Hyponatremia

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Synonyms and related keywords: low sodium

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Background: Serum sodium concentration and serum osmolarity normally are maintained under precise control by homeostatic mechanisms involving thirst, antidiuretic hormone (ADH), and renal handling of filtered sodium. Clinically significant hyponatremia is

relatively uncommon and is nonspecific in its presentation; therefore, the ED physician must maintain an appropriate index of suspicion for this disorder. Irreparable harm can befall the patient when abnormal serum sodium levels are corrected too quickly or too slowly. The ED physician must have a thorough understanding of the pathophysiology of hyponatremia to initiate safe and effective corrective therapy.

Hypovolemic hyponatremia

Total body water (TBW) decreases; total body sodium (Na^+) decreases to a greater extent. The extracellular fluid (ECF) volume is decreased.

Euvolemic hyponatremia

TBW increases while total sodium remains normal. The ECF volume is increased minimally to moderately, but edema is not present.

Hypervolemic hyponatremia

Total body sodium increases, and TBW increases to a greater extent. The ECF is increased markedly, and edema is present.

Redistributive hyponatremia

Water shifts from the intracellular to the extracellular compartment, with a resultant dilution of sodium. The TBW and total body sodium are unchanged. This condition occurs with hyperglycemia.

Pseudohyponatremia

The aqueous phase is diluted by excessive proteins or lipids. The TBW and total body sodium are unchanged. This condition is seen with hypertriglyceridemia and multiple myeloma.

Pathophysiology: Serum sodium is regulated by thirst, ADH, the renin-angiotensin-aldosterone system, and variations in renal handling of filtered sodium. Increases in serum osmolarity above the normal range (280-300 mOsm/kg) stimulate hypothalamic osmoreceptors, which, in turn, cause an increase in thirst and in circulating levels of ADH.

ADH increases free water reabsorption from the urine, yielding low urine volumes of relatively high osmolarity and returning serum osmolarity toward normal. ADH also is secreted in response to hypovolemia, pain, fear, nausea, and hypoxia.

Aldosterone, synthesized by the adrenal cortex, is regulated primarily by serum potassium but also is released in response to hypovolemia through the renin-angiotensin-aldosterone axis.

Aldosterone causes absorption of sodium at the distal renal tubule. Sodium retention obligates free water retention, helping to correct the hypovolemic state.

The healthy kidney regulates sodium balance independently of ADH or aldosterone by varying the degree of sodium absorption at the distal tubule. Hypovolemic states, such as hemorrhage or dehydration, prompt increases in sodium absorption in the proximal tubule. Increases in vascular volume suppress tubular sodium reabsorption, resulting in natriuresis and helping to restore normal vascular volume.

Generally, disorders of sodium balance can be traced to a disturbance in thirst or water acquisition, ADH, aldosterone, or renal sodium transport.

Hyponatremia is physiologically significant when it indicates a state of extracellular hypo-osmolarity and a tendency for free water to shift from the vascular space to the intracellular space. Although cellular edema is well tolerated by most tissues, it is not well tolerated within the rigid confines of the bony calvarium. Therefore, clinical manifestations of hyponatremia are related primarily to cerebral edema.

The rate of development of hyponatremia plays a critical role in its pathophysiology. When serum sodium falls slowly, over a period of several days or weeks, the brain is capable of compensating by extrusion of solutes and fluid to the extracellular space. Compensatory extrusion of solutes reduces the flow of free water into the intracellular space, and symptoms are much milder for a given degree of hyponatremia.

When serum sodium falls rapidly, over a period of 24-48 hours, this compensatory mechanism is overwhelmed and severe cerebral edema may ensue, resulting in brainstem herniation and death.

Frequency:

- **In the US:** Hyponatremia is the most common electrolyte disorder, with an incidence of approximately 1% of hospitalized patients.

Mortality/Morbidity: Pathophysiologic differences between patients with acute and chronic hyponatremia engender important differences in their morbidity and mortality.

- Patients with acute hyponatremia (developing over 48 hours or less) are subject to more severe degrees of cerebral edema for a given level of serum sodium. The primary cause of morbidity and death is brainstem herniation and mechanical compression of vital midbrain structures. Rapid identification and correction of serum sodium is necessary in patients with severe acute hyponatremia to avert brainstem

herniation and death.

