Hypernatremia

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LEARNING OBJECTIVES

1. Compare and contrast the pathophysiological mechanisms of hypernatremia.
2. Explain adaptions that occur as a result of elevated extracellular fluid tonicity.
3. Describe the pathophysiological outcome of high intracellular osmolyte concentration.
4. List considerations for correction of hypernatremia

ABSTRACT

Hypernatremia is biochemically defined by a blood sodium concentration ([Na^+]) above the normal reference range for the laboratory performing the test (typically >145 mmol/L). The clinical relevance of an above normal blood [Na^+] is largely determined by the severity of the clinical signs and symptoms associated with cellular shrinkage (crenation). High blood sodium concentrations are largely caused by: 1) excessive water loss with inadequate fluid replacement (thirsting); 2) excessive salt ingestion; or a likely combination of too little fluid with too much salt. Morbidity and mortality from hypernatremia has been documented in infants accidentally poisoned with salt or having difficulties breastfeeding, children ingesting excessive amounts of salt as an emetic or punishment, mentally or physically disabled individuals (often living in nursing homes) who cannot express thirst or have free access to fluids, athletes who refrain from drinking during heavy exercise in hot conditions, and hospitalized patients with under-replaced fluid or over-replaced sodium administration. Poor clinical outcomes and delayed recovery have been documented in hospitalized patients with hypernatremia, compared with patients who are admitted and remain normonatremic throughout their hospital stay. Clinically significant hypernatremia in free living humans is extremely rare, with "salt poisoning" often an indicator of abuse, neglect, or mental illness. Thus, the secret stories of hypernatremia often whisper tales of suicide from soy sauce, death by exorcism and salting rituals, extreme parental punishment, hunger strikes, getting lost in the sea or desert, and mass accidental poisonings whereas salt is mistaken for sugar.

ABBREVIATIONS: [Na^+] – sodium concentration, ICP – intracranial pressure, ICU – intensive care unit, TBI - traumatic brain injury

INDEX TERMS: Salt poisoning, dehydration, dysnatremia

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Definition and Diagnosis

Hypernatremia (hyper = high; natremia = sodium in the blood) is a biochemical diagnosis in which the sodium content (in millimoles) in relationship to plasma water (in liters) is above the range of normal for the laboratory performing the test. For most laboratories, hypernatremia represents any blood [Na^+] above 145 mmol/L. Both the magnitude and rapidity of increase in blood [Na^+] influence the severity of clinical signs, symptoms and prognostic outcomes from hypernatremia. Generally speaking, the higher the initial blood [Na^+] value, the more dire the clinical consequences, with the survival rates of infants (<1 year) greater than that of adults.[1] In one review of 11 adults (>13years) and 20 children developing salt poisoning, only 1/11 adults (9%) with a blood [Na^+] above 160 mmol/L survived while 4/9 children (44%) and 9/11

(82%) infants survived.[1] The highest recorded blood sodium concentration in a surviving infant appears to be 274 mmol/L.[2]

The development of hypernatremia may be acute (develops in less than 24 hours) or chronic (present for more than one day).[3] Acute hypernatremia generally occurs when sodium intake is excessive over a short period of time, as demonstrated in both accidental[2,4-6] and non-accidental [7,8] cases of "salt poisoning."[9] Chronic hypernatremia, by definition, develops more slowly and more often results from either water loss and/or water lack commonly seen in: newborns with breastfeeding difficulties,[10,11] children with diarrheal disease,[12] nursing home patients with mental or physical disabilities[9,13-17]or individuals participating in hunger strikes.[18] The brain adapts to chronic hypernatremia via active intracellular reuptake of sodium, potassium, and organic osmolytes (glutamine, glutamate, taurine, and myo-inositol), which serve to maintain cellular size (i.e. limit brain shrinkage), thereby reducing the severity of neurological symptoms.[3]

Hypernatremia triggers intense thirst in healthy humans,[19] infants,[1,2,20] and in individuals with Down Syndrome[1] to protect against cellular dehydration. Nausea, vomiting, and falls and weakness are common symptoms of severe hypernatremia in patients presenting to an emergency department.[21] Other neurological symptoms associated with hypernatremia include lethargy, weakness, irritability, drowsiness, hyperreflexia, involuntary muscle twitching, spasticity, seizures, and coma.[6,11,22] Hyperthermia[1,5] fever, tachycardia, tachypnea, and mild leukocytosis may also be associated with acute hypernatremia.[6] Body weight loss exceeding 10% of birth weight has been useful in the diagnosis of breastfeeding-associated neonatal hypernatremia,[10,23] except during the first week of life where weight loss is expected and false positives occur.[23]

