Managing Sodium Disorders

Adesola Odunayo, DVM, MS, DACVECC
University of Tennessee

You have asked…

What is the current best practice in diagnosing and managing hyper- and hyponatremia in dogs and cats?

The expert says…

Sodium, the most abundant extracellular cation, is the primary determinant of serum osmolality. In general, as serum sodium increases, osmolality increases; the converse is also true. Measured serum sodium concentration is a reflection of sodium amount relative to volume of water in the body.

Serum osmolality can be calculated as follows:

$$2Na\ (mEq/L) + 2K\ (mEq/L) + \frac{glucose\ (mg/dL)}{18} + \frac{BUN\ (mg/dL)}{2.8}$$

that is,

$$2(Na + K) + \frac{BUN}{2.8} + \frac{glucose}{18}$$

Sodium disorders are relatively common in dogs and cats, particularly in critically ill patients, and can lead to significant neurologic sequelae, especially when not identified and managed quickly. Serum electrolyte concentrations, including sodium concentration, should be routinely measured in all ill and hospitalized patients, especially during continuous administration of IV fluids.

Hypernatremia

Hypernatremia is an electrolyte disturbance demonstrated by serum sodium concentration of greater than 160 mEq/L, although reference ranges may vary slightly among laboratories. Hypernatremia is caused by excess sodium intake or retention, pure water loss, or hypotonic water loss (**Table 1**, next page). Because sodium is the major contributor to osmolality, hypernatremia is associated with hyperosmolality.

Clinical Signs

Signs of hypernatremia are typically related to the CNS and may be nonspecific, including lethargy, weakness, behavior changes, ataxia, seizures, stupor, and coma. They become evident when plasma sodium concentration is greater than 170 to 175 mEq/L,[1] which is also associated with increased serum osmolality. Hypernatremia and hyperosmolality can cause fluid to shift from the intracellular to extracellular space, resulting in decreased cerebral cellular volume, which can ultimately lead to vascular

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rupture with cerebral bleeding, subarachnoid hemorrhage, permanent neurologic damage, and death.

If hypernatremia develops acutely, clinical signs can appear rapidly and be present at lower sodium concentrations. When hypernatremia evolves over a longer period, clinical presentation and sequelae can be less severe because the neurons have had time to reestablish intracellular volume through accumulation of idiogenic osmoles (eg, amino acids, carbohydrates) in the brain, providing a diffusion gradient that helps keep water in the intracellular space. When chronic hypernatremia is corrected rapidly, the idiogenic osmoles draw water into the intracellular space, leading to cerebral edema.[2]

Table 1 Causes of Primary Hypernatremia[1]

Sign	*Differentials*
Hypotonic fluid loss	Chronic renal failure Diarrhea Pancreatitis Peritonitis Small intestinal obstruction Vomiting
Pure water deficit	Central diabetes insipidus Fever Heatstroke Inadequate access to water Nephrogenic diabetes insipidus Primary hypodipsia
Sodium gain	Hypertonic fluid administration Primary hyperaldosteronism Salt poisoning Sodium bicarbonate infusion

Management

Management of hypernatremia can be challenging; the underlying cause must be identified and the condition treated while managing hydration status, with attention to the rate of sodium correction.

In patients that develop hypernatremia rapidly, sodium concentration can be corrected rapidly without increasing risk for cerebral edema. Idiogenic osmoles generally take approximately 24 hours to develop. If the hypernatremia is more chronic (>24h) or of unknown duration, sodium concentration should be corrected at a slower rate.

If a patient is in hypovolemic shock or has significant hypovolemia (identified by tachycardia, prolonged CRT, poor pulses, and/or arterial hypotension), 10 to 20 mL/kg of 0.9% saline should be administered IV or IO rapidly over 15 to 20 minutes to replenish intravascular volume. This may be repeated if signs of hypovolemia do not resolve.[3]

Once the intravascular fluid deficit has been replaced, serum sodium concentration should be reevaluated and water deficits corrected if hypernatremia is still present. To prevent rapid fluid shifts into the brain and cerebral edema, the serum sodium concentration should be gradually reduced at a rate no greater than 0.5 mEq/L/hr and no more than 10 to 12 mEq/L/day.

Although hypernatremia caused by pure sodium gain is rare, affected patients may be managed via loop diuretic.

Fluid Choice

IV fluids should be administered to correct hypernatremia in dogs and cats: hypotonic fluids are recommended. Usually the more hypotonic the fluid, the lower the infusion rate required (**Table 2**).[2] Normal serum osmolality ranges from 290 to 310 mOsm/kg in dogs and 308 to 335 mOsm/kg in cats.

Fluid Volume

Calculations to estimate the volume of hypotonic fluid to administer are rough estimations, as dynamic fluid shift can occur. Serum electrolytes should be monitored q2–4h to ensure that sodium concentration does not change too rapidly. The author's preferred formula provides an estimate of the effect

Table 2 Fluid Types & Sodium Concentrations

Fluid	*Sodium (mEq/L)*	*Osmolality (mOsm/kg)*
Lactated Ringer's solution	130	273
0.9% saline	154	308
Normosol R (hospira.com)	140	295
Plasmalyte A (baxter.com)	140	294
5% dextrose in water	0	252
0.45% saline	77	154
0.45% saline + 2.5% dextrose	77	280
3% saline	513	1026

that 1L of any infusate has on serum sodium concentration; total body water is calculated as body weight in kg × 0.6:

$$\text{Change in serum } Na^+ = \frac{\text{infusate Na} - \text{Serum Na}}{\text{total body water} + 1}$$

Complications of Therapy

Rapid correction of hypernatremia may lead to cerebral edema, which can be identified by obtundation, seizures, or rapid deterioration in neurologic status after fluid therapy has been initiated. If cerebral edema is suspected, rate of fluid infusion should be reduced or discontinued. Mannitol may reduce cerebral interstitial edema. Most important, serum electrolytes should be monitored frequently and adjustments made to the type and rate of fluid administered.

