), drug-induced SIADH (SSRIs, oxcarbazepine/carbamazepine, desmopressin for enuresis), CNS infections/trauma; hypervolemic states (heart failure, nephrotic syndrome) less common but possible.

Risk factors

- Prematurity, immature renal handling, high insensible losses with fever.
- Caregiver-determined feeding/fluid practices; formula preparation errors.
- Post-operative status; chronic neurologic disease; endocrine/metabolic disorders.

Common triggers

- Hypotonic IV fluids; unrestricted water access in hot weather or post-sports.
- Rapid catch-up feeds or free-water boluses during illness.
- New psychoactive or antiepileptic medications; desmopressin use.
- Acute CNS or respiratory infections precipitating SIADH.

EVALUATION FOR DIAGNOSIS

Clinical features

- Onset: acute (<48 h) vs chronic (≥48 h).
- Symptoms: nausea, headache, confusion, lethargy; severe seizures, coma, respiratory arrest.
- History: fluid intake pattern; medications (diuretics, SSRIs, carbamazepine, NSAIDs, desmopressin); comorbidities (heart failure, cirrhosis, renal disease, hypothyroidism, adrenal insufficiency).

Physical examination

- Hydration: poor skin turgor; orthostatic hypotension/tachycardia.
- Volume overload: elevated JVP, peripheral edema, ascites.

Laboratory workup

- Confirm hypotonic hyponatremia: serum sodium and osmolality.
- Etiology: urine sodium and osmolality (renal vs non-renal loss; SIADH pattern).
- Organ/endocrine screen: creatinine/BUN, TSH, morning cortisol.
- Glucose to exclude translocational hyponatremia.

Children (including neonates and infants)

Clinical features

- Rapid onset in neonates/infants with subtle warnings: irritability, poor feeding, vomiting, high-pitched cry, apnea.
- Feeding history: breast/formula type, preparation accuracy, recent changes.
- Recent illnesses: gastroenteritis, bronchiolitis, pneumonia, meningitis; fever with poor intake.
- Caregiver input: urine output, stool frequency, hydration behaviors.
- Older children/adolescents: hot-weather sports, excess water intake, behavioral polydipsia.

Physical examination

- Hydration: assess skin turgor over abdomen/thigh; capillary refill and extremity temperature.
- Infants: sunken fontanelle suggests hypovolemia; bulging fontanelle suggests raised intracranial pressure from severe hyponatremia.

Laboratory workup

- Use age-adjusted reference ranges.
- Core tests: serum sodium/osmolality; urine sodium/osmolality.
- Early blood gas in the acutely ill.

- If congenital adrenal hyperplasia or salt-wasting suspected: 17-hydroxyprogesterone, cortisol, renin/aldosterone (as per local protocol).

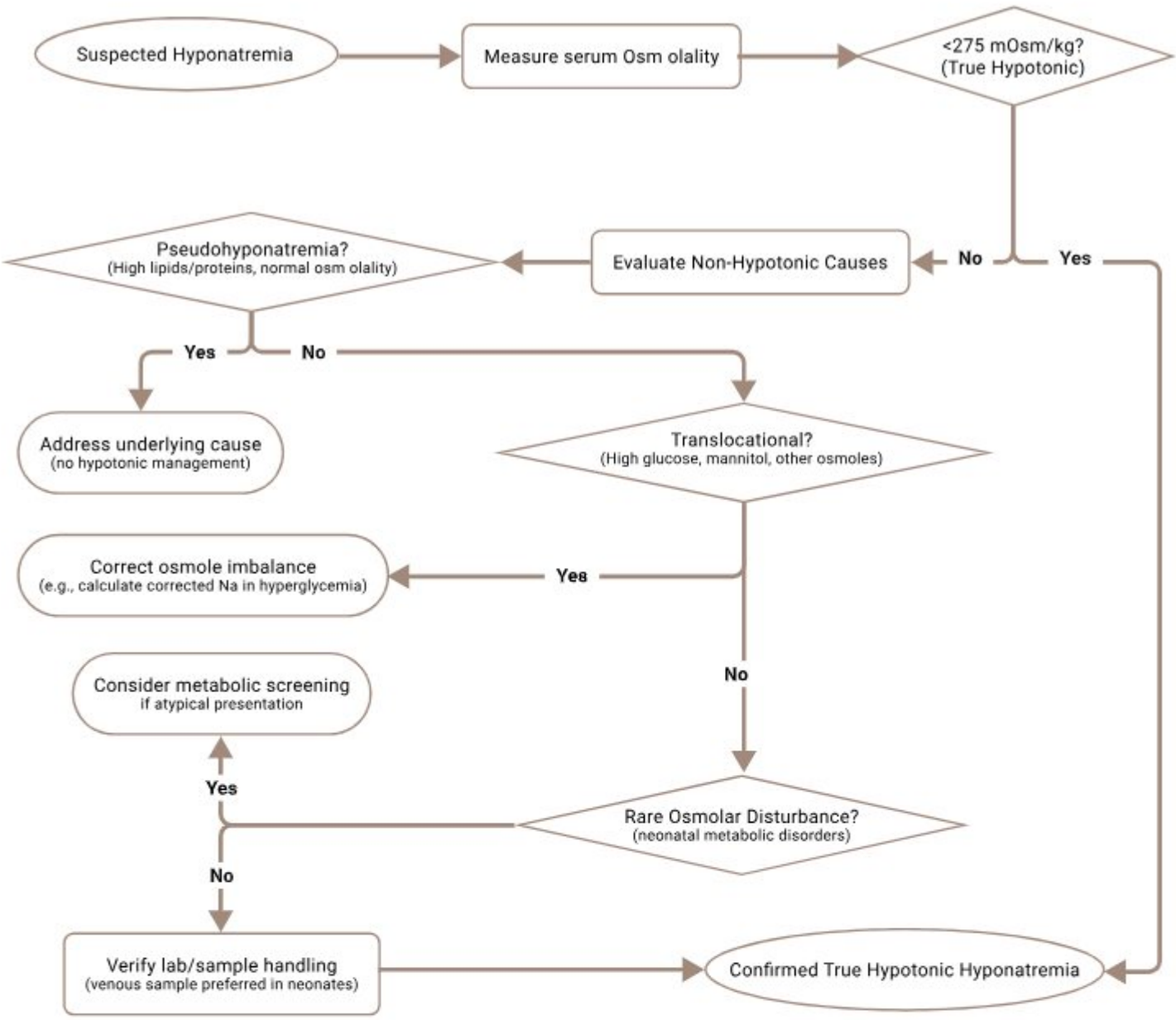
EVALUATION FOR DIAGNOSIS

Classification of hyponatraemia based on volume status and urinary sodium concentration.

Volume status	Urine sodium (mmol/L)	Likely mechanism	Common causes	Clues
Hypovolemic	≤30	Extrarenal sodium loss with water replacement	Vomiting/diarrhea, third spacing (pancreatitis, peritonitis), burns, hemorrhage	Orthostasis, dry mucosa, high BUN:Cr, low skin turgor
Hypovolemic	>30	Renal salt loss	Thiazide/loop diuretics, mineralocorticoid deficiency (Addison disease), salt-wasting nephropathies, cerebral salt wasting	Hyponatremia with natriuresis, history of diuretics, hyperkalemia in Addison
Euvolemic	>30	Water retention via inappropriate antidiuretic hormone (ADH) or endocrine disorders	SIADH (CNS, pulmonary, pain, postop), drugs (SSRIs, carbamazepine, cyclophosphamide), secondary adrenal insufficiency, hypothyroidism	Normal exam volume, low serum uric acid, high urine osmolality
Euvolemic	≤30	Low solute intake or excess free water	Primary polydipsia, beer potomania/tea-and-toast diet	Very low urine osmolality, history of high water intake or poor diet
Hypervolemic	≤30	Effective arterial underfilling with avid sodium/water retention, low distal delivery	Heart failure, cirrhosis, nephrotic syndrome	Edema/ascites, low urine Na despite total body Na excess
Hypervolemic	>30	Renal failure with impaired sodium handling	Advanced chronic kidney disease, acute kidney injury	Edema with azotemia, variable urine osmolality

Note:

- First, verify that serum osmolality is low (<275 mOsm/kg) to confirm true, hypotonic hyponatremia. If osmolality is normal or elevated, consider non-hypotonic causes:
- Pseudohyponatremia: Laboratory artifact when high lipids or proteins displace plasma water. Serum osmolality remains normal.
- Translocational (hypertonic) hyponatremia: Excess osmoles, such as glucose in hyperglycemia or mannitol, pull water into the extracellular space, lowering measured sodium while effective osmolality is high.
- In hyperglycemia, always calculate corrected sodium before deciding management.