Pathophysiology

In a simplistic description of a highly complex and overlapping spectrum of fluid and sodium imbalance, the development of hypernatremia can be broadly categorized into: hypovolemic (negative fluid balance), euvolemic (stable body water), and hypervolemic (positive fluid balance) variants. Proper identification of the associated variant of hypernatremia is important when considering the most appropriate treatment as well as preventative strategies. Since water intake is stimulated by behaviorally-driven thirst (in response to high osmotic and/or low circulating blood volume stimuli) hypernatremia rarely occurs in individuals given free access to fluids and responsive to physiologically-mediated thirst sensations. Our physiological reliance on the thirst mechanism, to actively seek fluids in order to sustain life, is best evidenced in patients who either do not synthesize (central diabetes insipidus), or are not responsive (nephrogenic diabetes insipidus) to anti-diuretic hormone.[24,25] Individuals with diabetes insipidus cannot retain water (all fluid intake is promptly excreted by the kidneys as urine) but able to maintain normal sodium concentrations (normonatremia) by drinking to thirst, upwards of 15-20 L of fluid per day, to match maximal urinary losses.[26,27] Additionally, the thirst drive is so robust that dehydrated patients will actively seek out water, drinking out of flower vases[28] and sinks,[19] to satisfy an innate biological requirement to maintain whole body fluid homeostasis at all times. Figures 1, 2, and 3 summarize the three different volemic classifications and treatment detailed below.

1) Dehydration and negative water balance (hypovolemia): The hypovolemic variant of hypernatremia (or more commonly referred to as "hypernatremic dehydration") is associated with a negative water balance and results from either significant fluid losses (i.e. urine, sweat, or gastrointestinal losses), inadequate water intake or a likely combination of both. In one study, 82% of hypernatremic patients admitted to the hospital were volume depleted.[22] This suggests that hypovolemic hypernatremic is the dominant variant seen in sick individuals who lose disproportionately more water than they can replace.[22] Hypernatremic dehydration from excessive fluid losses is typically associated with high gastrointestinal fluid losses[12] or sweat losses[29,30] coupled with nausea and vomiting, which would preclude voluntary fluid intake in response to osmotic and/or volemic-driven thirst.

The most common cause of hypernatremic dehydration, however, is inadequate fluid intake. Unsuccessful breastfeeding[10,11,31] coupled with parental inability to suspect poor hydration status[31,32] has led to morbidity and mortality in infants during the first two weeks of life. Severe hypovolemic hypernatremia with

osmotic demyelination has also been documented in a 19-year old asylum seeker whereas no food or fluid was ingested for six days.[18] Hypernatremic dehydration is also seen in mentally and physically handicapped