- Patients with chronic hyponatremia (developing over more than 48 hours) experience milder degrees of cerebral edema for a given level of serum sodium. Brainstem herniation has not been observed in this group of patients. The principal causes of morbidity and death are status epilepticus (when chronic hyponatremia reaches levels of 110 mEq/L or less) and cerebral pontine myelinolysis (an unusual demyelination syndrome that occurs when chronic hyponatremia is corrected too quickly).

- The distinction between acute and chronic hyponatremia has critical implications in terms of morbidity and mortality and in terms of proper corrective therapy.

Sex: Incidence is equal in males and females.

Age: Hyponatremia is most common in the very young and in the very old; these groups are less able to experience and express thirst and less able to regulate fluid intake autonomously. Specific high-risk groups include the following:

- Infants fed tap water in an effort to treat symptoms of gastroenteritis

- Elderly patients with diminished sense of thirst, especially when physical infirmity limits independent access to food and drink

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History:

- The number and severity of symptoms increase with the degree of hyponatremia and the rapidity with which it develops. When serum sodium falls gradually, over a period of several days or weeks, sodium levels as low as 110 mEq/L may be reached with minimal symptomatology. In contrast, a fall in serum sodium over 24-48 hours may overwhelm compensatory mechanisms, leading to severe cerebral edema, coma, or brainstem herniation.

- Symptoms may be limited to mild anorexia, headache, or muscle cramps, or the patient may present with obtundation, coma, or status epilepticus.

- Hyponatremia often is seen in association with pulmonary disease or disorders of the CNS. The ED physician should have an increased index of suspicion in patients with

pneumonia, active tuberculosis, pulmonary abscess, neoplasm, or asthma, or in those patients with CNS infection, trauma, or neoplasm.

- Hyponatremia is associated with numerous medications, including amiodarone, chlorpropamide, clofibrate, carbamazepine, oxcarbazepine, cyclophosphamide, tolbutamide, opiates, thiazides, oxytocin, desmopressin, selective serotonin reuptake inhibitors, trazodone, and vincristine. The patient's medication list should be examined for drugs known to cause hyponatremia.
- Hyponatremia has been noted in patients with poor dietary intake who consume large amounts of beer (called beer potomania) and after use of the recreational drug *N*-methyl-3,4-methylenedioxyamphetamine (ie, MDMA or ecstasy).
- A history of hypothyroidism or adrenal insufficiency should be sought because each is associated with hypo-osmolar hyponatremia.
- Patients with clinically significant hyponatremia present with nonspecific symptoms attributable to cerebral edema. These symptoms, especially when coupled with a recent history of altered fluid balance, should suggest the possibility of hyponatremia.
    - Anorexia
    - Nausea and vomiting
    - Difficulty concentrating
    - Confusion
    - Lethargy
    - Agitation
    - Headache
    - Seizures

Physical: Physical findings are highly variable and dependent on the degree and the chronicity of hyponatremia. Patients with acutely developing hyponatremia are symptomatic at a level of 120 mEq/L. Those patients with chronic hyponatremia tolerate much lower levels.

- Most abnormal findings on physical exam are neurologic in origin.

- Level of alertness ranging from normal to agitation to coma
- Variable degrees of cognitive impairment (eg, difficulty with short-term recall; loss of orientation to person, place, or time; frank confusion or depression)
- Focal or generalized seizure activity
- In those patients with acute severe hyponatremia, signs of brainstem herniation, including coma; fixed, unilateral, dilated pupil; decorticate or decerebrate posturing; and respiratory arrest

- In addition to neurologic findings, patients may exhibit signs of hypovolemia or hypervolemia. Determining the hydration status of the patient may help establish the etiology of the hyponatremia and suggest the best treatment course.
  - Dry mucous membranes, tachycardia, diminished skin turgor, and orthostasis suggest hypovolemic hyponatremia due to excessive loss of body fluids and replacement with inappropriately dilute fluids.
  - Pulmonary rales, S3 gallop, peripheral edema, or ascites suggest hypervolemic hyponatremia due to excess retention of sodium and free water (ie, cirrhosis, nephrotic syndrome, congestive heart failure).
  - Patients who lack findings of hypovolemia or hypervolemia are considered to have euvolemic hyponatremia, which is consistent with such etiologies as exogenous free water load, hypothyroidism, cortisol deficiency, or syndrome of inappropriate antidiuretic hormone (SIADH).
- Other nonspecific signs include muscle weakness and cramping. Rhabdomyolysis is an occasional consequence of hyponatremia and should be considered in patients with muscle pain or tenderness.