Pediatric - Specific Considerations

- Use age-appropriate reference ranges for osmolality; normal neonatal values may be slightly lower than in older children and adults.
- In neonates, pseudohyponatremia can occur with extreme hyperbilirubinemia, high serum triglycerides (e.g., from parenteral nutrition), or certain rare metabolic disorders, laboratory methodology should be verified.
- Translocational hyponatremia in children often follows severe hyperglycemia in diabetic ketoacidosis (DKA) or after administration of hyperosmolar agents (e.g., mannitol for raised intracranial pressure).

- In critically ill infants, unmeasured osmoles from inborn errors of metabolism (e.g., organic acidemias) may produce mixed osmolar disturbances, consider targeted metabolic screening if presentation is atypical.
- Confirm sample handling quality, as capillary heel-stick samples in neonates are more prone to dilution or artifact than venous samples.

Only after excluding these conditions should management proceed for true hypotonic hyponatremia.

Classification & Severity Assessment

Severity is determined by serum sodium level and the presence or absence of symptoms. As sodium falls, the risk of neurologic injury increases, and in pediatric patients, especially neonates, the onset of symptoms can be faster and more severe.

Severity	Serum Sodium	Adults -Presentation	Pediatrics -Presentation	Pediatric - Specific Notes
Mild	130-134 mEq/L	Often asymptomatic; may have thirst or mild nausea.	Subtle irritability, decreased feeding, mild lethargy, or behavioral changes in older children; often incidental finding on labs.	Symptoms can appear at higher sodium levels in neonates; interpret in context of age norms.
Moderate	125-129 mEq/L	Headache, malaise, subtle confusion, gait disturbance.	Poor feeding, persistent vomiting, irritability, sleepiness, behavioral changes; school-aged may show attention deficits or unsteady gait.	Shorter monitoring intervals (2-4 h) during correction to detect deterioration early.
Severe	<125 mEq/L (or any <120 mEq/L)	Marked confusion, seizures, coma, respiratory arrest; needs urgent intervention and continuous monitoring.	High risk of seizures, apnea, bulging fontanelle (infants), rapid neurological decline; in neonates, low 120s can cause cerebral edema and collapse.	Earlier intervention thresholds; critical care support required immediately.

DIFFERENTIAL DIAGNOSIS

Distinguish true hypotonic hyponatremia from other causes of low measured sodium or dilutional states.

Cause	Adults	Pediatrics
Pseudohyponatremia	Laboratory artifact from high serum lipids or proteins reducing plasma water fraction. Serum osmolality remains normal. Confirm with lipid/protein measurement; use direct ion-selective electrode method.	May occur in neonates/infants on parenteral nutrition with high lipid content, with severe hyperbilirubinemia, or rare metabolic disorders causing hyperlipidemia. Capillary heel-stick samples in newborns prone to dilution errors, prefer venous sampling.
Hyperglycemia - Induced Hyponatremia	Elevated glucose increases extracellular osmolality, shifting water from cells to plasma, diluting sodium. Serum osmolality is high. Corrected Na ~ measured Na + [1.6 × (glucose - 100)/100].	Common in diabetic ketoacidosis (DKA), especially in adolescents. Corrected sodium is essential to guide safe fluid therapy and avoid masking concurrent hypernatremia or worsening cerebral edema risk.
Beer Potomania/Low solute feeding	Very low solute intake from excessive beer consumption limits renal free-water excretion. Urine osmolality <100 mOsm/kg. Associated with heavy beer use and poor dietary intake.	No direct equivalent; similar physiology in low-solute feeding, e.g., over-diluted formula or exclusive breast milk in older infants without complementary foods, reducing renal water excretion capacity.
Psychogenic Polydipsia	Massive voluntary water intake overwhelms renal excretion capacity. Urine osmolality maximally dilute (<100 mOsm/kg). Often linked to psychiatric illness.	Seen in adolescents with psychiatric disorders or developmental delay with compulsive water drinking. In younger children, accidental excessive water ingestion (e.g., pool water swallowing, inappropriate free-water use in hot weather) may cause similar presentation.

MANAGEMENT GOALS & PRINCIPLES IN HYPONATREMIA (ER/ICU)

For all patients

- Restore serum sodium safely.
- Treat the underlying cause.
- Maintain hemodynamic stability. Prevent complications from rapid correction and from ongoing cerebral edema.
- Use frequent labs and bedside neurologic checks to guide every step.

However, the approach differs because of physiological, metabolic, and safety considerations in children.

Adults

Initial stabilization

- Secure airway, breathing, circulation. Treat acute neurologic symptoms (e.g., seizures) promptly with hypertonic saline before gradual correction.
- Give hypertonic saline for severe neurologic symptoms: 3% NaCl 100 mL IV over 10 minutes; may repeat 1-2 times until symptoms abate or sodium rises by 4-6 mmol/L.

Correction targets

- After the initial 4-6 mmol/L rise, limit to ≤8-10 mmol/L in 24 hours.
- In chronic hyponatremia, aim around ≤0.5 mmol/L per hour.

Volume - status approach (Fluid restriction)

- Tailor to severity and urine osmolality; Spread intake evenly through the day to avoid sodium swings.
- Hypovolemic: 0.9% saline 250-500 mL, titrate to euvolemia; address GI losses and diuretics.
- Euvolemic SIADH: fluid restriction, use about 800-1,000 mL/day, isotonic maintenance, treat trigger; consider loop diuretic if urine osmolality is high; consider oral salt; use vaptans only with inpatient monitoring when other measures fail.
- Hypervolemic: fluid and sodium restriction, loop diuretic, disease-specific therapy (heart failure, cirrhosis, nephrotic syndrome).

Monitoring during active correction

- Check serum sodium every 4-6 hours, then daily when stable.
- Focused neuro exam every 1-2 hours in the acute phase: consciousness, gait, focal deficits.
- Strict input/output.

Daily monitoring

- Record daily weight and fluid input/output.
- Arrange frequent outpatient sodium checks until stable.

If sodium increases to >8 mEq/L in 24 h

- Give DDAVP (controlled dose) and a small hypotonic bolus to slow/partly reverse the rise.

- Recheck labs in 2-4 hours.

Step - up (ongoing severe symptoms)

- Escalate to a controlled hypertonic saline infusion with ICU monitoring.

Step - down (near 130 mEq/L and symptoms resolved)

- Transition to fluid restriction plus isotonic maintenance fluids.
- Set up outpatient monitoring plan.

Preventing over - or under - correction

- If sodium is rising too fast or exceeds plan, stop hypertonic fluids and consider desmopressin with small volumes of D5W to re-lower gradually.
- Avoid abrupt withdrawal of desmopressin if used to cap diuresis.
- Review and stop precipitating drugs when possible: thiazides, SSRIs, carbamazepine, NSAIDs.

Dietary solute

- Keep normal protein and salt intake.
- Consider salt tablets with meals if tolerated.

Behavioral measures

- Avoid excess water, hypotonic drinks, and “zero-calorie” beverages.
- For psychogenic polydipsia, use behavioral therapy and a structured drinking schedule.

Discharge and follow - up

- Stable sodium trajectory for at least 24 hours.
- Clear plan for cause, medications, and monitoring.

Children (neonates and infants)

Initial stabilization

- ABCs as in adults, but dose everything by weight.
- Hypertonic saline for seizures or severe encephalopathy: 3% NaCl 2-4 mL/kg IV over 10-20 minutes (max about 100 mL); may repeat once or twice until symptoms improve or sodium rises by 4-6 mmol/L.

Correction targets

- Same rescue target initially, then total rise ≤8 mmol/L in 24 hours.
- Neonates and infants are highly vulnerable; calculate all fluids precisely.

Age - specific causes to address

- Infants: gastroenteritis, over-diluted formula, excess free water, hypotonic IV fluids, CNS or respiratory infection with SIADH, adrenal disorders, renal salt-wasting.
- Older children/adolescents: exercise-associated hyponatremia from over-drinking, drug-induced SIADH (SSRIs, carbamazepine).

Volume - status approach

- Hypovolemic: 0.9% saline 10-20 mL/kg bolus; reassess; continue isotonic fluids and replace ongoing GI losses.
- Euvolemic SIADH: restrict to about 60-80% of maintenance; use isotonic maintenance; treat the trigger; consider oral NaCl 1-3 mmol/kg/day in divided doses and a loop diuretic if urine osmolality remains high. Avoid vaptans and demeclocycline.
- Hypervolemic: careful fluid and sodium restriction, loop diuretic, and disease-specific therapy with specialist input.

Monitoring during active correction

- Check serum sodium every 2-4 hours (higher vigilance in neonates/infants).
- Age-appropriate neuro checks: feeding, cry, alertness, fontanelle tension, seizure activity; in preverbal children, watch for irritability or decreased responsiveness.