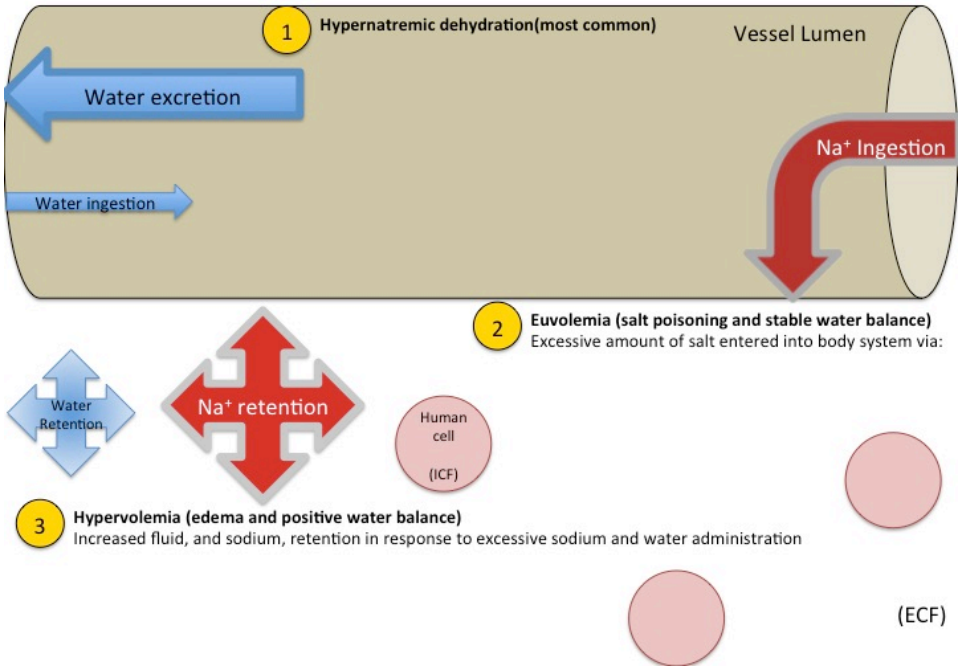

Figure 1. Schematic diagram of the pathophysiology of hypernatremia. See text for more information regarding the pathophysiology corresponding to the circled numbers.

1 **Hypernatremic dehydration(most common)**
lack of water in the body system via:
- Inadequate fluid intake
- Unsuccessful breastfeeding
- Diarrheal disease
- Excessive urination
- Excessive sweat production
- Gastrointestinal water losses

$$\uparrow[Na^+] = \frac{Na^+(mmol)}{\downarrow Volume(L)}$$

2 **Euvolemia (salt poisoning and stable water balance)**
Excessive amount of salt entered into body system via:
- Ingestion of supersaturated salt solution
- Salt poisoning
- Mistaking salt for sugar
- Salting skin of newborns
- Salting rituals
- Salt as an emetic

$$\uparrow[Na^+] = \frac{\uparrow Na^+(mmol)}{Volume(L)}$$

3 **Hypervolemia (edema and positive water balance)**
Increased fluid, and sodium, retention in response to excessive sodium and water administration in association with:
- Hypoalbuminemia
- Azotemia
- Excessive salt ingestion during prolonged exercise

$$\uparrow[Na^+] = \frac{\uparrow\uparrow Na^+(mmol)}{\uparrow Volume(L)}$$

Figure 2. Examples of the underlying etiologies of hypernatremia. See text for more information regarding the pathophysiology corresponding to the circled numbers.

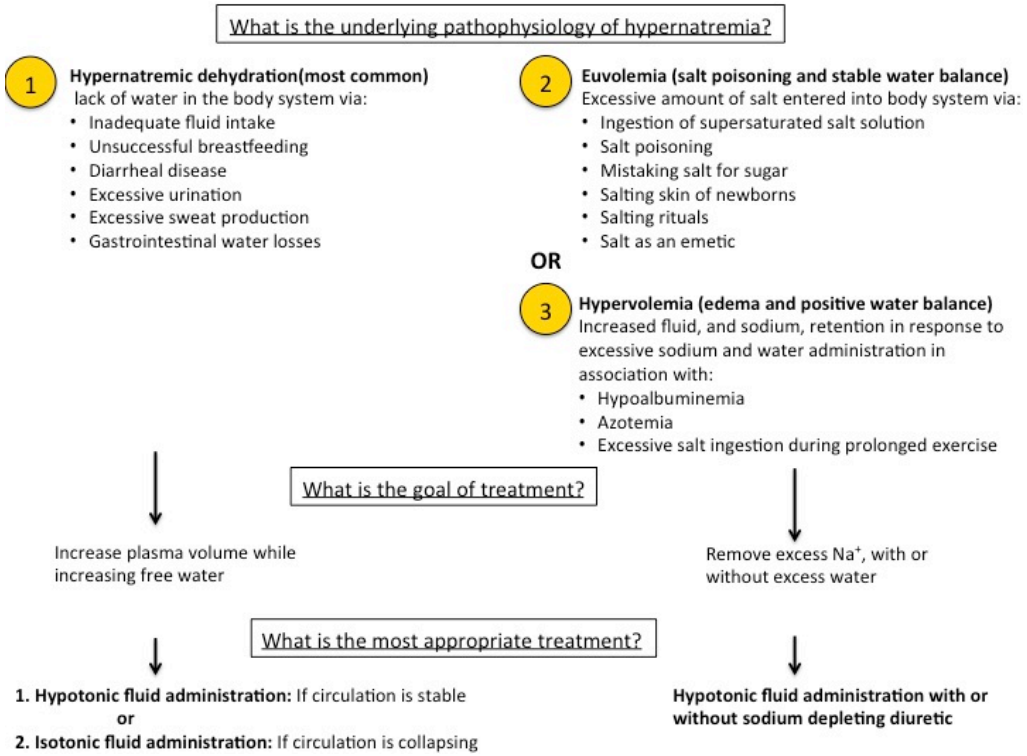

Figure 3. Treatment of hypernatremia categorized by underlying pathophysiology. See text for more information regarding the pathophysiology corresponding to the circled numbers.