Causes:

- Hypovolemic hyponatremia develops as sodium and free water are lost and replaced by inappropriately hypotonic fluids, such as tap water, half-normal saline, or dextrose in water. Sodium can be lost through renal or nonrenal routes. Nonrenal routes include GI losses, excessive sweating, or third spacing of fluids (eg, peritonitis, pancreatitis, burns).
  - Excess fluid losses (eg, vomiting, diarrhea, excessive sweating, GI fistulas or drainage tubes, pancreatitis, burns) that have been replaced primarily by hypotonic

fluids

- Acute or chronic renal insufficiency in which patient may be unable to excrete adequate amounts of free water
- Salt-wasting nephropathy
- Prolonged exercise in a hot environment, especially in patients who hydrate aggressively with hyposmolar fluids during exertion. Severe symptomatic hyponatremia has been reported in marathon runners and in recreational hikers in the Grand Canyon.

- Euvolemic hyponatremia implies normal sodium stores and a total body excess of free water. This occurs in patients who take in excess fluids.
  - Psychogenic polydipsia, often in psychiatric patients
  - Administration of hypotonic intravenous (IV) or irrigation fluids in the immediate postoperative period
  - Infants who may have been given inappropriate amounts of free water
- Hypervolemic hyponatremia occurs when sodium stores increase inappropriately. This may result from renal causes, such as acute or chronic renal failure, when dysfunctional kidneys are unable to excrete the ingested sodium load. It also may occur in response to states of decreased effective intravascular volume.
  - History of hepatic cirrhosis, congestive heart failure, or nephrotic syndrome, in which patients are subject to insidious increases in total body sodium and free water stores
  - Uncorrected hypothyroidism or cortisol deficiency
  - SIADH
  - Consumption of large quantities of beer or use of the recreational drug MDMA (ecstasy)
- Hyponatremia can be caused by many medications. Known offenders include acetazolamide, amiloride, amphotericin, atovaquone, thiazide diuretics, amiodarone, basiliximab, angiotensin II receptor blockers, angiotensin-converting enzyme inhibitors, carbamazepine, carboplatin, carvedilol, celecoxib, cyclophosphamide, clofibrate, desmopressin, donepezil, eplerenone, gabapentin, haloperidol, indomethacin, ketorolac, loop diuretics, mitoxantrone,

nimodipine, oxcarbazepine, opiates, oxytocin, pimozide, propafenone, proton pump inhibitors, sirolimus, ticlopidine, vincristine, selective serotonin reuptake inhibitors, sulfonylureas, trazodone, tolbutamide, zalcitabine, and zonisamide.

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Adrenal Insufficiency and Adrenal Crisis
Congestive Heart Failure and Pulmonary Edema
Gastroenteritis
Hypothyroidism and Myxedema Coma
Renal Failure, Acute
Renal Failure, Chronic and Dialysis Complications
Syndrome of Inappropriate Antidiuretic Hormone Secretion

Other Problems to be Considered:

Cirrhosis
Nephrotic syndrome
Psychogenic polydipsia
Pseudohyponatremia
Iatrogenic
Medication effects

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Lab Studies:

- The diagnosis of hyponatremia depends entirely upon the ability to properly obtain a sample of the patient's serum and to accurately measure its concentration of sodium.
- When interpreting serum sodium levels, always consider the possibility of sampling error, especially when the reported value does not seem consistent with the history or physical findings.
  - Was the patient's blood sample properly labeled?
  - Was it obtained from a venous site proximal to an infusion of hypotonic saline or dextrose in water?
  - Is laboratory measurement or reporting in error?
  - If an error is suspected, a second sample should be submitted for testing before therapeutic measures are initiated.