Daily monitoring

- Weigh daily; in neonates/infants, weigh more frequently during admission.
- Track feed volumes, urine, and stool with caregiver input.
- After discharge, check sodium every 2-4 days until normal.

If sodium increases to >8 mEq/L in 24 h

- DDAVP **0.3 µg/kg IV/SC** plus a weight-calculated hypotonic bolus.
- Repeat labs in 2-4 hours to confirm stabilization.

Step - up (ongoing severe symptoms)

- 3% saline **2-4 mL/kg IV** over 10-15 minutes; may repeat once if seizures persist.
- Continuous cardiorespiratory and neurologic monitoring in PICU.

Step - down (near 130 mEq/L and symptoms resolved)

- Gradually return to maintenance fluids; avoid over-restriction in infants.
- Arrange close outpatient follow-up with sodium checks every 2-4 days and caregiver education on limits and early warning signs.

Nutrition and safety

- Do not impose very tight fluid restriction without a feeding plan. Prevent hypoglycemia and poor caloric intake.
- For infants, plan with a dietitian to meet calories and prevent malnutrition; distribute feeds/fluids evenly over 24 hours.
- Older children: encourage age-appropriate protein and salt.
- Infants: adjust formula sodium only under specialist guidance; avoid excess to prevent hypernatremia.

Preventing over - correction

- Watch for sudden water diuresis as antidiuretic hormone wanes.
- If sodium rises too fast, pause therapy and use D5W and/or low-dose desmopressin to re-establish a safe slope.

Behavioral measures

- Adolescents with psychiatric/developmental disorders: involve caregivers in fluid scheduling.
- Younger children: limit unsupervised water access, especially in hot weather or after sports.
- Confirm correct formula preparation; avoid free-water supplements.

Discharge and follow - up

- Stable sodium for at least 24 hours, caregiver teaching on warning signs, and early outpatient labs if risk persists.

Correct sodium at safe rates

Use hypertonic saline judiciously for severe or symptomatic cases; isotonic or hypotonic fluids for other types based on volume status. Adjust infusion rates to prevent rapid shifts. **Pediatric - specific: Always use weight - based dosing (e.g., 3% saline bolus at 2-4 mL/kg for seizures). In acute symptomatic hyponatremia, correction may initially be faster but must slow once symptoms resolve. In chronic cases, keep correction ≤8 mmol/L in 24 hours. Avoid large - volume boluses in infants to prevent sudden overcorrection.**

Monitor neurological status and serum sodium frequently

Check mental status and neurologic signs every 1-2 hours in acute management. Measure serum sodium every 4-6 hours during active correction, then daily once stable. **Pediatric - specific: In neonates and infants, monitor every 2-4 hours during active correction, as rapid shifts can cause seizures or apnea even within a short interval. Use age - appropriate neurologic assessment, irritability, feeding difficulty, altered cry, bulging fontanelle, or abnormal posturing may be early warning signs before overt seizures occur.**

PHARMACOLOGICAL THERAPY

1. Choose agents based on severity, volume status, and underlying etiology (see Table).

Selection Principles

- Use hypertonic saline only for life-threatening symptoms such as seizures, coma, or signs of impending herniation.
- In hypovolemic cases, restore intravascular volume with isotonic fluids before addressing water-sodium balance.
- In SIADH or hypervolemic states, combine fluid restriction with aquaretics (vaptans, urea) or loop diuretics if indicated.

2. **Monitoring During Active Correction**

- **Adults:** Neurologic exam every 1-2 hours during hypertonic therapy; serum sodium every 4-6 hours until within safe correction limits, then daily.

Pediatrics: Neurologic exam every 1-2 hours; serum sodium every 2-4 hours during active correction (especially in neonates/infants) due to higher cerebral edema risk. Use age - appropriate neuro checks (feeding behavior, alertness, cry, fontanelle).

3. **Adjustment**

If sodium rises >8 mEq/L in 24 hours, pause therapy and consider administering DDAVP to slow correction. In children, err on the side of slower correction if clinical status allows.

4. **Medication Review:** Stop or reduce drugs that cause water retention (NSAIDs, SSRIs, certain anticonvulsants). Substitute safer alternatives when possible. Review prescriptions for agents like vincristine, cyclophosphamide, carbamazepine, desmopressin. Adjust doses for age/weight; ensure sodium monitoring is in place.

5. **Drug and fluids**

Situation	Adults	Children
Severe symptoms (seizure, coma)	3% NaCl 100 mL IV over 10 min, repeat 1-2times to raise Na by 4-6 mmol/L	3% NaCl 2-4 mL/kg IV over 10-20 min, repeat as needed to raise Na by 4-6 mmol/L
Hypovolemic hyponatremia	0.9% NaCl 250-500 mL, titrate	0.9% NaCl 10-20 mL/kg bolus; reassess
Loop diuretic (overload/ SIADH adjunct)	Furosemide 20-40 mg IV/PO	Furosemide 0.5-1 mg/kg/dose IV/PO q6-12h

Oral salt adjunct	3-9 g NaCl/day in divided doses	1-3 mmol/kg/day Na in divided doses
Vaptans	Consider in selected SIADH, inpatient only	Not recommended; specialist exceptions only
	Tolvaptan 15 mg PO once; may increase to 30-60 mg daily; ≤30 days **Caution:** Hepatotoxicity; initiate in hospital; avoid if unable to sense thirst	Not routinely used in <18 years; safety and efficacy not established, pediatric use only in research/specialist settings
	Conivaptan 20 mg IV over 30 min, then 20 mg/day continuous IV infusion (max 4 days) **Caution**: IV only; phlebitis risk; monitor site	Not approved for pediatric use; consult pediatric nephrology/endocrinology
Demeclocycline	Chronic SIADH when vaptans unavailable/ contraindicated 600 mg PO BID; may increase to 600 mg TID for 3-7 days initial, then adjust **Caution**: Nephrotoxicity; photosensitivity	Contraindicated in children <8 years (risk of teeth/bone effects)

Note:

Hyponatremia: Adult chronic hyponatremia: correct ≤8-10 mmol/L in 24 h where in a child acute hyponatremia with seizures: small bolus of 3% saline (2-4 mL/kg) rapidly, then slower correction.

Hyperkalemia: Adults: IV calcium gluconate dose fixed (e.g., 10 mL of 10% over 2-5 min) where as in children: IV calcium gluconate 0.5-1 mL/kg of 10% solution (max dose) to avoid overdose.

Additional Cautions During Active Correction

- *Use Central Access for Hypertonic Saline:* Peripheral veins tolerate only limited hypertonicity. Central line reduces risk of phlebitis and tissue injury.
- *Avoid Hypotonic or Free-Water Loads:* Do not give hypotonic fluids (e.g., D5W) or allow large oral water intake during correction phase; this undermines controlled sodium rise.
- *Account for Ongoing Losses:* Monitor input and output. Unmeasured losses (vomiting, sweat, diuretics) can accelerate sodium changes unexpectedly.
- *Adjust for Concurrent Medications:* Hold or dose-reduce agents that affect ADH (NSAIDs, SSRIs, carbamazepine). Reintroducing them too soon may blunt response.
- *Watch for Volume Shifts:* In hypovolemic patients, over-rapid volume restoration can precipitate pulmonary edema or worsen heart failure.

- *Avoid Rapid DDAVP Withdrawal:* If DDAVP was used to control water diuresis, taper rather than stop abruptly to prevent sudden sodium jumps.
- *Monitor Electrolytes Beyond Sodium:* Potassium and magnesium shifts influence free-water handling and may worsen over-correction if neglected.
- *Reassess Neurologic Status Continuously:* Frequent checks detect early signs of osmotic demyelination (e.g., dysarthria, ataxia) or cerebral edema (headache, bradycardia).
- *Have a "Rescue" Plan:* If sodium rises >10 mEq/L in 24 h, pause hypertonic fluids and consider a small DDAVP dose with hypotonic fluid to re-lower sodium safely.

PROGNOSIS AND PROGRESSION

When hyponatremia is corrected at safe rates, most patients, adults and children, regain normal sodium levels without lasting harm. Acute cases treated within 48 hours carry a very low risk of permanent injury in both groups. In adults, chronic or severe hyponatremia can leave residual effects such as gait instability, subtle cognitive slowing, or attention deficits. ***In pediatric patients, particularly neonates and infants, prolonged or severe hyponatremia increases the risk of long - term neurodevelopmental impairment, motor delays, and learning difficulties due to greater vulnerability of the developing brain to osmotic shifts.***

REFERRAL CRITERIA

Refer to a higher-level center when:

When to Escalate Care	
Adults	Pediatrics
Severe neurologic symptoms (seizures, coma) persist despite initial management.	Ongoing seizures, altered consciousness, or raised ICP signs despite initial therapy.
Serum sodium fails to improve or recurs after correction.	Persistent/recurrent hyponatremia after correction, especially in neonates/infants.
Need for specialized therapies (vasopressin antagonists, urea therapy, endocrine testing).	Need for specialized therapies or rare-cause workup (e.g., CAH, hypothalamic-pituitary disorders).
Underlying cause unclear after standard evaluation.	Unclear etiology after full age-appropriate workup.
Complex comorbidities (advanced HF, cirrhosis, renal failure) complicate management.	Underlying conditions needing multidisciplinary pediatric care (e.g., congenital heart disease, CKD, liver failure).
	PICU-level monitoring required due to rapid sodium shifts or unstable hemodynamics.