individuals, and widely considered a warning sign (or "sentinel event") of caretaker neglect in nursing homes.[9,13-17]

2) Salt poisoning and stable water balance (euvolemia): The euvolemic variant of hypernatremia may occur after massive and rapid intake of sodium, which overwhelms the body's capacity to excrete the excess sodium or effectively dilute rapidly rising blood sodium levels. Exuberant salt intake is commonly referred to as "salt poisoning" or "salt intoxication" and further subdivided into accidental or non-accidental poisoning.[9] As little as two tablespoons of salt (30 g) can rapidly elevate blood [Na^+] depending on weight,[33] with the minimum lethal dose of salt ranging between 0.75-3 g/kg of body weight.[9] Fatalities from accidental salt poisoning have resulted from individuals mistaking salt for sugar,[2,4] using saltwater as an emetic,[5] ingesting saltwater as an exorcism ritual,[33] and salting the skin of newborns as a Turkish custom.[34] Fatalities from non-accidental salt ingestion include a depressed woman who committed suicide by drinking ~1 L of soy sauce as well as children forced to ingest saturated salt solutions as an emetic or as a form of punishment.[9]

3) Edema and positive water balance (hypervolemia): The hypervolemic variant of hypernatremia (often referred to as "edematous hypernatremia") is associated with positive water balance in which increased fluid retention in response to excessive sodium and water administration results in marked edema.[22,35] This variant of hypernatremia is most often seen in critically ill patients and is being documented with increasing frequency in hospitalized patients.[22,35-37] Edematous hypernatremia in hospitalized patients is often associated with hypoalbuminemia and azotemia, although the pathophysiology of both sodium and fluid overload is complex and poorly understood.[22,35]

Hypovolemic hypernatremia may also occur in healthy individuals who ingest exuberant amounts of sodium during prolonged endurance exercise. Accordingly, in a subgroup of 231 endurance athletes who gained body weight during a marathon run (42.2 km) or Ironman

Triathlon (226.3 km), 5% were found to be hypernatremic immediately post-race.[38] It is speculated that these hypervolemic hypernatremic athletes ingested exuberant amounts of both sodium and fluid during the race, which were then retained via exercise-induced increases in both antidiuretic hormone and aldosterone.[39,40]

Morbidity and Mortality from Hypernatremia in Community Dwellers

Severe, life-threatening, hypernatremic encephalopathy (with seizures and coma) has occurred in otherwise healthy individuals (community dwellers) who either: 1) do not have free access to fluids (to fully satisfy thirst sensations) or 2) ingest high amounts of sodium within short periods of time. Morbidity directly attributed to hypernatremia is uncommon, with a 2009 United Kingdom Pediatric Guideline estimating the annual incidence around ~5/100,000 children.[9] While the incidence of hypernatremic dehydration in breastfed infants is more frequently encountered (ranging between 1/200 and 1/1,400),[9] a single case of salt poisoning is likely to occur once every 40-50 years (in children) in a typical city of one million inhabitants (of all ages).[9] Thus, hypernatremic deaths are not expected in healthy, free-living, humans but rather seen in infants and children, abused individuals, nursing home patients, the mentally ill, athletes, and those lost on land or sea. More specific examples are illustrated below.

Infants and children: Reports of infant morbidity and mortality have been reported in children with diarrheal disease, from inadequate breastfeeding, salt poisoning and skin salting. The diagnostic distinction between hypernatremic dehydration and salt poisoning is of critical importance, both from a treatment and medicolegal perspective.

Diarrheal disease: Diarrheal disease is common in infants and the second leading cause of death (9% of 6.3 million worldwide) in children <5 years of age.[12] In 1955, it was reported that 85% of children admitted into the hospital with hypernatremia had diarrheal disease.[41] However, the incidence appears to be decreasing with a 1999 study documenting only a 20% incidence of hypernatremia in children admitted to the hospital with gastroenteritis.[42] Recent data collected from a Diarrhea Hospital in Bangladesh document a 19.5% incidence of hypernatremia and 23.8% incidence of hyponatremia.[12] The case fatalities were higher in hypernatremic (12%) children, however, compared with hyponatremic (5%) children.[12] Estimated diarrheal sodium losses range between 30 and 90 mmol/L which, like sweat, are hypotonic to plasma.[43] Thus, hypernatremia from diarrheal disease more likely develops from inadequate fluid replacement rather than exuberant gastrointestinal salt losses.