- In addition to sampling and analysis errors, several physiologic states exist in which correct laboratory analysis yields low serum sodium levels, but these levels do not reflect a true hypo-osmolar state.
    - The most common example is serum hyperglycemia.
        - Accumulation of extracellular glucose induces shift of free water from the intracellular space to the extracellular space.
        - Serum sodium is diluted by a factor of 1.6 mEq/L for each 100 mg/dL increase in serum glucose.
        - Systemic osmolarity is normal or even increased, not decreased as in true (ie, hypo-osmolar) hyponatremia.
        - This hypertonic hyponatremia has no physiologic significance, and serum sodium corrects as normoglycemia is reestablished.
    - A similar phenomenon is observed in patients treated with glycerol or mannitol in an effort to control acute glaucoma or intracranial hypertension.
    - Hyponatremia may be noted in patients whose serum contains unusually large quantities of protein or lipid.
        - In these patients, an expanded plasma protein or lipid fraction leads to a decrease in the plasma water fraction in which sodium is dissolved.
        - Laboratory techniques that measure absolute sodium content per unit of plasma water report low sodium levels despite the fact that the concentration of sodium in serum water remains within the normal range.
        - This phenomenon, known as pseudohyponatremia, occurs when flame emission spectrophotometry or indirect potentiometry is used to assay serum sodium levels rather than direct potentiometry techniques. This occurs in approximately 60% of US laboratories.
        - Serum osmolarity remains undisturbed, and attempts at correcting serum sodium are not indicated.
        - Hyperlipidemia that is severe enough to produce pseudohyponatremia almost always is accompanied by a lipemic appearance of the serum sample.
        - Hyperproteinemia of sufficient magnitude to induce pseudohyponatremia commonly is due to coexisting multiple myeloma.
- Serum osmolarity is helpful in establishing the diagnosis of true hypo-osmolar hyponatremia. Serum osmolarity is abnormally low in patients with hypo-osmolar hyponatremia, but it is normal in patients with pseudohyponatremia due to hyperlipidemia or hyperproteinemia and normal or elevated in patients with hypertonic hyponatremia due to serum hyperglycemia.

- Urine sodium levels are helpful in distinguishing renal causes of hyponatremia from nonrenal causes.
    - Patients with hypovolemic hyponatremia due to nonrenal causes (eg, vomiting, diarrhea, fistulas, GI drainage, third spacing of fluids) have avid renal absorption of tubular sodium and urine sodium levels of less than 20 mEq/L, whereas those with hypovolemic hyponatremia due to renal causes (eg, diuretics, salt-losing nephropathy, aldosterone deficiency) have inappropriately elevated urine sodium levels in excess of 20 mEq/L.
    - Patients with hypervolemic hyponatremia due to decreases in effective circulating volume (eg, cirrhosis, nephrosis, congestive heart failure) have urine sodium levels of less than 20 mEq/L, whereas those with renal causes of hypervolemic hyponatremia or with SIADH have urine sodium levels in excess of 20 mEq/L.
- Urine osmolarity may be helpful in establishing the diagnosis of SIADH.
    - Typically, patients with SIADH have inappropriately concentrated urine with urine osmolarities in excess of 100 mOsm/L.
    - Patients with other forms of hyponatremia and appropriately depressed levels of ADH have urine osmolarities below 100 mOsm/L.
- Serum thyroid-stimulating hormone (TSH) and free thyroxine should be checked if the clinical presentation is consistent with hypothyroidism.
- Adrenal function should be assessed, via random serum cortisol levels or adrenocorticotropic hormone (ACTH) stimulation test, in patients who recently have taken oral steroids or in any patient suspected of having cortisol deficiency.

Imaging Studies:

- Imaging studies may be indicated depending upon the underlying etiology of the hyponatremia (eg, chest radiograph in a patient with CHF).
- Usually, a head CT scan is indicated in the patient with altered mental status to ensure that no other underlying cause for the mental status is present.

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Prehospital Care: Hyponatremia is necessarily a hospital-based diagnosis, but patients may exhibit signs of severe neurologic dysfunction during prehospital evaluation and transport.

- Address acute life-threatening conditions and initiate supportive care.
- Establish reliable IV access and give supplemental oxygen to patients with lethargy or obtundation. In these patients, evaluate the possibility of hypoglycemia with a rapid glucose check; administer IV glucose to hypoglycemic patients.

- Administer standard prehospital anticonvulsant therapy to patients experiencing seizures related to hyponatremia; they probably will respond poorly to this therapy, but it should be administered until a more definitive diagnosis and therapy are available.

- Intubate and initiate hyperventilation to reduce intracranial pressure in patients exhibiting signs of brainstem herniation (eg, obtundation; fixed, unilateral, dilated pupil; decerebrate or decorticate posturing) until a more definitive therapy can be initiated.

- Avoid giving hypotonic IV fluids, because they may exacerbate cerebral edema.

Emergency Department Care: The ED evaluation of patients with hyponatremia has 2 goals: to determine the chronicity of the hyponatremic state and to determine the cause. Careful consideration of chronicity and cause allow rational selection of ED therapy.