COMPLICATIONS

Adults	Pediatrics
Acute Cerebral Edema: Rapid Na drop → water influx into brain cells lead to swelling, raised ICP, headache, vomiting, altered consciousness, risk of herniation.	Acute Cerebral Edema: Higher risk due to low intracranial compliance in infants. Can present with bulging fontanelle, irritability, seizures, apnea, rapid neuro decline.
Osmotic Demyelination Syndrome: Rapid Na correction leads to water loss from brain cells and myelin breakdown (pons). Delayed onset dysarthria, dysphagia, quadriparesis, "locked-in" syndrome. Often incomplete recovery.	Osmotic Demyelination Syndrome: Rare but possible with rapid correction, especially in chronic cases; manifests with delayed neurologic deficits.
Falls & Fractures: Mild chronic hyponatremia impairs balance & attention, increases fall risk, hip/wrist fractures, prolonged hospital stay, loss of independence.	Feeding/Developmental Impact: In recurrent or prolonged hyponatremia, feeding difficulties, failure to thrive, and developmental delays may occur if unrecognized.

PREVENTION AND HEALTH PROMOTION

- **Limit hypotonic IV fluids:** Use isotonic solutions by default. Reserve hypotonic fluids (e.g., 0.45% saline) for specific indications only. This minimizes the risk of diluting serum sodium in vulnerable patients.
- **Monitor at - risk groups:** Check serum sodium regularly in older adults, endurance athletes and psychiatric patients prone to polydipsia. Early lab monitoring lets you catch trends before symptoms develop.
- **Promote balanced electrolyte intake:** Advise exercisers to use drinks with measured sodium content rather than plain water. Encourage modest salt snacks or broths during prolonged activity. This prevents overhydration and supports steady electrolyte levels.

Patient Education

- Empower patients to follow prescribed fluid limits. Clearly explain daily volume goals, why restricting fluids prevents worsening hyponatremia and how to measure intake.
- Teach recognition of warning signs. Ensure patients and caregivers know how to report nausea, headache, confusion or dizziness at onset rather than waiting for severe symptoms.
- Reinforce the importance of follow-up. Stress attending scheduled lab checks and clinic visits so adjustments can be made before complications arise.

Instructions to the Patient/Caregiver

- Measure all fluid intake. Use a marked container or water bottle to track daily volume.
- Weigh yourself each morning. Report a gain or loss of more than 1 kg in a day.
- Note and report early symptoms immediately: headache, nausea, confusion, dizziness.
- Stick to prescribed fluid limits. Do not sip extra water or hypotonic drinks without approval.
- Take medications as directed. Do not skip doses or stop suddenly.
- Follow dietary advice: include moderate salt or electrolyte snacks if recommended.
- Keep all lab and clinic appointments. Bring fluid-intake logs and weight records.
- Avoid self-medicating with diuretics or herbal remedies. Check with your doctor first.
- Seek urgent care if you develop seizures, severe confusion or difficulty breathing.

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HYPERNATREMIA

QUICK REFERENCE GUIDE

Hypernatremia is an electrolyte imbalance where serum sodium exceeds 145 mmol/L, usually due to water deficit rather than sodium excess. It affects all ages but is most common in neonates, infants, and frail elderly because of impaired thirst or feeding errors. Causes include diarrhea, vomiting, diabetes insipidus, inadequate water intake, and iatrogenic sodium load. Morbidity includes neurologic injury from brain cell shrinkage, seizures, and coma, while mortality rises sharply when Na^+ >155 mmol/L. With timely, controlled correction, most patients recover, but delayed or rapid correction can lead to cerebral edema and long-term cognitive or developmental deficits, especially in infants and older adults.

Definition

Hypernatremia is defined as serum sodium (Na^+) > 145 mmol/L with elevated serum osmolality (usually > 295 mOsm/kg). By acuity: Acute (<48 h) vs chronic/unknown (≥48 h). By volume status: Hypovolemic (water loss > sodium loss), euvolemic (pure water deficit, e.g., diabetes insipidus), hypervolemic (sodium gain > water gain).

Causes, Risk factors & Triggers

- Hypovolemic: Gastrointestinal losses (diarrhea, vomiting), osmotic diuresis, diuretics, burns, excessive sweating, poor access to water.
- Euvolemic: DI (diabetes insipidus - central or nephrogenic), inadequate intake (impaired thirst, sedation), high insensible loss (fever, tachypnea).
- Hypervolemic: Iatrogenic sodium load (hypertonic saline, sodium bicarbonate, tube feeds), hyperaldosteronism, seawater ingestion.
- Risk groups: Neonates, infants, frail elderly, patients with cognitive impairment, ventilated or sedated patients.
- Common triggers: Fever, uncontrolled hyperglycemia, tube-feed concentration errors, laxatives/osmotic cathartics, desiccating environments.

Evaluation for Diagnosis

- **Clinical features:** Thirst, dry mucosa, lethargy, irritability; severe: confusion, seizures, coma. Children: poor feeding, high-pitched cry, irritability, decreased urine, apnea.
- **Physical examination:** Volume status: orthostasis, capillary refill, mucous membranes, jugular venous pressure, edema/ascites.
- **Neurologic screen each review;** infants - fontanelle (sunken = dehydration; bulging = raised intracranial pressure).
- **Laboratory investigations:** Serum: Na^+, K^+, Cl^-, HCO_3^-, urea/creatinine, glucose, serum osmolality. Urine: osmolality and sodium (differentiate central DI vs nephrogenic DI vs extrarenal losses). Consider cortisol, thyroid-stimulating hormone if etiology unclear; blood gas in the unwell child.

Confirmation of diagnosis

- True hypernatremia = high Na^+ with high effective osmolality; exclude pseudohypernatremia (rare).

Classification / severity assessment criteria

- **Mild:** 146-150 mmol/L
- **Moderate:** 151-160 mmol/L
- **Severe:** >160 mmol/L
- **High-risk contexts:** Chronic/unknown duration, malnutrition, liver disease, alcoholism, pregnancy, neonates/infants.

Differential Diagnosis

- Hyperglycemia-related water shifts (usually cause hyponatremia; review measured vs corrected Na^+).
- Osmotic loads (mannitol/urea).
- Pseudohypernatremia (severe hyperlipidemia/proteinemia with indirect ion-selective electrode methods).
- Mixed disorders (simultaneous hypovolemia + DI).

Management Goals & principles

- Restore intravascular volume if shocked.
- Replace free water to reduce Na^+ ≤10 mmol/L in 24 h (~0.25-0.5 mmol/L/h).
- Slower correction if chronic/unknown (often over 48-72 h).
- Treat the cause (losses, DI, sodium load).
- Monitor Na^+ and neurologic status frequently; adjust the plan to the slope.

Approach to management

1. Stabilize: ABCs (airway, breathing, circulation), oxygen, IV access; bedside glucose.
2. Classify: Acute vs chronic; hypovolemic/euvolemic/hypervolemic; adult vs pediatric dosing.
3. Volume first (if hypovolemic): 0.9% saline boluses (adults 250-500 mL; children 10-20 mL/kg).
4. Estimate free-water deficit:
   - TBW (total body water) = 0.6×wt (men), 0.5×wt (women), 0.7 neonates/infants.
   - Deficit (L) = TBW × [(Na^+/140) − 1].
5. Replace free water: Prefer enteral if safe; IV by pump (no hypotonic boluses).
6. Set rate: Target Na^+ fall 0.25-0.5 mmol/L/h; cap 24-h fall at ≤10 mmol/L.
7. Reassess q4-6 h (adults) / q2-4 h (children): Titrate rate/tonicity to stay on slope.
8. Address ongoing losses: Replace mL-for-mL; account for fever/insensible losses.
9. Nutrition: Avoid over-restriction that compromises calories (key in infants/older adults).

Non - Pharmacological interventions

- Use oral/NG water when safe; WHO-style oral rehydration solution for mixed losses.
- Simple tools: marked bottles, measured cups, intake/output charts, daily weight.
- Shade/cooling for febrile or sweating patients; caregiver teaching on feed preparation.
- If limited labs: smaller, steady adjustments with clinical monitoring; avoid rapid shifts.