Breastfeeding: The incidence of hypernatremic dehydration due to inadequate breastfeeding has been estimated between 2.5-7.1 infants for every 10,000 live births,[11,23] with death occurring in 2.3% of all reported cases.[10,31] As breastfeeding is encouraged worldwide, cases of hypernatremic dehydration appear to be increasing due to a combination of poor suckling by the infant and lack of recognition of dehydration by the mother.[32] High breast sodium milk content and weight loss >10% of birth weight (from poor feeding) are common findings in infants with hypernatremic dehydration,[10] with an onset typically between 3 and 21 days post-delivery.[32] Other warning signs of hypernatremic dehydration in breastfeeding infants include: poor hydration state, jaundice, elevated body temperature, irritability or lethargy, decreased urine output, and seizures.[10]

Salt poisoning: In 1962, fourteen infants in a Baltimore hospital were salt poisoned by drinking formula accidentally made with salt instead of sugar.[2] Five infants died from hypernatremic-induced hemorrhagic encephalopathy before the error was recognized.[2] Symptoms of hypernatremia in these infants included muscle twitching, vomiting, avid thirst, and convulsions. When tested later, the implicated milk formula contained between 739 to 1170 mmol/L of sodium.[2]

In another case report, a 10 week-old infant with cystic fibrosis developed salt poisoning when given a formula supplemented with one heaping teaspoon of salt to presumably offset sweat sodium losses.[44] When tested, the infant's formula contained 358 mmol/L of sodium. After three weeks of ingesting 30 oz/day of this sodium supplemented feed, the child was admitted to the hospital for irritability, vomiting, back arching, lung

crackles, and an initial serum sodium level of 204 mmol/L.[44] Astonishingly enough, the infant survived and appeared to be developing normally after one year.
One particularly noteworthy case of accidental salt poisoning involved a 19-year old male college student who drank ~1 L of soy sauce (~17% sodium chloride) as part of a fraternity hazing ritual.[6,45] Once diagnosed with life-threatening hypernatremia, 6 L of 5% dextrose in water (D5W) was rapidly infused within 30 minutes. Despite a peak blood [Na^+] of 191.3 mmol/L, the quick dilution of serum sodium levels (to 154 mmol/L) led to his survival. Other less fortunate cases of salt poisoning have been summarized elsewhere in the literature.[9]

Salting rituals: The salting of infants is an ancient Turkish custom, whereas the skin of newborns is scrubbed with table salt to promote health.[34,46,47] Exposing the skin to salt for long periods of time can induce separation of the epidermis from the dermis, or "burn" the skin. When the salt crystals directly contact the vascular beds of the dermis, or when full-thickness skin abrasions develop from vigorous salt rubbing, sodium chloride freely enters the circulation and often causes significant morbidity[46,47] and mortality[34,46] from hypernatremia. Babies that are small for gestational age (under-developed skin) or with breastfeeding difficulties (dehydration) appear at increased risk for salt poisoning from skin salting.[46]

Salt as an emetic: Table salt (saline emetic) was once recommended in first-aid manuals (and in early editions of Harrison's Principles of Internal Medicine)[48] to induce vomiting (emesis) after accidental poisoning or drug overdose.[49,50] As such, deaths have occurred in a variety of instances including when: parents used saltwater to induce vomiting in a teen who "may have" taken a pill at a party,[5] parents gave their 4-year old several glasses of salt water after she swallowed bath foam,[7] and in nurses who wished to induce vomiting after patients swallowed cigarette butts, a fellow patient's pills, or overdosed on aspirin.[7,50] The fatal consequences of table salt as well as baking soda (sodium bicarbonate) were acknowledged by the US Consumer Product Safety Commission in 1977, with syrup of ipecac recommended as a replacement[44] (an outdated practice which is now also discouraged).