- Acute hyponatremia is less common than chronic hyponatremia and typically is seen in patients with a history of sudden free water loading (eg, patients with psychogenic polydipsia, infants fed tap water for 1-2 days, patients given hypotonic fluids in the postoperative period).
    - Acute evolution of hyponatremia leaves little opportunity for compensatory extrusion of CNS intracellular solutes.
    - The ultimate danger for these patients is brainstem herniation when sodium levels fall below 120 mEq/L.
    - The therapeutic goal is to increase serum sodium rapidly by 4-6 mEq/L over the first 1-2 hours.
    - First, the source of free water must be identified and eliminated.
    - In patients with healthy renal function and mild to moderately severe symptoms, serum sodium may correct spontaneously without further intervention.
    - Patients with seizures, severe confusion, coma, or signs of brainstem herniation should receive hypertonic (3%) saline to rapidly correct serum sodium toward normal, but only enough to arrest the progression of symptoms.
    - An increase in serum sodium of 4-6 mEq/L is generally sufficient.
- Chronic hyponatremia is more common than acute hyponatremia.
    - Patients with mild symptoms and serum sodium of 125 mEq/L or less often have chronic hyponatremia.
    - These patients lack any history of sudden free water loading.
- Chronic hyponatremia must be managed with extreme care.

Appropriate treatment of hyponatremia depends upon the correct classification of hyponatremia, the concomitant disease state, the severity of symptoms, and the sev of hyponatremia.

Drug Category: *Electrolyte supplements* -- Hypertonic saline may be used to rap increase serum sodium in patients with severe acute or chronic hyponatremia, as manifested by severe confusion, coma, seizures, or evidence of brainstem herniation.

Drug Name	Hypertonic (3%) saline -- Contains 513 mEq/L of NaCl. Volume of hypertonic saline administered depends upon current and desired serum sodium levels and patient's weight. In general, increase of 4-6 mEq/L in serum sodium is sufficient to arrest progression of symptoms in severe hyponatremia. Further rapid increase in serum sodium not indicated.
Adult Dose	Required volume = (desired change in serum sodium) (TBW) / (Na in IV fluid - current serum Na), where TBW = body weight x 0.6 For example, a 60-kg woman with serum sodium of 113 mEq/L would require 360 mL of hypertonic saline In general, 300-500 mL of 3% NaCl is reasonable dose in most adult patients with severe symptomatic hyponatremia Give IV over first 1-2 h until resolution of seizures or herniation
Pediatric Dose	Administer as in adults
Contraindications	Hypernatremia; fluid retention; hypertonic uterus
Interactions	May decrease lithium levels
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Caution in congestive heart failure, hypertension, edema, renal insufficiency, liver cirrhosis, and sodium toxicity

FOLLOW-UP

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Further Inpatient Care:

- Admit patients with severely symptomatic hyponatremia manifested by coma, recurrent seizures, or evidence of brainstem dysfunction to an ICU and monitor serum sodium levels closely.
- Admit patients with a propensity toward inappropriate free water ingestion to a unit where free water access is restricted. Clozapine appears to be effective in the long-term treatment of schizophrenic patients with compulsive water drinking.
- Discontinue medications known to be associated with hyponatremia. Thiazide diuretics are a well-known cause of profound hyponatremia, especially in elderly patients, and should be discontinued in all admitted patients.

Complications:

- Complications related to hyponatremia include rhabdomyolysis, seizures, permanent neurologic sequelae related to ongoing seizures or cerebral edema, respiratory arrest, and death.
- Complications related to therapy of hyponatremia include fluid overload and CPM.

Prognosis:

- Prognosis is dependent on the underlying condition.

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Medical/Legal Pitfalls:

- Failure to consider the possibility of sampling or analysis error, hyperglycemia, hyperproteinemia, or hyperlipidemia before making the diagnosis of hypovolemic hyponatremia
- Failure to recognize high-risk groups, including elderly patients, patients on diuretics, infants, postoperative patients, and patients with malignancy
- Failure to quickly recognize severe acute hyponatremia and to correct it promptly, allowing ongoing risk of brainstem herniation.
- Correcting serum sodium too rapidly in patients with chronic hyponatremia (in excess of 0.5 mEq/L/h or 12 mEq/L/d), thereby incurring the risk of CPM

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Hyponatremia excerpt