Pharmacological therapy

- **Desmopressin (DDAVP) - central DI (confirmed or highly suspected)**
  - Adults: 1-2 µg IV/SC or 10-20 µg intranasal q12-24h; titrate to urine osmolality >300 mOsm/kg without causing hyponatremia.
  - ***Children: ~0.3 µg/kg IV/SC (local pediatric protocol); careful lab checks.***
  - *Caution:* Water intoxication if free water not matched to dose response.
- **Thiazide diuretics ± amiloride - nephrogenic DI (reduce urine volume)**
  - Adults: Hydrochlorothiazide 25 mg PO q12-24h; Amiloride 5-10 mg PO daily.
  - *Children:* Specialist dosing (weight-based).
  - *Caution:* Hypokalemia, hyponatremia; monitor electrolytes.
- **Loop diuretics - hypervolemic hypernatremia with sodium excess**
  - Adults: Furosemide 20-40 mg IV/PO; titrate.
  - ***Children*: 0.5-1 mg/kg/dose IV/PO q6-12h.**
  - *Caution:* Volume depletion, electrolyte loss; replace K^+/Mg^{2+}.
- **Dialysis in refractory cases with renal failure or massive sodium load**
  - Individualize sodium bath (dialysis fluid) and rate of correction; ICU/PICU setting.

Electrolyte co - management: Replete potassium/magnesium early; adding Na^+/K^+ to infusate reduces free-water delivery - recalculate rates.

Assessment of response, Review; follow - up and adjustment

Targets: Na^+ fall 0.25-0.5 mmol/L/h; ≤10 mmol/L/24 h.

- **Monitoring:**
  - Adults: serum Na^+ q4-6 h during correction; neuro check each draw.
  - ***Children: serum Na^+ q2-4 h (neonates may need q1-2 h initially).***
- If targets not achieved, actions to be taken:
  - Falling too fast - slow/raise tonicity; consider brief pause.
  - *Too slow - increase free-water fraction/ rate; check for ongoing losses or DI.*
- Before step-down: Symptoms resolved, Na^+ near normal and stable ≥24 h, cause addressed, home plan in place.

Referral / tiered approach

- Primary care: Identify risk, start safe oral/enteral rehydration, urgent referral for neurologic signs or severe dehydration.
- Secondary care: Full diagnostic work-up, controlled free-water replacement, sodium and neuro monitoring at defined intervals.
- Tertiary care: Complex DI (central/ nephrogenic), refractory or mixed etiologies, renal failure needing dialysis, severe pediatric cases specialist-led management.

Complications

- Cerebral hemorrhage (acute cell shrinkage), rhabdomyolysis, acute kidney injury, hypotension/shock.
- Over-rapid correction: in case of cerebral edema, seizures, herniation - avoid by adhering to rate limits.

Objectives of Patient education & Instructions to the patient/ caregiver

- Recognize symptoms early: intense thirst, dry mouth, confusion, lethargy; infants - poor feeding, irritability.
- Follow the fluid plan: exact volumes, timing, and type; do not improvise.
- Track and share: daily intake/output and weight (report ≥1 kg/day change in adults; frequent weights in infants).
- Keep appointments: sodium checks and reviews enable safe adjustments.
- Red flags for emergency care: seizures, sudden confusion/coma, breathing difficulty, very low urine.
- Do: use marked containers, measure feeds, bring logs to visits, clarify doubts early.
- Don't: give free water to infants, dilute formula, binge water or sports drinks outside the plan, change diuretics/salt on your own.

HYPERNATREMIA

INTRODUCTION

Hypernatremia is serum sodium >145 mmol/L and almost always reflects a water deficit rather than true sodium overload. Cells shrink as water leaves, driving neurologic and cardiovascular injury. It affects roughly 1-4% of hospital admissions (up to 6% develop it in-house), with higher rates in ICUs; neonates and the elderly are at greatest risk because of impaired thirst or concentrating ability. Common drivers are poor intake and increased losses (fever, diuretics, diarrhea, insensible loss), especially during acute illness. Mortality is higher than in normonatremia (~12% vs 2%), and risk rises further when Na^+ >155 mmol/L. Preliminary data, however, suggest that in adults with severe hypernatremia, more aggressive, rapid correction, regardless of chronicity, can improve outcomes without increasing neurological risk. This evolving evidence prompts reconsideration of strict time-based protocols in favor of severity-guided correction strategies.

Neonates and elderly face the highest risk due to immature or reduced renal concentrating ability, impaired thirst, cognitive deficits and comorbidities. Community-acquired cases arise in frail, elderly patients with impaired thirst or limited access to fluids, often during acute infections. Hospital-acquired hypernatremia affects patients across all adult age groups similarly to the general inpatient population. In both settings, reduced water intake and increased losses (fever, diuretics, diarrhea, insensible losses) drive hyperosmolarity. As extracellular fluid becomes hypertonic, water shifts out of neurons, causing cell shrinkage and potential brain injury. Volume loss may also precipitate tachycardia, hypotension and end-organ hypoperfusion. Common mistakes: ignoring ongoing losses and overcorrecting with unchecked hypotonic infusions or precipitous DDAVP in diabetes insipidus

SCOPE OF THESE GUIDELINES

This guideline addresses hypernatremia in both adults and pediatric patients, from initial recognition to stabilization, treatment, and follow-up. It applies across healthcare levels and includes age-specific considerations for diagnosis, correction rates, and monitoring. Hypernatremia related solely to surgical fluid shifts is excluded.

Intended users

Physicians, pediatricians, specialists, nurses, and allied healthcare professionals at primary, secondary, and tertiary care levels.

Primary Care

- **Adults:** Identify high-risk patients (elderly, impaired thirst, diuretic use), initiate safe oral/enteral rehydration, and recognize red flags for referral.
- **Pediatrics:** Identify risk in neonates, infants, and children with diarrhea, feeding errors, or fever; provide initial weight-based rehydration; activate urgent referral when neurologic symptoms or severe dehydration are present.

Secondary Care

- **Adults:** Conduct full diagnostic evaluation, initiate controlled free-water replacement, and monitor serum sodium and neurologic status.
- **Pediatrics:** Perform weight-based fluid and electrolyte assessment, correct deficits with strict adherence to pediatric correction rates, and ensure close monitoring every 2-4 hours during active correction.

Tertiary Care

- **Adults:** Manage complex diabetes insipidus (central or nephrogenic), initiate and titrate specialized pharmacologic therapies (DDAVP, thiazides, amiloride), and address refractory or mixed-etiology cases.
- **Pediatrics:** Provide subspecialist-led management of congenital or genetic DI, refractory hypernatremia, or cases requiring advanced therapies (DDAVP titration, combined diuretic regimens, dialysis). Offer caregiver education to prevent recurrence.

DEFINITIONS

Hypernatremia is defined as a serum sodium concentration above 145 mmol/L in both adults and children. It reflects a relative deficit of water in relation to body sodium, most often from water loss, less commonly from sodium gain.

Adults: Usually associated with impaired thirst, limited water access, or excessive water loss from renal, gastrointestinal, or insensible sources.

Pediatrics: Often results from diarrhea, vomiting, high-feeding solute load, incorrect formula preparation, or congenital/endocrine disorders; neonates and infants are at higher risk due to greater baseline water turnover and immature renal concentrating ability.

Hypernatremia is always a marker of disturbed water balance and requires prompt evaluation of onset, duration, and underlying cause to guide safe correction.