Cognitive and physical impairment: Cases of fatal salt poisoning have been reported in a 41 year-old man with Down's syndrome who swallowed a supersaturated salt solution intended for gargling and whose foster parents removed access to fluids because his resulting polydipsia was "uncharacteristic,"[1] a 45 year-old obese (86kg) woman with Prader-Willi syndrome who became ill 2-3 hours after eating four cups of jam topped with 3-4 heaping teaspoons of salt (mistaken as sugar),[4] and in two adult patients living in nursing homes who were given salt as an emetic.[7] All four individuals suffered from severe mental handicaps, which limited their ability to both express and detect fatal errors of salt ingestion.

In contrast, it has been debated whether or not otherwise healthy children (or adults) would willfully ingest enough sodium (as per palatability) to induce symptomatic hypernatremia. As such, three separate case reports made the suggestion that such "voluntary salt poisoning" may occur. However, subsequent critical appraisal by a panel of 16 pediatricians dismissed the credibility of the (authors) suggestions in all three case reports, collectively concluding it was highly unlikely that children (or adults) would spontaneously or voluntarily ingest sufficient quantities of salt to induce symptomatic hypernatremia.[9] Isolated case reports of avid salt cravings are uncommon and mainly seen in individuals with severe corticoadrenal insufficiency.[51]

In contrast to salt poisoning, hypernatremic dehydration has been documented more frequently in mentally ill or elderly persons living in nursing facilities[15-17] and is viewed as a sign of neglect.[13,14] In one United Kingdom study, comprising 21,610 patients over the age of 65 years admitted to hospital, individuals admitted from nursing homes had a ten-fold higher incidence of hypernatremia compared with patients admitted from their own homes.[16] The highest probability of being admitted to the hospital with hypernatremia was in individuals living in care homes with dementia followed by nursing home patients without dementia, individuals living in their own homes with dementia and lastly in those patients living in their own homes without dementia. Thus, it is apparent

that elderly or handicapped individuals who cannot ambulate, unable to express thirst and who rely on others to provide access to fluids are most at risk for developing hypernatremic dehydration.[13-16]

Suicide/depression: In China and Japan, saturated salt solutions have been used as a traditional suicide method.[7,8,52] As such, it has been verified in the literature that at least two, depressed, Japanese women committed suicide by drinking 700-1000 mL of soy sauce.[8,52] In another documented case from Israel, a 20-year-old female, suffering from post-partum depression, drank six glasses of a mixture containing 1 kg of table salt in 1 L of water as part of an exorcism ritual on the advice of her family.[33] Eleven hours after this exorcism, the patient became lethargic, began seizing, presented to the hospital with an initial serum sodium level of 255 mmol/L, and died a few hours later.[33]

Lost on land or sea: There appears to be a paucity of scientific reports detailing mortality from dehydration hypernatremia in the desert.[53] Most of these "death by dehydration" cases lack actual data and principally derived from the lay literature, detailing tragic stories of hikers getting lost in the desert and running out of water (unexpectedly) in hot climates.[54-56] Alternatively, there has been one case of salt poisoning in a fisherman who ingested large amounts of seawater after drifting at sea for 11 hours during a hurricane. His initial serum sodium (after rescue) was 175 mmol/L. Once his sensorium returned, he complained of severe thirst and drank "voraciously" from the sink and later survived.[19]

Athletes: It is well documented that endurance athletes finish often races with asymptomatic[57-59] and symptomatic[29,30] hypernatremia. In one study following a 90 km footrace, 58% of all runners treated within the medical tent were diagnosed with hypernatremia.[30] In this cohort of collapsed athletes, nausea and vomiting were reported more frequently in those runners diagnosed with hypernatremia compared with normonatremic, collapsed runners. It remains unclear, however, whether the vomiting limited adequate fluid intake (hypernatremia) or if the hypernatremia triggered the nausea and vomiting in this cohort of collapsed runners.[30] Regardless, runners with dysnatremia demonstrated a delayed recovery compared with normonatremic runners treated in the medical tent.[29] Not a single death has been reported from exercise-associated hypernatremia, although three cases of fatal hyperpyrexia in military recruits have been reported in association with elevated sodium (and potassium) concentrations.[60]