Hyponatremia

Hyponatremia is an electrolyte disturbance demonstrated by serum sodium concentration of less than 140 mEq/L, although reference ranges may vary slightly among laboratories. Causes include sodium loss or increased water conservation. Hyponatremia may also occur if there is an increase in osmotically active solutes (eg, glucose, mannitol). Estimation of plasma osmolality may help determine the cause of hyponatremia, which is usually associated with low osmolality (**Table 3**). Hyponatremia with normal osmolality is usually associated with pseudohyponatremia, whereas hyponatremia with high osmolality is typically caused by an increase in osmotically active solutes. Pseudohyponatremia may occur in the presence of hyperlipidemia or hyperproteinemia, usually because of the specific laboratory methodology used, falsely lowering the measured serum sodium concentration.

Clinical Signs

Signs associated with hyponatremia include lethargy, anorexia, weakness, altered mentation, obtundation, and seizures, which can ultimately lead to death; neurologic signs typically predominate. A decrease in plasma osmolality can cause fluid to move

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Table 3 Causes of Hyponatremia[1]

Sign	*Differentials*
Low plasma osmolality	Advanced liver failure Advanced renal failure Congestive heart failure Cutaneous burns Diuretic administration GI fluid loss Hypoadrenocorticism Hypotonic fluid administration Nephrotic syndrome Pancreatitis Peritoneal effusion
Normal plasma osmolality	Hyperlipidemia Hyperproteinemia
High plasma osmolality	Hyperglycemia Mannitol infusion

The rate of correction should not exceed 0.5 mEq/L/hr with chronic changes in serum sodium concentration.

from the extracellular to intracellular space, causing cerebral edema and cell lysis. Similar to those for hypernatremia, clinical signs of hyponatremia are related to rapidity of onset more than to severity of associated plasma hypoosmolality.

Management

The goals of managing hyponatremia are to diagnose and correct the underlying cause and, if necessary, increase the serum sodium concentration.

Severe symptomatic acute hyponatremia is uncommon. Isotonic IV crystalloid solutions (eg, 0.9% saline, Plasmalyte A, lactated Ringer's solution) are recommended to correct serum hypoosmolality. In cases of refractory seizures from hypotonic hyponatremia, 3% saline may be considered. Treatment should address signs rather than serum sodium concentration. The rate of correction should not exceed 0.5 mEq/L/hr with chronic changes in serum sodium concentration.

Patients with chronic hyponatremia usually have few or no clinical signs attributable to hypoosmolality, likely because the brain has had time to compensate for decreased plasma osmolality. Treatment of chronic hyponatremia may be more dangerous than the disorder; asymptomatic patients are usually treated with mild water restriction and monitoring of serum sodium concentration. Symptomatic patients should be treated conservatively with crystalloid solutions at correction rates that do not exceed 0.5 mEq/L/hr IV.

Conclusion

Sodium disorders are potentially life-threatening and may be challenging to manage. While the formulas presented here can help estimate the volume of fluid required to correct sodium disorders, it is important to monitor sodium absorption q2–4h to avoid rapid changes in serum sodium concentration. ■ cb

See **Aids & Resources**, back page, for references & suggested reading.

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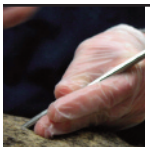
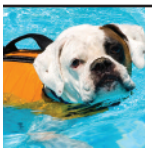
Lane completed an internship at Texas A&M University and earned her MS and internal medicine certification at Colorado State University, her doctorate of education from University of Tennessee, and her DVM from University of Georgia. DIAGNOSTIC TREE PAGE 18



LISA POWELL, DVM, DACVECC, is full clinical professor at University of Minnesota Veterinary Medical Center. Her professional interests include sepsis, mechanical ventilation, trauma, and toxins. Dr. Powell completed an internship at The Animal Medical Center and an emergency and critical care residency at Tufts University. She earned her DVM from Texas A&M University. PROCEDURES PRO PAGE 61

MARGARET E. GRUEN, DVM, MVPH, DACVB, is a veterinary behaviorist at North Carolina State University. Her interests include gerontology, feline pain behavior, and canine and feline behavior. She has written and been published on medically treating canine anxiety disorders and developing outcome measures for degenerative joint disease-associated pain assessment in cats. She frequently contributes to the Veterinary Behavior Symposium held in conjunction with the AVMA convention. Dr. Gruen earned her DVM from University of Illinois and her MVPH from NCSU. HOW I TREAT PAGE 13



SUZANNE STARR, DVM, CCRP, practices at and owns Paws in Motion Veterinary Rehabilitation Center in Natick, Massachusetts. Her areas of interest include senior and postoperative patient care. Dr. Starr completed an internship at Angell Animal Medical Center and earned her DVM from Tufts University. TOP 5 PAGE 31 ■ cb