CAUSES, RISK FACTORS & TRIGGERS

Category	Details	Pediatric - Specific Considerations
Common Risk Factors	■ Advanced age with reduced thirst sensation ■ Cognitive/physical impairment limiting access to fluids ■ Uncontrolled diabetes mellitus (osmotic diuresis) ■ Polyuria disorders (central/nephrogenic DI) ■ Nursing home residency, inadequate caregiving ■ Hospitalization with sedatives or hypertonic infusions	■ High water turnover, immature renal concentrating ability (neonates/infants) ■ Incorrect formula preparation ■ Gastroenteritis, vomiting, diarrhea ■ Fever/high ambient temperature without adequate fluids ■ Congenital/genetic DI, adrenal disorders ■ Inadequate breastfeeding or feeding interruptions
Hospital - Specific Triggers (Both Groups)	■ Decreased consciousness or sedation ■ Hypertonic saline, lactulose, sodium bicarbonate, excess mineralocorticoids ■ Tube feeding without free water ■ Mechanical ventilation without humidification/ free-water provision	Same mechanisms apply, risk is higher in neonates/infants due to smaller fluid reserves
Mechanisms	**Water Loss:** Renal - DI, osmotic diuresis, diuretics Extrarenal - fever, burns, vomiting, diarrhea, sweating **Sodium Gain:** Iatrogenic - hypertonic saline, sodium bicarbonate, high-sodium enteral feeds Endogenous - mineralocorticoid excess	Children more likely to develop rapid shifts due to smaller total body water; incorrect formula preparation is a frequent cause
Clinical Features	**Mild:** Thirst, dry mucous membranes, irritability, restlessness **Moderate:** Confusion, lethargy, muscle weakness, cramps, tachycardia, orthostatic hypotension **Severe:** Seizures, coma, intracranial hemorrhage, shock	Infants may present with irritability, high-pitched cry, poor feeding, sunken fontanelle
Physical Exam	Dry mucous membranes, poor skin turgor, sunken eyes, tachycardia, orthostatic hypotension	Infants: sunken fontanelle, delayed capillary refill
History Clues	Identify inability to access water (mental status changes, mobility limits, staffing/environmental restrictions) Determine acute (<24h) vs. chronic (>48h) onset	Neonates: feeding history, preparation of formula, breastfeeding adequacy
Common Contexts	**Community - acquired:** Frail elderly with febrile illness, impaired thirst/access to fluids **Hospital - acquired:** Any age, often due to medical interventions or fluid restriction	Neonates: NICU, incorrect IV fluid composition, inadequate feeding during illness
Laboratory Evaluation	**Serum Sodium & Osmolality:** Na >145 mmol/L, osmolality >295 mOsm/kg **Urine Osmolality & Sodium:** Low osmolality (<300 mOsm/kg) → DI; High osmolality + low urine Na → extrarenal water loss	

CONFIRMATION OF DIAGNOSIS

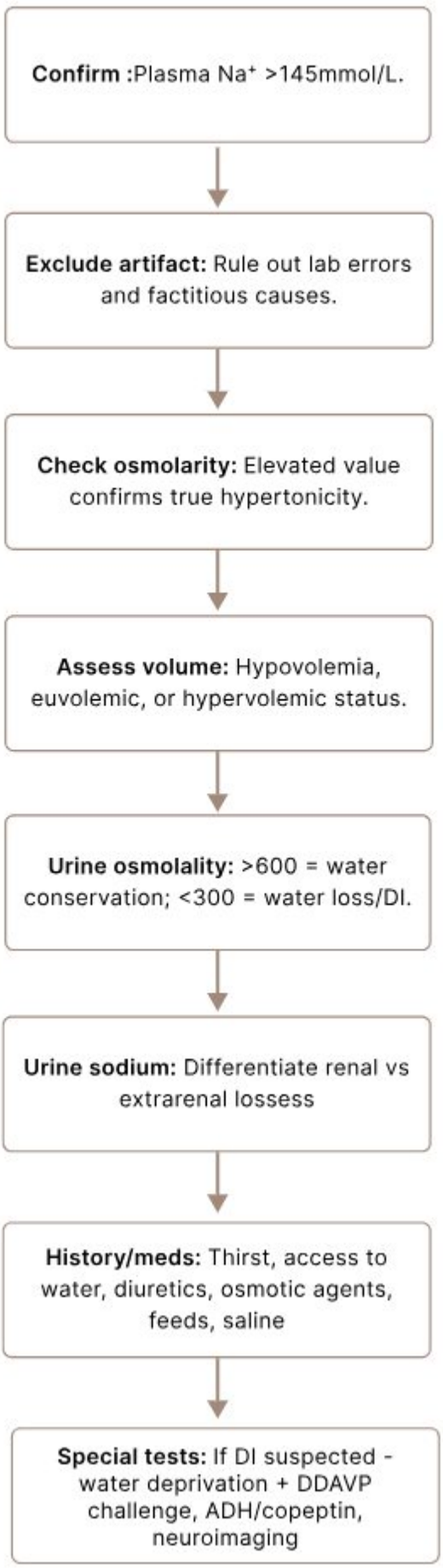
- Verify serum sodium >145 mmol/L using a direct ion-selective electrode.
- Confirm hypertonicity with serum osmolality >295 mOsm/kg.
- Rule out spurious results (e.g., lab artifact) by repeating measurement or using an alternate method if sample lipemia or paraproteinemia is suspected.

CLASSIFICATION & SEVERITY ASSESSMENT

Hypernatremia reflects a disturbance in body water balance rather than a primary disease process. To guide evaluation and management, it is classified by clinical severity, onset and volume status. Each category carries distinct diagnostic and therapeutic implications. Severity helps set targets for safe correction rates. Onset guides urgency, the brain adapts to chronic hypernatremia, so over-rapid correction risks cerebral edema. Volume status directs fluid choice: isotonic or hypotonic fluids for hypovolemia, free water replacement for euvolemia, and diuretics plus free water for hypervolemia.

Severity (serum Na^+ concentration) & Neurological impact	Onset (time of development)	Volume status (clinical assessment)
■ Mild (146-150 mmol/L): often limited to thirst and mild restlessness. ■ Moderate (151-160 mmol/L): confusion, muscle weakness or tachycardia may develop. ■ Severe (>160 mmol/L or any level with neurologic symptoms): seizures, coma or signs of intracranial hemorrhage.	■ **Acute:** Rise in serum sodium within ≤ 48 hours ■ **Chronic:** Rise over >48 hours or time unknown	■ **Hypovolemic:** Loss of both water and s with water loss predominating ■ **Euvolemic:** Net water loss without significant change in total body sodium ■ **Hypervolemic:** Gain of sodium or hypertonic fluids exceeding water gain

Eight diagnostic steps for hypernatremia



DIFFERENTIAL DIAGNOSIS

Distinguishing true hypernatremia from other states is essential because management targets differ radically:

Condition	Mechanism	Clues
Pseudohypernatremia	Lab artifact (high proteins)	Normal osmolality, raised protein levels
Central DI	Low ADH secretion	High urine output, low urine osmolality
Nephrogenic DI	Renal ADH resistance	History of lithium use, CKD
Osmotic diuresis	Glucose/mannitol load	High glucose/mannitol, corresponding osmolar gap

MANAGEMENT GOALS

- Aim to lower serum sodium by no more than 10 mmol/L over 24 hours (~0.5 mmol/L per hour).
- In chronic hypernatremia, extend correction over 48-72 hours, when possible, to reduce cerebral edema risk.
- Stabilize hemodynamics and maintain urine output while avoiding overhydration.

Management principles

- Apply the same core approach to adults and children; in pediatrics use weight-based doses, tighter monitoring, and nutrition safeguards.
- Correct coexisting volume and electrolyte deficits in parallel.
- Replace potassium and/or calcium if low; choose fluids accordingly.
- When adding electrolytes, account for their effect on free-water delivery (add K^+/Na^+ to IV fluids or run separately to preserve planned free water).

PHARMACOLOGICAL THERAPY

Pharmacologic interventions target the underlying cause of water loss rather than correcting sodium directly. In most cases, controlled free-water replacement is primary; drugs limit ongoing losses. They play a central role when hypernatremia results from diabetes insipidus or other disorders of ADH secretion or action. Drugs are adjuncts to fluid replacement. Always monitor serum sodium and volume status every 4-6 h (every 2-4 hours in pediatric patients) during active correction.

Stepwise Fluid Management of Hypernatremia in adults

1. **Stabilization**
   - ABCs, oxygen as needed, IV access, cardiac and pulse-ox monitoring.
   - Bedside glucose; treat hypoglycemia.
   - If shock/hypotension: 0.9% NaCl 250-500 mL boluses, reassess frequently.
   - Avoid hypotonic boluses; all hypotonic fluids by pump only.
2. **Rapid assessment: volume status & neurologic risk**
   - Volume: orthostasis, mucous membranes, JVP, edema/ascites, urine output.
   - Neurologic: mental status, focal signs, seizures; consider aspiration risk.
   - Risk for cerebral edema during correction: chronic/unknown duration, severe Na^+ (>160), malnutrition, advanced liver disease, older age.
3. **Estimate free - water deficit (sets the plan)**
   - TBW: men ~0.6 × wt(kg); women/elderly ~0.5 (frail ~0.45).
   - Deficit (L) = TBW × [(measured Na^+/140) − 1].
   - Add maintenance and ongoing losses (GI, drains, fever/insensible).
4. **Identify and reverse the cause**
   a. Stop/treat drivers of water loss or sodium gain; hold diuretics/lactulose; control vomiting/diarrhea; treat fever or hyperglycemia; fix tube-feed concentrations; relieve obstructions.
5. **Gauge onset and symptom severity**
   a. Acute (≤48 h): lower Na^+ 1-2 mmol/L/h for 6-8 h, then slow (aim ≈145 within 24 h if safe).
   b. Chronic (>48 h): ≤0.5 mmol/L/h (≤12 mmol/L/day).
6. **Calculate water deficit & replacement rate**
   a. Use formula above; plan delivery over 48-72 h, plus replacement of ongoing losses.