Hospital-acquired Hypernatremia and Patient Prognosis

The incidence of hypernatremia in patients presenting to the emergency department is between 0.9% and 2%,[21,61] with severe hypernatremia (blood [Na^+] >160 mmol/L) seen in 0.04% of patients and an independent predictor of death.[62] While hypernatremia upon hospital admission is low (~2%), hospital-acquired hypernatremia within intensive care units (ICU) during treatment is higher, ranging between 26-61%.[62,63] The incidence of *hypo*natremia is consistently higher than *hyper*natremia in hospitalized patients with a clear U-shaped relationship documented between blood [Na^+] and mortality.[62-64] Data obtained from 18 French ICU's even suggests that correction of dysnatremia within 48 hours of admission to the ICU increases patient survival to equal that of normonatremic patients.[65]

Hypernatremic patients that are either admitted into the hospital or develop hypernatremia while in the hospital (hospital acquired) often have associated febrile illness,[22] sepsis,[36] and/or uncontrolled diabetes.[22] Hospitalized hypernatremic patients tend to be older (>80 years) and be more dependent on others compared with normonatremic patients.[66] A recent study, investigating the incidence (%) of both hyponatremia and hypernatremia in patients admitted into the intensive care unit (ICU) over a 21-year period, documented a decrease (47% to 25%) in hyponatremia with a concomitant increase (13% to 24%) in severe hypernatremia (serum [Na^+] >155 mmol/L) over time.[37] The authors of this retrospective study proposed that the shift from hyponatremia to hypernatremia in hospitalized patients likely resulted from liberal use of intravenous fluids, increased diuretic use, and increased administration of hydrocortisone.[37] Another retrospective study performed on 130 ICU patients with hospital-acquired hypernatremia found that one-third of the hypernatremia cases were

preceded by hyponatremia.[36] These authors suggested that too little water was administered, along with too little salt, in these critically ill individuals.[36]

Morbidity and mortality from hypernatremia primarily results from neurological damage secondary to cellular desiccation. The rapid, osmotically induced, "shrinking" of cells may cause osmotic demyelination (similar to rapid overcorrection of hyponatremia) along with intracerebral and subarachnoid hemorrhaging from the rupture of cerebral veins. Alternatively, temporary induction of hypernatremia has been utilized as a medical treatment following traumatic brain injury (TBI) as a strategy to reduce intracranial pressure (ICP).[67] The success of sustained hyperosmolar therapy to reduce TBI-induced cerebral edema has been limited, however, but worthy of mention as an experimental option.[67]

Treatment and Prevention of Hypernatremia

As with any disease state, the hallmark of treatment is to appropriately identify and reverse the underlying pathophysiology (i.e. gastroenteritis, infection, fever, improper feeding, or diuresis etc.). The hypernatremia must then be corrected by replacing total body water stores or removing excess sodium (with or without excess water) from the body. As stated previously, careful attention should be given as to whether or not hypernatremia developed slowly (chronic, as in the cases of dehydration hypernatremia) or quickly (acute, as in cases of salt poisoning). Care should be taken not to correct blood [Na^+] too rapidly or overcorrect, as cerebral and non-cardiogenic pulmonary edema may result, mimicking the pathophysiology of water intoxication.[1,5,7,8,31]

After the cause and onset (acute or chronic) of hypernatremia is assessed, the most appropriate choice of infusate as well as the rate of blood sodium correction must be determined. Most guidelines suggest hypotonic fluid replacement (D5W, 0.2% or 0.45% NaCl) unless signs of frank circulatory collapse are noted (in which case 0.9% saline is the better options).[68] The oral route is also preferred, although this mode of fluid resuscitation is not often possible due to nausea, vomiting, and other central nervous system signs and symptoms. The recommended correction rate for acute hypernatremia is 1 mmol/L/h while a lower correction rate of 0.5 mmol/L/h is preferred for chronic hypernatremia (or of unknown origin)[68,69] with under-correction of severe hypernatremia associated with increased mortality.[69] As with any fluid resuscitation therapy, frequent clinical and laboratory reassessment is strongly advised due to the uncontrollable nature of ongoing fluid losses. More detailed explanations and treatment guidelines can be found in other clinical reviews.[3,68,70]

In summary, morbidity and mortality from hypernatremia is rare outside of the age group extremes (i.e. infants or elderly persons who cannot express thirst and rely on others to provide fluid) and hospitalized patients. Free-living, community dwellers with free access to fluids plus an intact thirst drive will not willfully develop hypernatremia unless septic, mentally ill, abused, lost in extreme environments, or gullible to erroneous advice. Thus, these sodium stories collectively suggest that hypernatremic deaths are both tragic and avoidable.

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