7. **Choose the right fluid**
    a. Hypovolemic: resuscitate with 0.9% NaCl, then switch to hypotonic (e.g., D5W or D5 0.45% NaCl).
    b. Euvolemic: D5W or 0.45% NaCl.
    c. Hypervolemic: D5W + loop diuretic; consider dialysis if renal failure or severe sodium load.
8. **Set an initial infusion regimen**
    a. Fixed-dose guide: D5W 1.35 mL/kg/h (slow) or 3 mL/kg/h (~1 mmol/L Na^+ drop per 3 mL/kg of D5W).
    b. Titrate to a fall of ~0.25-0.5 mmol/L/h.
9. **Monitor and adjust frequently**
    a. Serum Na^+ q4-6 h during active correction; full neuro check at each draw.
    b. If falling too fast → reduce rate/increase Na^+ content; if too slow → increase free-water fraction/rate.
    c. Track I/O, weight, urine output; watch glucose with dextrose infusions.
10. **Address diabetes insipidus**
    a. Central DI: DDAVP intranasal 10-20 µg q12-24h or oral 0.1-0.8 mg; titrate to urine Osm >300 mOsm/kg without causing hyponatremia.
    b. Nephrogenic DI: stop offenders; thiazide ± amiloride; low-Na diet; careful free-water replacement. NSAIDs as adjunct (e.g., indomethacin 50 mg PO 2-3 times a day) to reduce urine output. Cautions: GI, renal toxicity; avoid in dehydration.
    c. Partial DI: consider in select cases carbamazepine 200-600 mg/day PO in divided doses or Chlorpropamide: 125-500 mg/day PO. Cautions: Hypoglycemia, hyponatremia.

Hypervolemic Hypernatremia (e.g., sodium overload)

- Loop diuretics (Furosemide 20-40 mg IV/PO q6-12 h) with free-water replacement. Duration: Until sodium normalizes. Cautions: Monitor K^+, Mg^{2+}, volume status.

Hyperglycemia with Hypernatremia

- Insulin per adult DKA protocol. Cautions: Monitor corrected Na; avoid rapid osmolality shifts.

Cerebral Edema during Correction

- Mannitol 0.25-1 g/kg IV over 20 min OR 3% saline 2-4 mL/kg over 10-15 min. Cautions: Use only if signs of cerebral edema; avoid if fluid overload.

11. Correct co - existing electrolyte/volume deficits

a. Replete $K^+/Ca^{2+}/Mg^{2+}$ once urine output is established.

b. If adding Na^+/K^+ to IV fluids, account for the reduced free-water delivery and recalculate rates.

STEPWISE FLUID MANAGEMENT OF HYPERNATREMIA IN PEDIATRICS PATIENTS (INCLUDING NEONATES)

Goal

Acute: Lower Na by max 1 mmol/L/h for first 6 h, then slow to ≤0.5 mmol/L/h.

Chronic: Strict limit ≤0.5 mmol/L/h and ≤10 mmol/L/day. Infants are at higher risk for cerebral edema, err on slower side if unclear onset.

1. Triage and classify

- **Confirm:** Serum Na^+ >145 mmol/L; serum osmolality high.
- **Assess:** Neurologic status, dehydration/shock, urine output, weight change.
- **Acuity:** Acute (<48 h) vs chronic/unknown (≥48 h).
- **Volume status:** Hypovolemic / Euvolemic / Hypervolemic.
- **Red flags:** Seizures, coma, shock - manage immediately.

2. Stabilize first (if shocked)

- 0.9% NaCl 10-20 mL/kg IV over 20-30 min; repeat until perfusion improves.
- Once stable, switch to correction plan below (avoid further large isotonic boluses).

3. **Decide route**

- Enteral free water (oral/NG) preferred if alert, safe swallow, no ileus.
- IV route if NPO, vomiting, ileus, severe illness.

4. **Calculate the free - water deficit (plan the target)**

- **Total body water (TBW) factor:** neonate/infant 0.70, child 0.60, adolescent 0.55 × weight (kg).
- Deficit (L) = TBW × [(Measured Na^+ / 140) − 1].
- Set a safe reduction: ≤10 mmol/L per 24 h (often 6-8 on day 1), over 48-72 h if chronic/unknown.

5. **Choose fluid and rate (then titrate)**

- **Maintenance (M):** Holliday-Segar (100/50/20 mL/kg/d) or local equivalent.
- **Replacement (R):** Deliver the calculated deficit plus ongoing losses spread across 48-72 h.
- **Typical IV choices** (tailor to labs/urine Osm and glucose needs):
  - **D5W** (pure free water) for DI or marked free-water need (use pump; avoid boluses).
  - **D5 0.45% NaCl** when some sodium is needed (common start for non-DI hypernatremia).
  - **Avoid hypotonic boluses,** all hypotonic fluids via controlled infusions only.
  - **Neonates** often need D10 for glucose support (e.g., D10 0.2-0.45% NaCl), then titrate.

  Start at Maintenance + a fraction of Rescue per hour, aiming for planned Na^+ fall (~0.25-0.5 mmol/L/h). Adjust q2-4 h.

6. **Replace ongoing losses**

- Measure vomit/diarrhea/urine; replace mL-for-mL with isotonic for volume, then adjust free water in the plan.
- Fever/sweat → increase insensible allowance.

7. **Correct electrolytes in parallel**

- Add K^+ once urine flow is established; correct hypokalemia/hypocalcemia early.
- If adding Na^+/K^+ to maintenance, remember you reduce free-water delivery - recalculate.

8. **Identify and treat the cause**

- **Hypovolemic (most common):** continue controlled D5 0.45% (or D10 in neonates) after initial isotonic resuscitation.
- **Euvolemic/Hypervolemic:** restrict sodium/fluids as appropriate; specialist input for heart/renal/liver disease.
- **Hypervolemic Hypernatremia (e.g., sodium overload)**
  - Furosemide: 1 mg/kg/dose IV/PO q6-12 h. Duration: Short-term until Na ≤145 mmol/L. Cautions: Monitor electrolytes, weight, urine output; avoid hypovolemia.
- **Hyperglycemia with Hypernatremia**
  - Insulin per pediatric DKA protocol (0.05-0.1 units/kg/h IV after fluid resuscitation). Cautions: Monitor corrected Na and glucose q1-2 h; avoid rapid drops in effective osmolality.
- **Cerebral Edema during Correction**
  - weight-based: Mannitol 0.25-1 g/kg IV over 20 min OR 3% saline 2-4 mL/kg over 10-15 min. Cautions: Immediate neuro monitoring; avoid overcorrection of Na.
- **Diabetes insipidus (polyuria, urine Osm low):**
  - Central DI: cautious DDAVP titration (0.3 µg/kg IV/SC or local pediatric dosing; start low), plus free water.
  - Nephrogenic DI: low-solute diet, thiazide ± amiloride, guided by nephrology.
  - **Avoid vaptans** and **demeclocycline** in children.

9. **Etiology work - up (child - specific)**

- Look for feeding errors, over-diluted formula, diarrhea/fever with high insensible loss.
- Consider congenital/genetic causes (e.g., central/nephrogenic DI).

- Use age-adjusted reference ranges; add targeted hormones if salt-wasting suspected.

10. Replacement strategy

- Replace ongoing losses mL-for-mL, but recalibrate free-water delivery when adding Na^+/K^+ to prevent accidental under- or over-correction.
- Correct K^+/Ca^{2+} slowly to avoid arrhythmias; ensure separate calculation from free-water deficit to prevent rapid Na drop.

11. Monitoring and safety

- Serum Na^+: q2-4 h during active correction (neonates/infants may need q1-2 h early).
- Neuro checks: age-appropriate (feeding, cry, alertness, tone, fontanelle, seizures).
- Input/output (I/O), weight, urine output hourly if severe. Check glucose (especially neonates).
- Targets: cumulative fall ≤10 mmol/L/24 h; slower if chronic.

12. If sodium is falling too fast (over - correction)

- Immediately slow/stop hypotonic infusions.
- Add sodium to IVF (e.g., step up to 0.9% NaCl) or reduce total rate.
- If neurological signs of cerebral edema, give 3% NaCl 2-4 mL/kg IV over 10-15 min, PICU monitoring.

13. If sodium not falling as planned (under - correction)

- Increase free-water fraction (shift toward D5W/D10W) or modestly increase rate.
- Recheck for ongoing unseen losses or high urine Osm limiting correction.

14. Transition and discharge

- When Na^+ approaches normal and symptoms resolve, taper to maintenance.
- Discharge after Na^+ is stable ≥24 h with a clear feeding/hydration plan.
- Educate caregivers on red flags (vomiting, lethargy, seizures), exact fluid/feeding volumes, and early follow-up Na^+ checks q2-4 days after discharge if recent adjustments or residual risk.

Note: Key Pediatric Safety Notes

- Always calculate all fluids, electrolytes, and medications per kg.
- Infants and young children have higher cerebral edema risk, avoid rapid shifts.
- Normal sodium ranges and symptom thresholds may differ in neonates.

NON-PHARMACOLOGICAL INTERVENTIONS

- Offer free water orally whenever possible; small, frequent sips to avoid discomfort.
- Calculate free-water deficit and correct with hypotonic IV fluids (D5W, 0.45% NaCl) at controlled rate per correction target.
- Stop hypertonic saline, sodium bicarbonate, or high-sodium enteral feeds.
- In DI, implement structured water-drinking schedules to match urine output.

Non - pharmacological interventions in pediatric patients

- Infants: prioritize breast milk/formula; avoid plain water in neonates unless advised.
- Older children: small, frequent sips; low-sodium ORS may help compliance.
- Strictly **weight - based** free-water deficit calculation; correction ≤0.5 mmol/L/h and ≤10 mmol/L/24h.
- Avoid high-sodium feeds or formula preparation errors.
- Monitor sodium every 2-4 h initially, plus weight, urine output, neuro status.
- In pediatric DI, caregiver-assisted structured water intake matched to urine losses.

ASSESSMENT OF RESPONSE

Domain	Adults	Children	Action if target not met
Sodium trajectory	Fall 0.25-0.5 mmol/L/h; ≤10 mmol/L/24 h (slower if chronic/unknown or ODS risk).	Same ceiling; tighter adherence. Neonates/infants: consider q1-2 h early checks.	Too fast: reduce hypotonic rate or increase Na^+ content; consider pausing. Too slow: increase free-water fraction/rate.
Lab frequency	Serum Na^+ q4-6 h during active correction; then q12-24 h when stable.	Serum Na^+ q2-4 h (neonates may need q1-2 h initially); then q12-24 h when stable.	Adjust rate/fluids; recheck within 2-4 h after any change.
Neurologic status	At each lab check: mental status, headache, seizures, focal signs.	Age-appropriate: alertness, feeding, cry, tone, fontanelle, seizures.	If worsening or new deficits: slow/stop correction; consider imaging; escalate level of care.
Volume status	Vitals, mucosa, JVP, edema/ascites; capillary refill if shock risk.	Vitals, capillary refill, extremity warmth, daily weight change.	If hypovolemic give isotonic fluid; if overloaded give diuretic/ ultrafiltration. Reassess plan.
Urine output	Target ≥0.5 mL/kg/h; track trends.	Target ≥1 mL/kg/h (age-dependent); hourly if severe.	Low output: evaluate perfusion/ AKI. High output: consider DI; adjust therapy.
Urine osmolality & sodium	If response blunted or polyuria: check Uosm/UNa to identify DI or solute diuresis.	Same; helpful in unexplained polyuria or poor Na^+ decline.	Central DI: add/titrate DDAVP. Nephrogenic DI: thiazide ± amiloride; adjust free water.
Glucose	Check with dextrose infusions; avoid hyperglycemia-driven shifts.	Check more often in neonates/ infants (hypoglycemia risk).	Treat abnormal glucose; recalculate free-water needs.
Electrolytes (K^+/ Mg^{2+}/Ca^{2+})	Replete early; adding K^+/ Na^+ reduces free-water delivery - recalculate.	Same; weight-based dosing.	Correct deficits; adjust fluid composition/rates.
I/O and weights	Strict I/O; daily weight; look for hidden losses (fever, drains).	Strict I/O; daily or more frequent weights in infants; document feeds.	Replace losses mL-for-mL; increase insensible allowance with fever/tachypnea.
Signs of over-correction (cerebral edema)	Headache, confusion, bradycardia, hypertension, seizures.	Irritability, decreased responsiveness, bulging fontanelle, seizures.	Immediate: slow/stop hypotonic fluids; consider 3% NaCl rescue; ICU/PICU review.
When Na^+ fall exceeds plan	-	-	Adults: reduce/stop hypotonic fluid; step to 0.9% NaCl. Children: same principle; lower thresholds for action. Recheck Na^+ in 2-4 h.
When Na^+ not falling	-	-	Increase free-water fraction (shift toward D5W/D10W in kids), modestly raise rate; search for ongoing losses/high Uosm.

DI on therapy	Watch for water retention after DDAVP.	Same; use pediatric DDAVP dosing.	If hyponatremia risk: space/ titrate DDAVP; adjust free water.
Readiness for step-down/discharge	Symptoms resolved; Na^+ near normal and stable ≥24 h; clear cause addressed; follow-up labs arranged.	Same + caregiver education on feeds/fluids and red flags; early outpatient Na^+ checks q2-4 days if recent changes.	Delay discharge; tighten monitoring; finalize home plan.

PROGNOSIS & PROGRESSION

Adults	Pediatrics
Properly managed **acute cases:** low risk of permanent damage, if corrected early and safely. Full recovery likely if cerebral edema and intracranial hemorrhage are avoided.	Properly managed **acute cases**: generally full recovery expected. Infants at higher risk of seizures and cerebral edema during correction.
Chronic hypernatremia can lead to persistent cognitive and motor deficits from prior brain shrinkage/ demyelination, especially in elderly or those with neurologic comorbidities	**Chronic hypernatremia** can cause lasting developmental delay, learning difficulties, and fine/gross motor deficits; risk highest in neonates/infants due to vulnerable developing brain.
Prognosis poorer when linked to severe systemic illness (sepsis, advanced renal failure) compared to isolated fluid loss.	Neonates and preterms have very narrow safety margin; Outcomes worsen with delayed diagnosis, severe dehydration, or rapid correction leading to cerebral edema.

REFERRAL CRITERIA

Refer to tertiary care when	
Adults	Pediatrics
Severe or refractory diabetes insipidus (DI).	Severe or recurrent DI, especially if etiology uncertain or requiring frequent DDAVP adjustments.
Mixed-volume disorders unclear at secondary care level.	Mixed-volume disorders where diagnosis is challenging and safe correction requires pediatric endocrine input.
Need for DDAVP titration or advanced endocrine testing.	Need for specialized pediatric endocrine testing or neurocritical monitoring.
Complex comorbidities requiring multidisciplinary care.	Persistent/worsening neurologic symptoms (seizures, reduced consciousness) despite initial stabilization.
	Neonates/infants with suspected hypothalamic-pituitary disorders, genetic DI, or inborn errors of metabolism.

Complications

Complications common to both Adults & Pediatrics are:

- Cerebral hemorrhage rom rapid brain cell shrinkage during onset of hypernatremia
- Seizures, coma in severe, untreated cases.
- Cerebral edema if sodium is lowered too quickly during treatment. Note: Infants and young children have higher cerebral edema risk, avoid rapid shifts.
- Normal sodium ranges and symptom thresholds may differ in neonates.

PATIENT/CAREGIVER EDUCATION

Objectives

- Spot symptoms early: intense thirst, dry mouth, confusion, lethargy, reduced intake, low urine.
- Follow the fluid plan: stick to prescribed volumes, timing, and type (oral/NG/IV).
- Keep monitoring appointments: sodium checks and reviews prevent dangerous shifts.
- Know red flags: in case of seizures, sudden confusion, fainting, breathing trouble seek emergency care.

Adults

Do	Don't
Hydration as prescribed: meet but don't exceed your daily target.	Don't binge water or sports/"zero-calorie" drinks outside the plan.
Track intake/output: use a marked bottle and a simple log.	Don't self-start or change diuretics, laxatives, or salt tablets.
Daily weight: report ≥1 kg change in 24 hours.	Don't skip or double doses of prescribed medicines.
Report symptoms early: extreme thirst, confusion, weakness, drop in urine.	Don't miss sodium checks - dosage and fluids may need adjustment.
Take meds exactly as directed: DDAVP/diuretics/others per schedule.	
Attend all labs/visits: bring your logs.	

Children (Caregiver guidance)

Do	Don't
Follow the weight-based plan: fluids/feeds exactly as prescribed.	Don't dilute formula or give free water to infants unless told to.
Feed on schedule: breast milk or correctly prepared formula; measure volumes.	Don't restrict fluids so tightly that calories drop - ask for a feeding plan.
Watch early signs: irritability, excessive crying, lethargy, poor feeding, vomiting, ↓ urine, fever.	Don't allow unsupervised access to water (toddlers/young children), especially in hot weather or after sports.
Log daily: feeds, fluids, diapers/urine, weight (as advised).	Don't use over-the-counter diuretics, herbal mixes, or salt supplements without medical advice.
Give meds precisely: use oral syringes for small volumes; confirm DDAVP timing if prescribed.	
Keep all labs/visits: sodium checks every 2-4 days initially if directed.	

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